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AN INDEX OF
DIFFERENTIAL DIAGNOSIS
OF MAIN SYMPTOMS

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AN INDEX OF
DIFFERENTIAL DIAGNOSIS
OF MAIN SYMPTOMS

BY VARIOUS WRITERS

Edited by
HERBERT FRENCH, M.A., M.D. Oxon., F.R.C.P. Lond.
Physician, Pathologist, and Lecturer, Guy's Hospital; Consulting Physician to the Radium Institute.

THIRD EDITION

WITH THIRTY-NINE COLOURED PLATES
AND OVER THREE HUNDRED ILLUSTRATIONS IN THE TEXT

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NOTE TO THE THIRD EDITION.

The early exhaustion of the second edition called for an immediate re-issue. Advantage has been taken of this fact to introduce a few more coloured figures of cases which it was found impossible to complete in time for the second edition, and to replace some of the black and white illustrations by better examples.

These alterations, and the introduction of the appropriate references in the Text and in the General Index, constitute the only changes.

The publishers are gratified that the unprecedented demand for the volume has made these improvements possible at so early a date.

December, 1917.

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PREFACE TO THE SECOND EDITION

LITTLE need be said by way of preface to the second edition of this work; the gratifying sale of the first edition and the necessity there has been to reprint it several times is sufficient evidence that the book is one which the medical profession welcomes and appreciates.

In this edition every article has been revised and several new ones have been added.

The elaborate index, which has been much appreciated, has been made, if possible, even more complete, and at the same time it has been simplified in some particulars; the relative importance of the entries in it are indicated more clearly perhaps than was the case in the first edition, by the use of three degrees of type.

The illustrations are nearly doubled in number; the coloured plates especially having been increased from sixteen to thirty-seven, and neither time nor expense has been spared in the endeavour to make them characteristic of the conditions they represent.

The size of the type employed is larger than before, in response to suggestions from readers, and consequently the pages have had to be enlarged also.

It became a question, therefore, whether the book should be published in two volumes; in the belief, however, that in a work in which numerous cross-references are unavoidable it is advantageous to confine it within one cover, it has been decided to keep it as a single book. The general character of the volume remains otherwise unchanged.

It is hoped that this second edition will be as widely welcomed as was the first; and that it will prove even more helpful in its primary purpose, namely, to be of assistance in arriving at the diagnosis of the exact cause of particular symptoms.

Cordial thanks are extended to many helpers who, whilst not contributing written articles to the volume, have nevertheless assisted greatly in various ways; especially to Dr. D. S. Davies, Mr. C. Thurstan Holland, Dr. A. C. Jordan, Dr. T. Warner Lacey, Dr. T. M. Legge, Dr. Lindsay Locke, Professor Rutherford Morison, Sir Malcolm Morris, Dr. H. B. Newham, Dr. G. W. Nicholson, Dr. J. H. Ryffel, Mr. S. Gilbert Scott, Dr. W. P. Saunders, Dr. A. Rendle Short, Dr. Hugh Walsham, Dr. S. A. K. Wilson; also to the Royal Society of Medicine, the Gordon Museum, Guy's Hospital, the South Eastern Fever Hospital, and the London School of Tropical Medicine. Also to the publishers and proprietors of various journals and periodicals for unfailing courtesy in giving facilities for the use of copyright material and illustrations.

HERBERT FRENCH.

London,
February, 1917.

PREFACE TO THE FIRST EDITION

This book is a treatise on the application of differential diagnosis to all the main signs and symptoms of disease. It aims at being of practical utility to medical men whenever difficulty arises in deciding the precise cause of any particular symptom of which a patient may complain. It covers the whole ground of medicine, surgery, gynæcology, ophthalmology, dermatology, and neurology.

Whatever the disease from which a patient is suffering, the importance of diagnosing it as early as possible can hardly be over-rated. The present volume deals with diagnosis from a standpoint which is different from that of most text-books, having been written in response to requests for an *Index of Diagnosis* as a companion to the publishers' *Index of Treatment*, issued in 1907. The book is an index in the sense that its articles on the various symptoms are arranged in alphabetical order; at the same time it is a work upon differential diagnosis in that it discusses the methods of distinguishing between the various diseases in which each individual symptom may be observed. Whilst the body of the book thus deals with *symptoms*, the general index at the end gathers these together under the headings of the various *diseases* in which they occur.

The Editor lays particular stress upon the importance of using these two parts of the book together. Unless reference is made freely to the general index, the reader may miss a number of the places in which is discussed the diagnosis of the disease with which he has to deal; for while each *symptom* is considered but once, each *disease* is likely to come up for discussion under the heading of each of its more important symptoms.

The guiding principle throughout has been to suppose that a particular symptom attracts special notice in a given case, and that the diagnosis has to be established by differentiating between the various diseases to which this symptom may be due. One of many difficulties arising during the construction of the work was that of deciding where to draw the line as regards symptoms themselves. The exclusion of many borderline headings such as "Dullness at the base of one lung." "Inability to breathe through the nose," and various signs such as Romberg's, Stellwag's, von Graefe's, and so forth, may perhaps seem arbitrary; but reference to the minor symptoms and physical signs which have not been thought sufficiently important to merit separate articles will be found in the general index at the end of the volume.

Treatment, pathology, and prognosis are not dealt with except in so far as they may bear upon differential diagnosis—the employment of salicylates, for instance, in distinguishing acute rheumatic from other forms of arthritis; the use of the microscope in distinguishing malignant neoplasms from inflammatory or other tumours; the value of the lapse of time in distinguishing between tuberculous and meningococcal meningitis.

Coloured plates and other illustrations have been introduced freely wherever it was thought they might be helpful in diagnosis. Most of them are original, but a few are reproduced from other sources, and thanks are due to the authors and publishers who have kindly lent them.

So far as the Editor is aware, although there exist indices of symptoms, and medical works in which various maladies are discussed in alphabetical order, the present Index of Differential Diagnosis of Main Symptoms is unique in medical literature. It rests with the medical profession to decide whether it strikes the mark at which it aims. There must be room for improvement in many respects, notwithstanding the great amount of time and labour that have been bestowed upon it.

However this may be, the work undoubtedly owes much of what value it possesses to the suggestions and kindly help of the many contributors who have assisted in its making; and to the practitioners and the authorities of various institutions who have generously lent the material for many of the illustrations. Indeed, it is difficult to see how the book could have been produced in its present completeness without their willing collaboration: they are enumerated elsewhere, and to all of them the Editor tenders his sincere thanks.

Criticisms and suggestions are invited, and will be received with gratitude by the Editor,

HERBERT FRENCH.

62, *Wimpole Street, London, W.*
March, 1912.

LIST OF
CONTRIBUTORS AND THEIR SUBJECTS

William Cecil Bosanquet, M.A. Oxon., M.D., F.R.C.P.; Physician, Brompton Hospital for Consumption and Diseases of the Chest; Physician, Charing Cross Hospital.

Blood per anum, 75.—Colic, 114.—Glycosuria, 260.—Melæna, 385.

E. Farquhar Buzzard, M.A., M.D. Oxon., B.CH., F.R.C.P.; Physician to Out-Patients at St. Thomas's Hospital and at the National Hospital for the Paralyzed and Epileptic, Queen Square; Consulting Neurologist to the Royal Free Hospital and to the Hospital for Diseases of the Throat, Golden Square.

Amnesia, 19.—Ataxy 55.—Aura, 67.—Claw-foot, 109.—Claw-hand, 109.—Facies, abnormalities of, 233.—Girdle-pain, 260.—Incontinence of fæces, 313.—Irritability, 323.—Knee-jerk, abnormalities of the, 357.—Pain in the extremity (lower), 438.—Pain in the extremity (upper), 442.—Pain in the face, 446.—Paralysis, facial, 491.—Paralysis of one extremity (lower), 496.—Paralysis of one extremity (upper), 500.—Sensation, some abnormalities of, 604.—Speech, abnormalities of, 623

Percy John Cammidge, M.D. Lond., D.P.H. Camb.

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Herbert L. Eason, M.D., M.S. Lond.; Senior Ophthalmic Surgeon, Guy's Hospital.

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John W. H. Eyre, M.D., M.S. Durh., D.P.H. Camb.; Director of the Bacteriological Department, Guy's Hospital; Lecturer on Bacteriology to the Medical School and Dental School, Guy's Hospital.

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Carey F. Coombs, M.D., F.R.C.P.; Assistant Physician, Bristol General Hospital; Clinical Lecturer in Medicine, University of Bristol.

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Herbert Morley Fletcher, M.A., M.D. Camb., F.R.C.P.; Physician, St. Bartholomew's Hospital; Physician in Charge of Diseases of Children Department, St. Bartholomew's Hospital; Consulting Physician, East London Hospital for Children.

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Herbert French, M.A., M.D. Oxon., F.R.C.P.; Physician, Pathologist, and Lecturer, Guy's Hospital.

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Archibald Edwd. Garrod, C.M.G., M.A., M.D. Oxon., F.R.C.P., F.R.S.; Physician, St. Bartholomew's Hospital; Consulting Physician to the Hospital for Sick Children, Great Ormond Street.

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George Ernest Gask, F.R.C.S.; Surgeon with Charge of Out-patients, St. Bartholomew's Hospital; Joint Lecturer in Surgery, St. Bartholomew's Hospital Medical School.

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Hastings Gilford, F.R.C.S.; Consulting Surgeon, Kingwood Sanatorium, Reading.

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Arthur Frederick Hurst, M.A., M.D. Oxon., F.R.C.P.; Physician and Physician for Nervous Diseases and Lecturer on Therapeutics, Guy's Hospital.

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Robert Hutchison, M.D., C.M. Edin., F.R.C.P.; Physician to the London Hospital; Physician with Charge of Out-patients to the Hospital for Sick Children, Great Ormond Street.

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Arthur John Jex-Blake, M.A., M.D., B.Ch. Oxon, F.R.C.P.; Senior Assistant Physician, St. George's Hospital; Assistant Physician, Brompton Hospital for Consumption.

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Sir Malcolm Morris, K.C.V.O., F.R.C.S. Edin.; Consulting Surgeon, Skin Department, St. Mary's Hospital; Surgeon, Skin Department, Seamen's Hospital.

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Frederick John Smith, M.A., M.D. Oxon., F.R.C.P., F.R.C.S.; Physician and Senior Pathologist, London Hospital; Consulting Physician to the City of London Dispensary and to the National Orthopædic Hospital.

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THOMAS GEORGE STEVENS, M.D., B.S. Lond., M.R.C.P., F.R.C.S.; Obstetric Surgeon, St. Mary's Hospital; Gynæcological Surgeon, Hospital for Women, Soho Square; Physician to In-Patients, Queen Charlotte's Hospital.

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RUSSELL H. JOCELYN SWAN, M.B., M.S. Lond., F.R.C.S.; Surgeon, Cancer Hospital, Brompton.

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FREDERICK TAYLOR, M.D. Lond., F.R.C.P.; Consulting Physician, Guy's Hospital, and Evelina Hospital for Sick Children; Physician, Seamen's Hospital, Greenwich.

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PHILIP TURNER, B.SC., M.B., M.S. Lond., F.R.C.S.; Assistant Surgeon, Guy's Hospital; Demonstrator of Operative Surgery, Guy's Hospital Medical School.

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WILLIAM HALE WHITE, M.D. Lond., M.D. Dub., F.R.C.P.; Senior Physician and Lecturer on Medicine, Guy's Hospital.

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Cutaneous reaction (*von Pirquet*); Dermal reaction (*Woodcock*).

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Dermal reaction (*Moro*); Ophthalmo-test (*Calmette*).

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AN INDEX OF
DIFFERENTIAL DIAGNOSIS
OF MAIN SYMPTOMS

ACCENTUATION OF HEART SOUNDS.—It may be that, without cardiac bruit, one or other of the heart sounds is much louder than it ought to be. Such accentuation generally has important clinical significance. It is the first sound that is likely to be accentuated or prolonged at the impulse; whilst in the second right, or second and third left intercostal spaces close to the sternum, it is the second sound that is likely to be accentuated rather than the first. It is very unusual to find the first sound accentuated at the base or the second sound at the impulse, unless there is at the same time still greater accentuation of the first sound at the impulse, and of the second sound at the base respectively. Hence the three conditions under which accentuation of a cardiac sound becomes clinically important are: (1) *When the second sound is unduly loud in the second right intercostal space close to the sternum;* (2) *When there is accentuation of the second sound with maximum of intensity in the second or third left intercostal space close to the sternum*; (3) *When there is accentuation of the first sound with maximum intensity at or near the impulse.*

Accentuation of the second sound with maximum intensity in the second right intercostal space close to the sternum nearly always indicates that the systemic blood-pressure is above the normal. The latter can only be determined with certainty by actual measurement of the systemic blood-pressure instrumentally. The causes of the increase will probably be one or other of the following:—

Age.—Even healthy persons over fifty begin to show slight increase of BLOOD-PRESSURE (p. 81); and their aortic second sound begins to get louder than the first.

Arteriosclerosis or *granular kidney.*—These can be discussed together, because it is extremely difficult to tell where the one ends and the other begins. In both there is cardiac hypertrophy, increase in the blood-pressure, prolongation of the first sound at the impulse, possibly a blowing systolic bruit there, a ringing or clanging aortic second sound, albuminuria, a tendency to heart failure as time goes on, with all its concomitant symptoms, and albuminuric retinitis. It is sometimes stated that the accentuation of the aortic second sound is due to local atheroma; but this is inaccurate, for atheroma by itself, though it may easily produce an aortic systolic bruit, does not accentuate the second sound; and when in the second right intercostal space there is a soft systolic bruit replacing the first sound, and a clanging second sound, the former indicates atheroma of the aortic valves, and the latter arteriosclerosis. These two absolutely distinct vascular lesions often coincide in the same patient, atheroma affecting the aorta, and the coronary and cerebral arteries, whilst arteriosclerosis affects the middle-sized arteries, especially of the splanchnic area. There is often extensive visceral arteriosclerosis when the radial artery does not feel abnormal to the fingers.

Accentuation of the second sound with maximum intensity in the second or third left intercostal space close to the sternum, generally spoken of as accentuation of the pulmonary second sound, indicates a higher pressure than there should be in the pulmonary circulation, except in children, in whom it is not uncommon to find the pulmonary second sound normally much louder than the aortic. The most important cause of pathological accentuation of the pulmonary second sound is disease of the mitral valve; it occurs more markedly with mitral stenosis than with mitral regurgitation. It may sometimes be a marked feature of the latter, whether due to organic changes in the mitral valve itself, or secondary to dilatation of the otherwise normal orifice as the result of heart failure from aortic disease, myocardial degeneration, arteriosclerosis, or granular kidney. Sometimes, instead of accentuation of the pulmonary second sound, the latter may be reduplicated; the significance of its reduplication is identical with that of its accentuation, the probable reason for the reduplication being that when the pressure in the pulmonary circulation is relatively very much above the normal, the pulmonary semilunar valves close sooner than the aortic, the first part of the reduplicated second sound being due to closure of the pulmonary valves, whilst its second part is due to closure of the aortic valves. The cause of an accentuated or reduplicated pulmonary second sound will generally be obvious if the other cardiac physical signs are observed carefully; one way in which it may have particular significance is in distinguishing between old and recent changes in the mitral valve; when, for instance, a systolic and mid-diastolic bruit at the impulse are due to recent endocarditis which may possibly clear up, there is very much less accentuation of the pulmonary second sound than there would be if the same bruits were due to mitral stenosis and regurgitation due to old fibrotic changes. The greater the accentuation of the pulmonary second sound, the greater the mitral leakage or obstruction.

Accentuation of the first sound at the impulse may have one or other of two entirely different characters; it may be an accentuation of very short duration, difficult to describe in words, though obvious enough when heard, and often spoken of as a 'slapping' first sound at the impulse: this is one of the most characteristic physical signs in many cases of *mitral stenosis.* It may occur when there is neither a presystolic nor a mid-diastolic bruit, though even when there is a bruit the slapping character of the first sound is still to be distinguished. When there is failure of compensation in a mitral case, the driving power of the heart may become so feeble that bruits are no longer audible, and the heart's action is quite irregular; in such cases, the occurrence of this slapping character of the first sound, clearly audible here and there in an otherwise tumbling rhythm, is highly suggestive of mitral stenosis.

The second variety of accentuation of the first sound at the impulse consists in its being very much longer than it ought to be—a marked prolongation of the first sound as distinct from there being any bruit. This prolongation is obvious enough when heard. It indicates that there is considerable hypertrophy of the left ventricle, and therefore, in the absence of bruits, nearly always points to a high blood-pressure such as results from either arteriosclerosis, granular kidney, or the two combined; it is repeatedly met with in cases in which there is accentuation of the aortic second sound at the same time. In a person of middle age or over, in whom there is a prolonged first sound at the impulse —sometimes spoken of as a 'lumpy' first sound—and a clanging aortic second sound, with or without ALBUMINURIA (p. 11), a diagnosis of arteriosclerosis or of granular kidney is very probably correct, and instrumental determination of the blood-pressure will generally show that it has risen from the normal 120–150 mm. Hg to something between 180 and 300 mm. Hg, or even more.

It is noteworthy that transient accentuation of the first sound at the impulse may occur in nervous young patients examined while their hearts are acting rapidly; it vanishes in a few minutes when the patient becomes less nervous and the heart slower. The phenomenon is common in connection with life insurance examinations. *Herbert French.*

ACCOUCHEUR'S HAND is seen most characteristically in *tetany* (*Fig.* 1), though it may also occur in a few cases of other spasmodic neuro-muscular affections such as *athetosis.* In a typical case, the attitude of the fingers is almost pathognomonic. There is full extension of all the fingers and of the thumb at the interphalangeal joints, the four fingers are adducted firmly towards the middle finger, so as to form a cone, they are semi-flexed at the metacarpo-phalangeal joints, and the thumb is strongly adducted and opposed to the cone of which the middle finger forms the apex, or else into the palm of the hand. The spasmodic muscular contraction seldom ceases here, but generally affects the rest of the arm also, the wrist being flexed and abducted towards the ulnar side. The elbow is flexed to a right angle, and the arm rotated inward and adducted so as to lie in contact with the trunk. The affection is symmetrical. The feet and ankles are apt to show similar spasmodic contractions, the ankle being fully plantar-flexed, the toes and the distal half of the feet rotated inward, the knees extended rigidly, and generally the thighs also.

The contractions may be limited to the hands and feet—the so-called *carpo-pedal spasm*—especially in the tetany of young children suffering from rickets or from gastro-intestinal disorder such as diarrhœa. When adults are affected, the symptoms spread from the limbs to the trunk, the whole body being kept rigidly extended, the paroxysms lasting from a few minutes to many hours, and recurring for days, weeks, or even months. So far as the tetany itself is concerned recovery is invariable, though the patient may sometimes succumb to the associated malady, tetany itself being generally not a primary disease but a complication of gastric ulcer, gastrectasis, colitis, intestinal fermentation or putrefaction, thyroidectomy, or pregnancy. The diagnosis is seldom difficult.

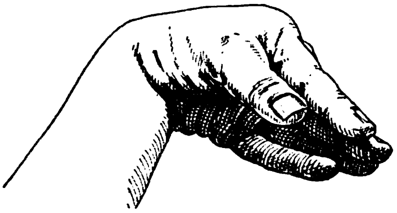


Fig. 1.—The hand in tetany.

One remarkable feature of the case is that in the intervals between the spasms, if the upper arm is grasped firmly between the observer's two hands, and the pressure maintained, the hand and wrist may be forthwith sent into the typical spasm, a sign described as Trousseau's. If the cheek close to the front of the ear is percussed gently but sharply from above downwards, the different groups of muscles supplied by the branches of the pes anserinus of the seventh nerve can be made to twitch successively—Chvostek's sign. The muscles of the limbs often show altered electrical reactions in that, though still responding to faradism, with galvanism A.C.C. is greater than K.C.C.—Erb's sign.

Herbert French.

ACETONURIA denotes the occurrence of acetone in the urine in amounts to be detected by ordinary clinical tests. In practice the laboratory method of distilling a quantity of urine to get a concentrated solution of any acetone that may be present takes too long, and yet without distillation it is difficult to apply the iodoform test for acetone. An easier and more useful plan is Legal's nitroprusside test, or Rothera's modification of it. Legal's test consists in taking 5 c.c. of urine in a test-tube, adding a few drops of liquor sodæ, then a few drops of fresh sodium nitroprusside solution, and finally acidifying with strong acetic acid. The liquor sodæ causes no change of colour, or at most an opalescence from the precipitation of phosphates ; the sodium nitroprusside produces a reddish-brown colour in almost all urines owing to the presence of creatinine ; if the red colour is due to creatinine only it is discharged on adding acetic acid, whereas when acetone is present the red deepens into a rich burgundy that is unmistakable. Rothera's modification of this test consists in adding a few drops of fresh nitroprusside solution to 5 c.c. of urine, liquor ammoniæ till the mixture is decidedly alkaline, and then ammonium sulphate crystals in excess ; as the solution becomes saturated with the latter, a colour like that of potassium permanganate develops if acetone is present, the maximum being reached in about fifteen minutes.

Acetone is often associated with diacetic acid, oxybutyric acid, and amido-oxybutyric acid ; the detection of these, however, affords no clinical information that is not afforded by the acetone alone, so that it generally suffices to test for the latter, and possibly for diacetic acid also. The tests for the butyric acids are difficult. When these substances are being produced, the patient is said to be suffering from *acidosis*, the result of unnatural metabolism. Acetonuria is indeed the chief practical evidence of acidosis. It occurs in the most extreme degree in certain cases of diabetes mellitus ; from the point of view of prognosis all cases of glycosuria may be divided broadly into two classes, namely, those with, and those without, acetonuria. The same patient may, of course, be passing acetone in his urine at one time and not at another ; the prognosis is always graver, however, when acetone is present, for it is the acidosis that causes the serious results of diabetes and glycosuria. A patient without acetonuria is in no immediate danger of coma, whereas, when acetone is present as well as sugar, coma may supervene at any time. Broadly speaking, once glycosuria has been diagnosed, it is more important to test the urine for acetone from time to time than it is for sugar, and that treatment which reduces the acetone to a minimum is, generally speaking, doing most good, whatever quantities of sugar may be passed.

Acetonuria may occur, however, without glycosuria; even a healthy person who is starved of carbohydrate food is apt to pass acetone and diacetic acid in the urine. This explains why it is that acetonuria occurs in such conditions as gastric ulcer; gastric carcinoma; gastrectasis; œsophageal stenosis; intestinal obstruction; cachexia, whether tuberculous, malignant, syphilitic or malarial; in cases of persistent vomiting of pregnancy; uræmia; severe migraine; infantile diarrhœa and vomiting; cyclical vomiting of children (p. 765); and probably in many other conditions in which there is either actual or virtual starvation. The same applies to surgical operations under anæsthetics—the patient is often starved beforehand, and may then be persistently sick afterwards; almost all patients who have been under a general anæsthetic for any length of time have acetonuria, and in some the acidosis increases instead of being transient, this being to a large extent, perhaps, the pathology of so-called delayed chloroform poisoning. It may also result from gross intracranial lesions, especially those of an inflammatory nature; thus, acetonuria may be pronounced even as early as the first two or three days in acute epidemic cerebrospinal meningitis.

The chief importance of acetonuria therefore from a diagnostic point of view lies, not so much in distinguishing one disease from another, as in detecting the existence of acidosis. The importance of this from the point of view of prophylaxis and treatment will be obvious when it is remembered that acidosis does not occur until the liver and tissues have lost their glycogen, and that glycogen storage depends largely upon the ingestion of carbohydrates either by the mouth, the rectum, or hypodermically. *Herbert French.*

ACIDOSIS.—(See ACETONURIA, p. 3.)

ACROPARÆSTHESIA.—(See PAIN IN THE EXTREMITY, UPPER, p. 442.)

ALBUMINURIA.—This term is used to denote the passage in the urine of proteid that is coagulable on boiling. More than one substance is included in this sense, and there are varying proportions of albumin and globulin in different cases. So variable may be the relative amounts of these, not only in different diseases, but also in different cases of the same disease, and in the same patient at different times, that little useful clinical information is to be obtained by dealing with them separately, at any rate so far as present knowledge goes. Nucleo-albumin (p. 424) comes in quite a different category.

Although numbers of tests for albumin have been devised and advocated, for clinical purposes there is little need to trouble about more than the two common ones, namely the *acetic acid and boiling*, and the *cold nitric acid* tests. It is true that each of these has fallacies; but the latter are not common to both, and therefore if there is doubt in the interpretation of one of the two tests, it can be confirmed or otherwise by the other. More delicate tests exist, but there is such a thing as too great delicacy in a clinical method. One does not want to find albumin in minute traces where it does not matter; and it seldom matters until its amount is sufficient to give both the common tests.

The Acetic Acid and Boiling Test.—A test-tube three parts full of urine—cleared by filtration if need be—is held by its lower end whilst its upper part is heated carefully to boiling point. It is best not to add any acetic acid before boiling unless the specimen is distinctly alkaline, in which case it may be just acidulated with a drop of acetic acid. After boiling, the tube should be held in a good surface-light against a dark background, such as the sleeve of one's coat; any opalescence will be obvious, and there may be a dense white cloud. Except in rare cases of Bence-Jones albumosuria (p. 16), this will be due to one or more of three things, namely, calcium and magnesium phosphate, calcium carbonate, or coagulated albumin. One, two, or more drops of acetic acid solution (B.P.) are now added; if the cloud disappears entirely, quickly, and at once, it was due to earthy phosphates, and no albumin is present; if it disappears entirely but with brisk effervescence, the latter is due to calcium carbonates amongst the phosphates, and no albumin is present; if, on the other hand, the cloud clears up but partially, or remains unaltered, or actually increases and becomes more flocculent, albumin is almost certainly present. There is only one serious fallacy remaining, and that is in regard to nucleo-proteid; this is precipitated by acetic acid, and it is possible for a cloud of phosphates to be cleared up by the latter and yet for a faint cloud of nucleo-proteid to come down in the place of the phosphates in such a way as to suggest that the original cloud was not wholly soluble in the acid, and therefore that albumin is present when it is not. There are three ways of obviating this source of fallacy: the first is to add a single drop of dilute non-fuming nitric acid to the suspicious cloud that remains after adding the acetic acid; if it is due to albumin it will persist or even increase, whilst if it is due to nucleo-proteid the nitric acid will disperse it; the second is to perform the cold nitric acid test for albumin as described below—nucleo-proteid will not give a definite localized white ring with it; and thirdly, a control test may be done, acetic acid being added to another specimen of the urine without boiling, and the cloud due to any nucleo-proteid present compared with the cloud in the acidulated and boiled specimen.

Heller's Cold Nitric Acid Test.—About an inch and a half of urine is poured into a test-tube, the latter is held much inclined, and colourless nitric acid is allowed to flow gently down the side until about one-third as much as the urine has been added. The nitric acid is heavier than urine and goes to the bottom; if albumin is present a white ring forms at the junction of the two fluids. Some prefer to pour the nitric acid into the test-tube first, and then add the urine with a pipette. It is important not to shake the tube, or the nitric acid and urine will mix and there will be no definite junction line between them. Fuming nitric acid must be avoided because the nitrous oxide fumes decompose the urea and the resultant bubbles mix the fluids; sometimes there is bubbling even when the nitric acid is colourless, in which case this is due tc CO_2 set free from carbonates. The test is very delicate; if any large quantity of albumin is present, the ring appears at once; if there is only a trace, the white ring may not appear for a little, and the tube should be set aside and looked at again in a few minutes. Broadly speaking it takes three minutes for it to develop when the amount of albumin is 1 part in 30,000. This test is open to more fallacies, however, than the acetic acid and boiling test, so that it should not be trusted to alone unless it is negative. In concentrated urines it is common to get a dark-brown, reddish-brown, or violet-brown ring of colour at the junction; this is nothing to do with albumin; it is generally most marked in cases of INDICANURIA (p. 314). White rings, more or less like that due to albumin, may also be due to any of the following:—

1. *Resin.*—If the patient is taking copaiba or other similar drug, enough of the resin may be excreted in the urine to form a diffuse white cloud above the nitric acid. This fallacy is best avoided by bearing it in mind and checking the nitric acid test by the heat test; this latter safeguard applies to all cases of suspected albuminuria.

2. *Albumoses.*—These generally occur in association with albumin; should they occur alone the ring will disappear with warming, to reappear with cooling, and there will be no cloud with the heat test.

3. *Bence-Jones's Albumose.*—This occurs without albumin, gives a white ring with nitric acid that disappears on warming, to reappear on cooling; with the heat test, a dense cloud appears about 60° C., to disappear on further heating to boiling-point (p. 16).

4. *Nucleo-albumin.*—The ring with this is not in contact with the nitric acid, but higher up, and diffuse; it may be a real difficulty in diagnosis from albumin, for it is also precipitated by acetic acid, and may therefore give a haze with the boiling test (see above).

5. *Urates.*—These may form a cloud near the nitric acid if the urine is very concentrated; the cloud will disappear on gentle warming, to reappear on cooling, so that it may also be mistaken for albumose; the fallacy may be avoided by diluting the urine with plain water before the nitric acid test is employed.

6. *Urea Nitrate.*—If the urine contains a large percentage of urea a crystalline deposit of urea nitrate may form at the junction; as a rule the crystalline nature of the ring is obvious on inspection; but in case of doubt the urine should be diluted and the test repeated.

It does not matter which test is most relied upon when the result is negative; but before the positive deduction that a urine contains albumin is drawn, both the acetic acid and boiling, and the cold nitric acid tests, should be positive.

In arriving at a diagnosis of the precise cause of albuminuria in any given case, *it is essential that a microscopical examination of the centrifugalized deposit from the urine should be made.* Whatever else may be found, the first question to be answered is: Are renal tube-casts present as well as albumin, or not? All cases of albuminuria may be divided into two main groups, namely: (I) *Cases with renal tube-casts*; (II) *Cases without renal tube-casts.*

Renal Tube-casts.—When one speaks of renal tube-casts, however, one has to bear in mind that modern methods of centrifugalizing with electrically-driven machinery have reached such perfection that hardly anything that a specimen of urine contains escapes detection; technique has become almost too perfect; for when clinical methods become too delicate they begin to lose some of their clinical value. The result, in connection with casts, is that even in a great many normal urines an occasional renal tube-cast and an occasional red blood corpuscle are found; therefore when one speaks of cases of "albuminuria with tube-casts," one means "with enough renal tube-casts to be pathological." The observer learns from experience to know when the 'occasional' tube-cast is inside or outside the normal limits. More than one examination may be required, and the urine should be as fresh as possible, for casts disintegrate on standing, especially in hot weather and in alkaline urines.

Renal tube-casts are of various sorts (*Plate I*), and a certain amount of help can be derived from a knowledge of the particular kinds of casts present in a given case. Their matrix or foundation is a structureless material whose origin is obscure, though thought to be due to some kind of proteid coagulation. Sometimes the casts consist of this structureless matrix only, and according as they are then less or more highly refractile, they are spoken of as *hyaline casts* or *waxy casts* respectively. The hyaline is commoner than the waxy, but neither is characteristic of any particular disease. Embedded in the hyaline matrix there may be various substances or structures; and according to the main features of the embedded substances the casts receive different descriptive names. If renal epithelial cells predominate, the cast is an *epithelial cast*; if leucocytes or pus corpuscles, a *leucocytic cast*; if red blood corpuscles, a *blood cast*; if bacteria, *bacterial casts*; if fat globules, probably derived from degenerated renal cells or leucocytes, *fatty casts*; if non-fatty granular débris, *granular casts*. It is not at all uncommon to find a long cast which in one part is simply hyaline, at one end is granular, and at the other epithelial—a *mixed cast*. Upon the whole one may say that the hyaline cast occurs in all forms of nephritic conditions, whether acute or chronic; that epithelial and leucocytic casts indicate active catarrh; that granular casts tend to occur along with epithelial casts, but that when they occur alone or in association with hyaline casts they are evidence of at least less acute mischief than are epithelial casts; whilst fatty casts come between the two. Blood casts may occur in almost any variety of renal hæmorrhage, and by themselves they are not evidence of inflammation, though in association with other casts they indicate very acute inflammatory changes.

I.—ALBUMINURIA WITH RENAL TUBE-CASTS.

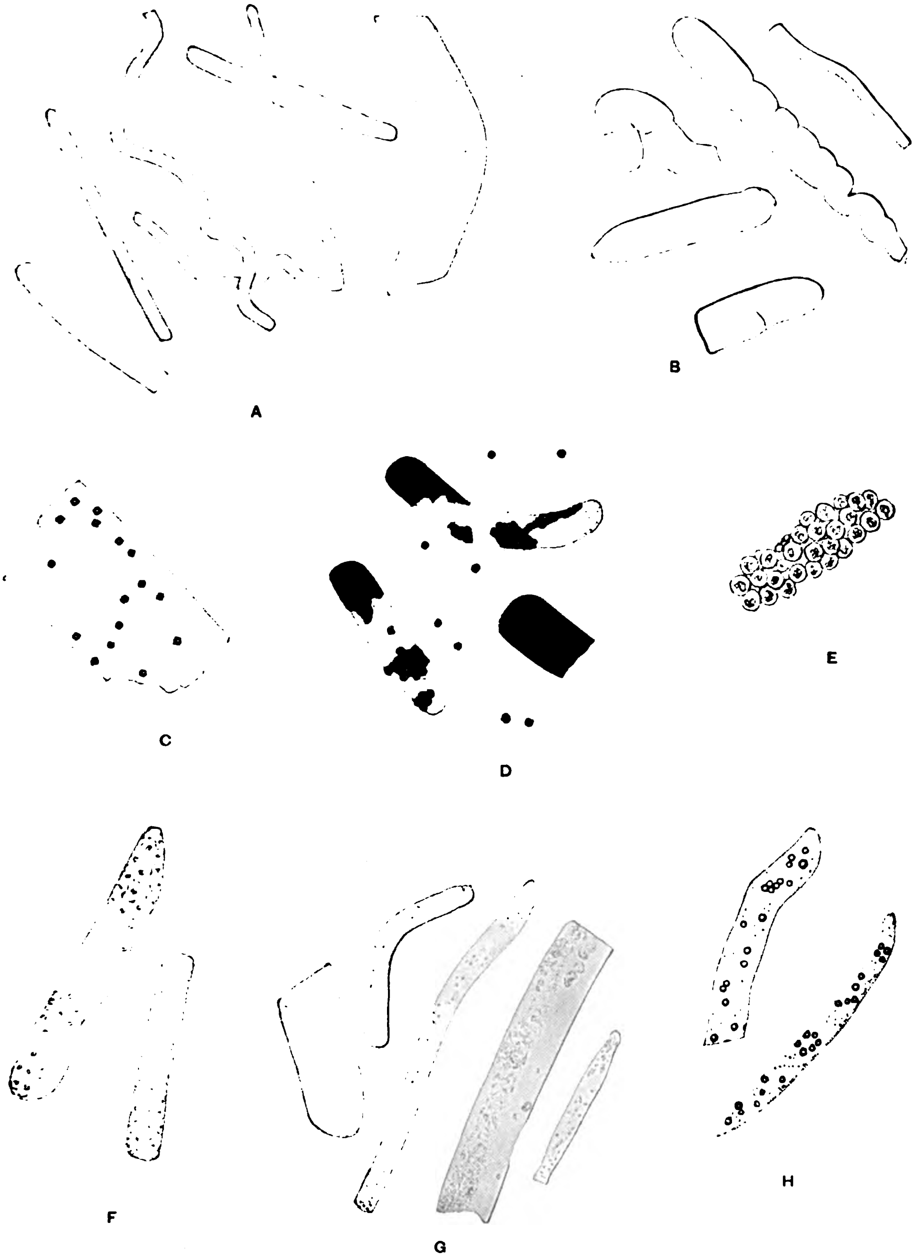
When it has been decided that there are a pathological number of renal tube casts as well as albumin in the urine, it is almost certain that there is an inflammatory lesion of the kidney. The next step in the diagnosis is to decide by microscopical examination whether pus is present also; in other words, the cases may be subdivided into two main sub-groups, namely: (*A*) *Albuminuria with renal tube-casts without obvious pus*; and (*B*) *Albuminuria with renal tube casts and obvious pus*. There are border-line cases in which leucocytes are present in excess, and yet not in sufficient numbers to constitute pus; other points about such a case will generally lead one to decide whether it comes in the apyuric or in the pyuric group. The differential diagnosis of the latter is discussed under PYURIA (p. 574), so that it only remains here to discuss:—

(*A*) *The Differential Diagnosis of Albuminuria with Tube-casts without Obvious Pus.*—The causes of this condition may be classified as follows:—

1. **The Various Forms of Bright's Disease:—**
    - (*a*) A primary acute nephritis.
    - (*b*) An acute exacerbation upon an underlying chronic nephritis.
    - (*c*) Chronic nephritis of young people: (i) Arising out of a known attack of acute nephritis; (ii) Arising without any known previous attack of acute nephritis.
    - (*d*) Chronic nephritis of old people: (i) cirrhosis of the kidneys; (ii) Arterio-sclerosis.
    - (*e*) Cystic disease of the kidneys.
2. **Nephritis of Pregnancy.**

PLATE 1

RENAL TUBE CASTS



A, Hyaline casts. B, Waxy casts. C, Hyaline cast containing small crystals of calcium oxalate. D, Blood casts. E, A leucocyte cast. F, Epithelial casts. G, Granular casts. H, Fatty casts.

3. **Chronic Ascending Nephritis,** leading to scarred contracted kidneys, the result of
    (*a*) Obstruction to urine outflow by :—
        (i) Urethral stenosis.
        (ii) Enlarged prostate.
        (iii) Displacement of the womb.
        (iv) Fibromyoma, ovarian cyst, or other pelvic tumour.
        (v) Pregnancies.
        (vi) Undue mobility of the kidney and kinking of the ureter.
        (vii) Rarities, such as abdominal aneurysm obstructing a ureter.
    (*b*) Irritation ascending from the pelvis of the kidney, the result especially of calculus, but also sometimes of chronic tuberculous lesions.
4. **Lardaceous Disease of the Kidneys.**
5. **Infarction of the Kidneys,** especially when the result of embolism in cases of fungating endocarditis; but also due to thrombosis, as in some blood diseases.
6. **Thrombosis** of the inferior vena cava involving the renal veins.
7. **New Growth of the Kidney,** some cases.

In many cases the diagnosis soon becomes obvious, but in some there may be great difficulty. The two following may serve to illustrate how such difficulties may arise:

A patient of middle age, who had not been strong for a long time, began to suffer from œdema of the ankles, which increased rapidly and spread to her legs, thighs, genital organs, and back. Within a few days her abdomen began to swell, and she began to pass very little water, the colour of blood. Upon examination the urine had a sp. gr. of 1030, was loaded with albumin and blood, and microscopically there was an abundance of red corpuscles, renal epithelial cells, leucocytes, and epithelial, fatty, granular, and blood casts, without pus, crystals, or bacteria. It seemed almost obvious that she must be suffering from acute Bright's disease; but there was no œdema of the eyelids, and there was definite enlargement of the left supraclavicular lymphatic gland; the discovery of the latter led to a very careful examination for malignant disease; and a latent and quite unsuspected carcinoma of the rectum was found. The diagnosis was carcinoma recti, secondary deposits in the retroperitoneal glands, obstruction and thrombosis of the inferior vena cava and of the renal veins, with consequent albuminuria, hæmaturia, and renal tube-casts from asphyxial nephritis, simulating acute Bright's disease.

Another case was that of a girl of 16, suffering from increasing anæmia, shortness of breath, œdema of her ankles and face, and slight pyrexia. The heart was a little enlarged, and there were soft systolic bruits that were regarded as secondary to the anæmia. The urine contained blood and albumin, with renal epithelial cells and tube-casts in abundance. Ascites developed, with increasing general œdema; there were also retinal hæmorrhages and neuro-retinitis. The diagnosis of acute nephritis, however, was only in small degree correct; for she was really suffering from malignant endocarditis of a subacute type, the nephritis being due to infected emboli of the kidney producing inflammatory changes around multiple renal infarcts.

These cases will serve to show how it may be impossible to arrive at a correct diagnosis except by thorough examination of all the systems, by watching the case carefully, and by repeating the full systemic examination at intervals. We will now deal with the headings in the above table in their reversed order.

If there is New Growth in a kidney the number of renal tube-casts is likely to be small; sooner or later a microscopic fragment of new growth may be detected in the centrifugalized urinary deposit. Albuminuria will not be extreme unless the renal veins and the inferior vena cava become involved (*Fig.* 804, p. 750), the same applying also to the œdema of the legs and trunk; hæmaturia is likely to occur at intervals, the attacks being separated by many weeks sometimes, and being relatively painless; there may be an increasing renal tumour; cystoscopic examination may show blood-stained urine (*see Plate XV, Fig. A*, p. 282) coming from one ureter only; and finally, when suspicion of new growth has been aroused, laparotomy may be indicated and the diagnosis confirmed thereby.

Thrombosis of the Renal Veins and Inferior Vena Cava has been referred to above as a condition that may simulate acute nephritis. Points to lay stress on in arriving at the diagnosis are: (1) To make a very careful and systematic examination, including that of rectum and vagina, in order not to miss anything, such as some latent growth whose secondary deposits are obstructing the veins; (2) To enquire carefully into the history—many cases of inferior vena caval thrombosis are due to extension upwards from iliac or saphenous clots, in which case there will nearly always have been swelling of one leg only to start with, followed later by extension to the back and to the other leg; (3) To note that although the œdema of the legs and back may be extreme, there is a definite upper level to it and no swelling of the eyelids or scalp; and (4) To note that if there are any distended or varicose veins upon the abdominal wall (see VEINS, VARICOSE ABDOMINAL, *Fig.* 303, p. 749), the current in them has become reversed—to being from below upwards instead of from above downwards.

Infarction of the Kidneys may be either embolic or thrombotic. The commonest cause of embolic renal infarction is fungating endocarditis. Each embolus gives rise to the sudden appearance of blood in the urine which may have contained none previously, or to increase in any existent hæmaturia; there may or may not have been a sudden pain in the back at the same time. Around each infarct acute nephritis develops, so that in some cases all the characters of the latter malady may be superposed upon those of the fungating endocarditis. If the patient is already known to have heart disease the diagnosis is easy enough; the difficulties arise in cases in which, notwithstanding the endocarditis, there is no bruit. If fungating endocarditis is suspected, the points that confirm the diagnosis are those mentioned on p. 34.

Thrombotic infarcts are less severe in their effects; they may produce no hæmaturia at all, and the albuminuria may be slight, and unaccompanied by tube-casts. They generally arise in cachetic conditions, or in blood diseases such as leukæmia or pernicious anæmia, in which cases the diagnosis will be arrived at on other grounds, albuminuria not being the prominent feature of the case.

Lardaceous Disease of the Kidneys used to be common in the days of septic surgery, but it is uncommon now. It is a risky diagnosis to make, therefore, unless there is some obvious cause for it, such as long-standing suppuration in association with a spinal, hip-joint, or empyema sinus, bronchiectasis, phthisis with cavitation, or the like; or clear evidence of tertiary syphilis with cachexia. There is nothing characteristic about the urine. In the earlier stages there may be but a trace of albumin in an otherwise normal urine; later, the albumin increases and it may reach very large amounts, such as 20 parts per 1000, casts being very few in proportion, the total amount of urine increased, its colour pale, and its sp. gr. low—1005 to 1012; later still, possibly as the result of superposed nephritis, the amount of urine falls until only a few ounces may be passed each day, of high colour and sp. gr. 1020 to 1035, loaded with albumin, and now containing hyaline, waxy, granular, fatty, and epithelial casts. Lardaceous casts may or may not occur, but they are not diagnostic, for they have also been found in cases of nephritis without lardaceous disease. Indeed, the diagnosis of lardaceous kidney resolves itself into one of guesswork in a case in which there has been prolonged suppuration or severe syphilis to give rise to it, and in which there may be smooth firm enlargement of the liver, moderate enlargement of the spleen, and more or less severe diarrhœa, to indicate corresponding lardaceous change in the other organs that are generally affected at the same time as the kidneys.

Chronic Ascending Nephritis arises from precisely the same causes as acute ascending nephritis or surgical kidney, and probably results from recurrent focal inflammations which heal, with the result that, in the course of months or years, the kidneys are converted into a mass of irregular fibrotic scars which together produce the same local and general changes and effects as are found in cases of ordinary red granular contracted kidney. It is important to bear in mind that any cause of prolonged obstruction to the urine outflow may cause granular kidney with albuminuria, without pus but with casts, in a pale abundant urine of low specific gravity. The diagnosis will generally be obvious when the obstruction is due to urethral stricture; it is more apt to be overlooked in other cases, though if one bears in mind the causes mentioned in the list above, the methods of diagnosis will generally be clear. One would only mention in particular that uterine tumours or displacements are a very common cause for slight albuminuria and a few renal tube-casts in women; and that in men of sixty and over enlargement of the prostate causes a precisely similar condition long before there is any definite pyuria.

Pregnancy Nephritis is sometimes spoken of as though it were altogether different from nephritis of the Bright's disease type in general. I do not subscribe to this view. I hold that Bright's disease has many different causes and many different types. It may be due to scarlet fever, in which case it is very possibly streptococcal; it may be due to pneumonia or empyema, in which cases it may be pneumococcal; it may be due to various other micro-organisms; it occurs in some cases of cholera, and in severe secondary sypnilis; it is frequent in malaria, especially the quartan type; it may be due to chemical substances such as turpentine, cantharides, or oxalic acid; it very often seems to come on from no known cause at all, though in such cases there must be a microbial or other cause that is not discovered; it may be due to pregnancy, in which case it is ascribed to unknown toxins. In all these cases the types of reaction on the part of the kidney are similar, and one can only regard pregnancy nephritis as a variety of non-suppurative nephritis in general. Very likely it is only a matter of degree whether it is non-suppurative or merges into the type in which there is pyuria as well as albuminuria—pyelitis of pregnancy. Pregnancy may cause a primary acute nephritis, which may recover either completely, or but partially and persist as chronic nephritis; or may seem to recover when in reality it is merely latent, or even slowly and insidiously progressive; it may produce what seems to be a primary acute nephritis which is really but an exacerbation superposed upon a chronic nephritis that has been unsuspected; and very possibly it may produce nephritic changes which are not associated with definite symptoms at the time, but which ultimately result in what is spoken of as chronic interstitial nephritis. When, therefore, albuminuria with renal tube-casts, but without pyuria, occurs during pregnancy, it matters little what name is given to the condition, provided it is realized that just the same difficulties offer themselves here as in Bright's disease in general, in arriving at a conclusion as to whether the renal lesion is acute, chronic, or acute on chronic.

Various Forms of Bright's Disease.—Of all these, the hardest to diagnose with certainty is *primary acute nephritis* in the adult. The majority of adult cases that are labelled acute Bright's disease are really suffering, not from primary acute nephritis, but from an acute exacerbation upon the top of already existent but possibly latent chronic nephritis. The difficulty is to arrive at the diagnosis between these two, particularly since many of the points mentioned in text-books as occurring in acute nephritis are really due, not to the acute attack, but to the subacute or chronic renal lesion which has, until then, been unsuspected.

The best examples of primary acute nephritis are to be seen in cases that are already under observation for some other disease, notably scarlet fever or lobar pneumonia. Sometimes the onset of the nephritis is indicated by general œdema, especially of the eyelids and face, ankles, genital organs, and loins; but it cannot be insisted upon too strongly that œdema is not essential, many cases of acute nephritis having no œdema at all, especially if the patient is already in bed when the kidney inflammation begins, as in scarlatina cases. If the urine were not examined the renal lesion would often escape recognition altogether; and there can be no doubt that many cases of primary acute nephritis do escape recognition in this way, coming under observation later when they present symptoms of chronic nephritis, or an acute exacerbation on chronic nephritis.

The essential point in the diagnosis is urine examination. According to the severity of the nephritis there will be more or less diminution in the total daily quantity; it is common for less than 20 oz. to be passed in the twenty-four hours, and often the amount falls to 10 oz., 5 oz., or even to none at all for a while. The specific gravity is raised to 1025, 1030, or even to 1035, but rarely to 1040. The reaction is generally acid at first, but it soon becomes alkaline on standing. The colour is extremely variable, according as little or much blood is present; sometimes it is almost normal or merely that of a concentrated urine; more often there is some tinging with blood, varying from bright red to brownish, brown, brown-black, or to that peculiar blackish tint which is described as smoky. There is a general cloudiness of the specimen, and on standing it deposits a heavy sediment which often has a dark brownish tint owing to the phosphates carrying the blood pigment with them. Microscopically, the centrifugalized deposit consists partly of amorphous débris due to earthy phosphates, and to the disintegration of cells and tube-casts; and one expects to find an abundance of red corpuscles, renal epithelial cells, variable numbers of epithelial, fatty, granular, hyaline, and blood-casts, an excess of leucocytes, an occasional crystal of calcium oxalate or uric acid, and irregular granular masses which are not definitely tube-casts. It is noteworthy, however, that in the very acute stages there may be no tube-casts, though shed renal epithelial cells are abundant; in such a case tube-casts will show themselves in a few days. It is important that each specimen should be examined as fresh as possible, owing to the tendency of casts and cells to disintegrate on standing. In addition to red corpuscles there is often much free hæmoglobin; the tincture of guaiacum test will be positive, and the spectroscope will show the bands of oxyhæmoglobin or of methæmoglobin. Coagulable proteid is generally present in abundance, the proportions of globulin and albumin varying greatly, but together amounting to anything between 2 and 20 parts per thousand—often about 15 parts per thousand at first, rapidly dropping to less after the first few days of treatment, until at the end of from a fortnight to a month it may be 1 part per thousand or less, or even absent altogether. In a few cases, however, there is very little coagulable proteid but an abundance of albumose, so that the boiling test gives but a faint cloud, whilst the nitric acid test yields a dense white ring, soluble on warming, to reappear on cooling. There is generally an excess of nucleo-proteid also. The urea, chlorides, and phosphates all fall below the normal totals, though their percentages may be increased if the urine is very concentrated.

With this condition of urine there will be little doubt as to the presence of acute nephritis; the only question then is whether it is primary, or an exacerbation upon chronic nephritis. The former is probable if it is known that the urine was free from albumin up to the time of the attack, if the patient is known to have suffered recently from scarlet fever, pneumonia, diphtheria, secondary syphilis, or some other similar fever; if the heart is of normal size and its sounds natural, the blood-pressure natural, and the retinæ healthy. It may be that the patient himself may have been exposed to scarlatinal infection, and without having had the rash may develop nephritis; the association of peeling of the skin, or recent sore throat with enlarged glands in the neck, or otitis media, might suggest the diagnosis in these mild cases of scarlatina, though sometimes acute nephritis in a child may be the sole evidence of the disease. The course of the malady will also assist the diagnosis; the albuminuria of primary acute nephritis may clear up entirely in from a fortnight to six weeks, though in unfavourable cases it persists and chronic nephritis develops out of the acute. If, on the other hand, it is found that, in a case of apparently recent acute nephritis, with general œdema, hæmaturia, and the other urinary changes described above, there is cardiac hypertrophy, with a prolonged lumpy first sound at the impulse, a ringing aortic second sound, a blood-pressure of more than 150 mm. Hg, and possibly albuminuric retinitis, the probability is that the acute nephritis is not primary, but an acute exacerbation of an unsuspected chronic nephritis. There is often a history of former scarlet fever or of syphilis in such cases; the patients may be of any age, from childhood to past middle life. If the patient survives, one or other of two conditions usually results: either the albuminuria, the scanty urine, and the tube-casts persist, whilst the patient remains waterlogged until the end comes in a few weeks or months, or else the acute exacerbation subsides and the clinical characters of chronic nephritis remain.

Some of these cases, but by no means all of them, are examples of primary acute nephritis, persisting and becoming chronic. It must, however, always be very difficult, and indeed almost a matter of opinion in many cases, to decide whether a patient is suffering from a chronic nephritis which is the result of a primary acute nephritis that has not cleared up, or from a chronic nephritis which was present but unrecognized before an acute exacerbation drew attention to it; my own view is that many cases in which young adults seem to develop acute nephritis from no more definite cause than exposure to damp or cold, are really examples of acute on chronic, and not of primary acute, Bright's disease. The albuminuria in these cases does not clear up, and it is a mistake to restrict the diet or the daily occupation after the acute exacerbation has subsided. In spite of the persistence of albuminuria, these patients do best if they are given iron and allowed to go about their ordinary avocations; they have diseased kidneys, and they will not live many years, but there is no need to adopt treatment which constantly reminds them of the fact. As the acute exacerbation subsides, the amount of urine rises rapidly to 60 or 70 oz. or more per diem, and remains increased even after all œdema has passed away; the specific gravity falls to 1012, 1010, or 1008; the albumin persists to the extent of anything between 0·5 and 8 parts per thousand; blood is absent, though an occasional red corpuscle may be seen under the microscope; and there are moderate numbers of hyaline, granular, or even fatty casts, with an occasional renal epithelial cell.

It happens not infrequently that a young patient suffering from *chronic nephritis* comes under observation for shortness of breath, palpitations, anæmia, or for inflammation of one or other of the serous membranes, without ever having had any symptoms of acute nephritis at all. The kidneys that would be found in such cases differ from the granular contracted kidneys of older people in that they are pale instead of red. They are pale granular contracted kidneys, precisely similar to those which may result from a long antecedent acute nephritis that has not entirely cleared up. When they develop without any known preceding attack of acute nephritis they have been referred to as Rose-Bradford kidneys. It is by no means impossible that they are really the result of a preceding acute nephritis which escaped recognition because there was no œdema to attract attention to the need for urine examination. The patient may be of any age, though generally between five and thirty-five. There may be no sign of anything wrong until acute uræmia, with convulsions, leads to rapid death. On the other hand, in a typical case, in addition to the urine changes mentioned above, one expects to find some of the following symptoms or signs; a great increase in the size of the left ventricle, as evidenced by displacement of the impulse downwards and outwards, even into the sixth left intercostal space below or outside the left nipple, with increase of the precordial impairment of resonance outwards to the left without corresponding increase upwards or to the right; a ringing second sound in the second right intercostal space close to the sternum, and a prolongation of the first sound at the impulse, or its replacement by a localized blowing systolic bruit; more or less anæmia, sometimes very considerable and of the chlorotic type; a maximum systolic blood-pressure, of 175 mm. Hg, or more, sometimes over 300 mm. Hg, even when the pulse feels comparatively soft to the finger; albuminuric retinitis; a tendency to hæmorrhages, especially to epistaxis; headache; insomnia; breathlessness on exertion; and inability to work with the usual energy, either mentally or physically.

The *chronic nephritis of old people* is diagnosed more often than it exists, if one understands by it the disease associated with small red granular contracted kidneys. On the other hand, the kidneys of most old people exhibit a certain amount of interstitial fibrosis, with occasional retention cysts and some granularity of the surface when the capsules are stripped off, without there being any material diminution in their size. Where senile changes that are almost normal end and chronic interstitial nephritis begins, is difficult to determine. The same applies to *arteriosclerosis* and the renal changes associated with this. Some regard arteriosclerosis and chronic interstitial nephritis as essentially different maladies; others regard the arterial as secondary to the renal changes; others hold that arteriosclerosis leads to a variety of red granular kidney that is not the same as the red granular contracted kidney of chronic interstitial nephritis; whilst others again favour what seems a likely view, namely that arteriosclerosis and sclerosis of the kidneys both have common causes, and that it is more or less an accident whether the patient, on post-mortem examination, presents more arterial or more renal changes, or about the same degree of both. During life the differential diagnosis between them is sometimes impossible. In either case there will be a hypertrophied left ventricle, a loud lumpy first sound, or a blowing systolic bruit, at the impulse, a markedly accentuated aortic second sound, a systolic blood-pressure somewhere between 150 and 320 mm. Hg, with a tendency to shortness of breath; giddiness, especially on sudden change of posture; singing in the ears; difficulty in concentration of mind; and very possibly cardiac symptoms, varying from a mere consciousness of the existence of the heart, to precordial pain of varying severity, or even extreme heart-failure, with œdema of the legs, ascites, nutmeg liver, orthopnœa, and pulmonary congestion. In the latter case the great difficulty will be to decide whether the heart failure is due to primary renal or arterial, primary cardiac, or to primary pulmonary disease, and the only sure methods of deciding that there is a renal lesion are: the discovery of more than an occasional granular and hyaline tube-cast in the urine; the detection of albuminuric retinal changes; and instrumental determination that the blood-pressure is much raised. Sometimes inflammation of one of the serous membranes is the first symptom; subacute or chronic peritonitis with ascites; pericarditis; or pleuritic effusion. On the other hand, the patient may seem to have been in robust health until the nature of the case is suggested by a sudden apoplectic seizure due to cerebral hæmorrhage. In yet another group of cases the malady is discovered accidentally as the result of examination for life insurance. It is not very uncommon to find glycosuria as well as albuminuria, the sugar occurring in a urine of normal specific gravity without any associated acetone or diacetic acid. The degree of albuminuria is very variable; when there are signs of cardiac failure there may be oliguria with much albumin and not a very large number of casts; when there is no heart failure there is generally polyuria, the patient having to rise several times in the night, passing from 60 to 120 oz. of pale urine in twenty-four hours, of sp. gr. 1008 to 1012, often containing only a trace of albumin, and even that not constantly; there are intermediate cases in which the amount of albumin varies from 0·25 to 4 or 5 parts per thousand. Upon the whole one may say that, if the increased albuminuria due to heart failure on the one hand, or to a super-added acute attack of nephritis on the other, can be excluded, the more the disease approaches the type of red granular contracted kidney, the more likely is the albumin to be small in amount and intermittent; whilst the more the disease approaches in type to arteriosclerosis with renal changes on the one hand, or to pale granular contracted kidneys on the other, the greater will be the amount of albumin, if any is present at all. There will be tube-casts, chiefly granular and hyaline, most numerous with pale granular contracted kidneys, fewest with arteriosclerosis, and intermediate in numbers with red granular contracted kidneys. It need scarcely be added that the absence of albuminuria does not exclude arteriosclerosis; but we are here dealing only with cases in which albuminuria occurs.

Cystic Disease of the Kidneys is found in three entirely different types of patients, namely, (1) the new born, (2) the young, and (3) the elderly. In the new born the main symptom is abdominal distention, which may be so extreme as to have caused difficulty in delivery; the bilateral cystic tumours can be felt, and the diagnosis in such cases is not difficult. Minor degrees escape detection at birth, and it may be that several years elapse before the diagnosis is arrived at as the result of finding bilateral uneven renal tumours associated with the passage of abundant pale urine of low specific gravity containing traces of albumin, a few granular and hyaline tube-casts, and an occasional red corpuscle. Sometimes a sudden and severe attack of hæmaturia is the first symptom in the case. The discovery of bilateral irregular renal tumours is the clinching point in the diagnosis. In at least one case they were so large as to meet in the middle line, so that a loop of intestine that had passed between and behind them could not get out again, and the patient came under observation for acute intestinal obstruction. The third type of cystic disease of the kidneys occurs in old persons, and is but a variety of chronic interstitial nephritis in which the agglomeration of retention cysts has reached an extreme degree; the enlargement of the kidneys is then much less than it is in young persons, where the lesion is probably congenital; the symptoms and urinary changes are precisely similar to those already described in cases of red granular contracted kidneys.

(*B*) *Albuminuria with Renal Tube-casts and with Pus.*—When pus is present in the urine along with albumin and renal tube-casts, the differential diagnosis resolves itself into that of pyuria that is partly or wholly of renal origin (see PYURIA, p. 574). It only remains to add: first, that it is not sufficient to rely upon the naked-eye characters of the urine, or upon chemical tests, in excluding minor degrees of pyuria; microscopical examination of the centrifugalized deposit is essential, especially in the detection of acute pyelitis and pyelonephritis the result of coli-bacilluria in children, pregnant women, and others (p. 69); secondly, that the amount of albumin actually due to pus itself is small, so that if there is any measurable quantity of albumin present it indicates that the kidneys are themselves affected, this being further confirmed when casts are also found; and thirdly, that blood, like pus, is in itself responsible for relatively little albumin, so that when there is considerable albuminuria associated with blood, there is strong ground for believing that the albumin is by no means all due to the blood. The presence of very small quantities of blood does not assist the differential diagnosis of the cause of albuminuria so much as might be expected; much blood indicates that the cause is due to one or other of the conditions discussed under HÆMATURIA (p. 275).

II.—ALBUMINURIA WITHOUT TUBE-CASTS.

Turning now to albuminuria without tube-casts, one would emphasize the fact that more than one microscopical examination may be required, for if the urine is alkaline, or has stood for any length of time, casts, originally present, may have become unrecognizable; besides which, even with definite nephritis, there may be very few casts at one time, many at another. This applies particularly to the very acute cases on the one hand and the very chronic on the other. Assuming that not more than a very occasional cast is found, the chief conclusion that can generally be drawn is that the albuminuria is not indicative of organic renal disease. The cases may then be subdivided into: (1) *Those in which the urine presents some other definite abnormality besides albuminuria,* especially (*a*) pyuria, (*b*) hæmaturia, (*c*) hæmoglobinuria, or (*d*) glycosuria; (2) *Those in which, were the albumin removed, the urine would be normal.*

1. These cases need not be discussed further here; the differential diagnosis will be found under PYURIA, HÆMATURIA, HÆMOGLOBINURIA, and GLYCOSURIA respectively.

2. These are clinically of importance in that, until the absence of casts has been determined, the absence of organic renal changes cannot be concluded. Even when casts are absent, a trace or a small amount of albumin may be the first evidence in elderly persons of enlargement of the prostate, chronic interstitial nephritis, or arteriosclerosis; or in younger persons of chronic ascending nephritis, the result of such things as former gonorrhœa, repeated pregnancies, uterine prolapse or other displacement, chronic vesical catarrh, or urethral stricture. The chronic effects on the kidneys of interference with the urine outflow are apt to be overlooked, though if they are borne in mind they are generally easy of diagnosis.

The following are a number of other conditions which may cause slight degrees of albuminuria without tube-casts, but which are obvious, or else diagnosed by other signs that are discussed elsewhere: burns, scalds, chronic alcoholism, cirrhosis of the liver, diabetes mellitus, exophthalmic goitre, gout, lead-poisoning, mumps, secondary syphilis, morphinism, mercurialism, vasomotor neuroses such as Raynaud's disease or angioneurotic œdema, obstruction to the vena cava inferior by thrombosis or by external tumours, the pressure of considerable ascites, ovarian cysts or solid tumours, pernicious anæmia, Hodgkin's disease or lymphadenoma, lymphosarcoma, lymphatic or splenomedullary leukæmia, splenic anæmia, pemphigus, phosphorus poisoning, chronic arsenical poisoning, pregnancy, severe anæmia the result of syphilitic, malarial, malignant, tuberculous, or phthisical cachexia, ankylostomiasis, or infection with other parasites such as *Bothriocephalus latus* or *Trichina spiralis.*

There remain three other groups of conditions in which albuminuria and its differential diagnosis are often important, and these are: (1) *Febrile conditions*; (2) *Heart-failure conditions*; and (3) so-called '*Physiological*' *albuminuria of adolescence.*

Febrile Conditions.—In nearly every fever there is some cloudy swelling of the parenchyma of various viscera, especially the kidneys; consequently most fevers may sometimes be associated with albuminuria, and, broadly speaking, the higher the patient's temperature the greater is the liability to it. The amount of albumin present is generally not great. We need not enumerate all the various fevers in this connexion. Suffice it to say that albuminuria is relatively common in scarlatina, diphtheria, variola, erysipelas, pyrexial phthisis, cholera, dysentery, Weil's disease, severe malaria, and yellow fever; not so common in lobar pneumonia, bronchopneumonia, typhoid fever, and empyema; and relatively uncommon in other febrile conditions, such as acute rheumatism, influenza, meningitis, measles, German measles, follicular tonsillitis, and so on. The albuminuria may, of course, be already present in a person who develops an intercurrent fever; the diagnosis then depends upon considerations mentioned above.

If, on the other hand, the albuminuria is known to have developed coincidently with the febrile illness, the chief point to decide will be whether it indicates actual nephritis or not. Many consider there is an essential difference between 'febrile albuminuria' and actual nephritis. This may or may not be so, but it is extremely difficult to be sure of the distinction clinically. It may be urged that—to take scarlet fever as an example—the albuminuria of the first few days is 'febrile,' whilst that of the second or third week is 'nephritic.' As a matter of fact, in not a few cases in which death has occurred in the first week the 'febrile' albuminuria has been associated with large mottled acute nephritic kidneys, even where there has been no œdema, no hæmaturia, and no very large numbers of renal tube-casts. Probably there are all degrees of acute nephritis, from very slight and transient, to very severe and possibly fatal; and it is a mistake to try and make a distinction in kind. The great majority of cases of albuminuria during fever recover completely; some seem to recover but come under observation years later with pale granular contracted kidneys; others die during the acute attack. The degree of albuminuria is not a direct measure of the renal changes unless the amount of albumin is large; a small amount of albumin does not necessarily indicate trivial nephritis. Absence of œdema is the rule. Microscopical examination of the centrifugalized urinary deposit is essential: the more the renal epithelial cells, red corpuscles, leucocytes, and various renal tube-casts, the more conclusively can some degree of actual nephritis be diagnosed.

When doubt lies between scarlatina and measles or German measles, or between diphtheria and other forms of sore throat, the existence of albuminuria sometimes assists in arriving at the diagnosis of scarlatina in the one case or of diphtheria in the other.

In pneumonia, albuminuria has become much less frequent since blistering with cantharides has gone out of fashion in treating this disease.

Heart-failure Conditions.—The right side of the heart may fail owing to many different causes, which may be arranged under four main headings, as follows: (*a*) Valvular disease; (*b*) Obstructive lung affections; (*c*) Myocardial affections; (*d*) Granular kidneys and other high blood-pressure conditions. Each of these main headings has many sub-headings (see ORTHOPNŒA, p. 418). Any one of them may result in albuminuria, though the amount of the latter is extremely variable, some cases of severe heart failure exhibiting no albuminuria at all, whilst others may have as much as 10 parts per 1000, or more.

The first step in the differential diagnosis is to *exclude primary renal conditions* by negative microscopical examination of the centrifugalized urine deposit for casts, examination of the retinæ, and exact determination of the blood-pressure. Curiously, even with feeble irregular pulses, such as are found in panting cases of mitral stenosis, the blood-pressure is considerably higher than normal, doubtless owing to partial asphyxia; so that merely finding a systolic blood-pressure of 150 or 160 mm. Hg is no proof of granular kidney or arteriosclerosis; sometimes, however, the reading is as high as 200, 250, 300, or even 320 mm. Hg, and then the diagnosis of one or other of the latter is almost certain.

If renal and arteriosclerotic conditions can be excluded, the diagnosis lies between the other three main groups. The cardiac bruits, the history of growing pains, chorea, or acute rheumatism, the youth of the patient, the family history of heart disease or rheumatic fever, the association of other rheumatic affections such as recurrent tonsillitis, subcutaneous nodules, or erythema, will often serve to point to *primary valvular disease*; in older patients, especially in men between forty and fifty, there may be aortic disease and a history of syphilis and not of acute rheumatism. In severe heart failure in children under puberty, the result of mechanical difficulty with the circulation, an *adherent pericardium* is generally found, and clinically, the heart is large out of proportion to the general physical signs.

When there is a definite history of recurrent winter cough in an elderly person, with a hyper-resonant and over-expanded chest, the likelihood of *emphysema and bronchitis* will at once suggest itself. Similarly *fibroid lung*, or fibroid lung and *bronchiectasis*, as a cause of heart failure and albuminuria, only needs mentioning, the diagnosis generally being obvious from the physical signs, the clubbed fingers, and in the bronchiectatic cases, the abundant intermittent, and frequently foul, expectoration.

Myocardial affections, such as fibroid, fatty, or primary alcoholic heart, are generally diagnosed by guessing at them when other causes of heart failure can be excluded. The patients are generally middle-aged, shortness of breath on exertion, precordial pain and even angina pectoris occupying a prominent position amongst their cardiac symptoms; there may or may not be a high blood-pressure, the albuminuria is not associated with renal tube-casts, there is often no cardiac bruit, or at most a more or less localized blowing systolic bruit at the impulse; at the same time the heart is clearly enlarged, and it may be beating rapidly and irregularly; there may be a history of syphilis or of chronic alcoholism; the patient may be very stout in the fatty, though generally not so in the fibroid, cases. There may be a history, either of an extremely sedentary life upon the one hand, or of over-use of the heart by strenuous hard physical work—as a blacksmith, an athlete, and so forth—on the other. Electro-cardiographic tracings may be required in determining the nature of the heart lesion.

Needless to say, the exact nature of the cardiac lesion remains obscure or uncertain in many of these cases, many a patient who really has mitral stenosis being regarded during life as suffering from chronic bronchitis and emphysema, and so on.

'Physiological' Albuminuria.—Finally, we come to the albuminuria of apparently healthy males and females between the ages of fifteen and thirty. The condition was little known until medical examinations at schools, or for life insurance, or for the services became common. It has received a number of names, of which the following are some: 'accidental,' 'essential,' 'postural,' 'cyclic,' 'orthostatic,' 'intermittent,' 'physiological,' 'functional,' 'orthotic,' albuminuria, Pavy's disease, albuminuria 'of adolescence' or 'of puberty.' It derives its chief importance from the fact that young males who suffer from it may be rejected for life insurance or for the services, from the fear that they have some form of nephritis. A similar condition occurs in females of a similar age, but it is detected less often than in males because one has less occasion to examine the urines of healthy girls than is the case with boys and youths. Collier and others have thrown much light upon the nature of the affection by their investigations upon the urines of rowing men. It is found that the urine passed just before a boat-race being free from albumin, that voided immediately after is generally loaded with it. A few hours later this albuminuria is gone again. Now university oarsmen are, upon the whole, long lived, hence this recurrent albuminuria cannot matter in them; and the same applies to the albuminuria of many adolescents. A prominent feature of such a case is that the urine first voided in the morning is quite normal, whilst that passed later in the day may contain anything from a trace to five parts per thousand of albumin; the more the youth has exerted himself physically by walking or otherwise, and the more he has exposed himself to cold, for instance during a train journey to the city on a winter's day, or in a cold bath, the greater is the liability to this unimportant but possibly alarming albuminuria. Some youths may pass albumin for days together before an interval of freedom from it occurs. Sometimes they appear to be in robust health, sometimes they look a little pale, as though they had been overworking at an indoor occupation; they may be nervous, but often they are not. A natural nocturnal emission is supposed to predispose to albuminuria next day; so also is a diet which includes eggs, especially raw eggs. The point is that these individuals have to be differentiated from sufferers from Bright's disease. The method of diagnosis is as follows: a complete routine examination is carried out, and no obvious affection of the heart or other viscera is detected; the blood-pressure is normal; the albumin having been discovered, the patient is directed to supply a series of samples, at intervals of a few days, and preferably passed immediately after rising in the morning. If all samples contain albumin it will be very difficult to exclude organic disease; if some contain albumin in abundance, however, and others none at all, the presumption will be that it is 'functional'; before being finally satisfied, however, it is important that a careful microscopical examination of the centrifugalized deposit from a specimen containing albumin should be made, no casts or other abnormal constituents being found. The administration of calcium chloride or calcium lactate greatly diminishes the tendency to this form of albuminuria. In an adolescent male who has no symptoms, albuminuria discovered accidentally, present after exertion or after exposure to cold, but absent after rest in bed, and when present not associated with renal tube-casts or with signs of arterial, cardiac, or other disease that should be detected by physical examination, is almost certainly 'physiological,' needing no treatment and not indicative of any underlying disease.

Herbert French.

ALBUMOSURIA may be discussed under two main headings, namely: (1) *Ordinary Albumosuria*, which is not uncommon but is of little clinical importance; and (2) *Bence-Jones Albumosuria*, which is rare but is clinically important.

Ordinary Albumosuria is seldom recognized because the albumose generally occurs along with albumin, and is not detected until this has been removed by acidulating with acetic acid, boiling thoroughly, and filtering. Albumose may be recognized in the filtrate by the fact that with Heller's nitric acid test it gives a white cloud which disappears on warming, to reappear on cooling; and its presence may be confirmed by the violet-red colour it gives with the biuret test, which consists in adding excess of caustic soda to a drop of dilute copper sulphate solution, adding this mixture in drops to the urine, from which all albumin has been removed, and warming. Another test for albumose is Hofmeister's, which consists in acidulating the urine with acetic acid and then adding phosphotungstic acid; albumoses give a milky cloud with the latter. The deutero-albumose that gives these tests occurs in the urine under a great variety of circumstances: apparently the one essential factor is cell destruction within the body. It will suffice to mention some of the many diseases in which it has been found:—

(*a*) '*Febrile*' *Albumosuria*: in severe infective fevers, such as typhoid, scarlet, small-pox, measles, acute rheumatism, lobar pneumonia.

(*b*) '*Pyogenic*' *Albumosuria*: in empyema, phthisis with cavitation, bronchiectasis, appendicular subdiaphragmatic or hepatic abscess, suppurating gall-bladder, pyosalpinx, suppurative periostitis, arthritis or osteomyelitis, gangrene of the lung, gangrene of the leg, breaking-down cancer, acute peritonitis.

(*c*) '*Hepatogenous*' *Albumosuria*: in cancer of the liver, cirrhosis, catarrhal jaundice, phosphorus poisoning, acute yellow atrophy, infective cholangitis, suppurative pylephlebitis.

(*d*) '*Alimentary*' *Albumosuria*: in cases of gastric or duodenal ulcer, carcinoma of the colon or stomach, ulcerative colitis, tuberculous ulceration of the bowel, acute and chronic dysentery.

(*e*) '*Hæmatogenous*' *Albumosuria*: in leukæmia, scurvy, purpuric conditions, and with internal hæmatomata, such as pelvic hæmatocele.

(*f*) '*Albuminuric*' *Albumosuria*: many cases of acute nephritis, syphilitic, cardiac and other forms of albuminuria, are associated with albumosuria. There is some doubt, however, as to whether the reagents employed in the qualitative analysis do not themselves convert some of the albumin into albumose.

(*g*) *Albumosuria due to unclassified causes*: such as pregnancy, especially if the fœtus has died, though sometimes even without this.

The amount of albumose present in any of the above conditions is seldom large, and diagnostically it has little if any significance except when it occurs apart from albumin. Even then its main importance lies in the necessity of not mistaking it for albumin. This error would only arise with the nitric acid test, for albumose does not form a cloud on boiling with acetic acid. It is urged by some that albumosuria in appendicitis points to abscess rather than to simple inflammation; that in a pleuritic case it points to empyema rather than to serous effusion; that in a meningitic case it points to the suppurative or epidemic cerebrospinal forms rather than the tuberculous; and so on; but it is very doubtful if the symptom can carry so much weight as this. In a given case the presence of ordinary albumosuria points to a graver prognosis upon the whole than if no albumose were present, but it is not particularly helpful in differential diagnosis.

Bence-Jones Albumosuria, on the other hand, though rare, is clinically important. The nature of the proteid present is still undecided; it certainly is not ordinary albumose. Its most striking characteristic appears when the urine is warmed after acidulation with acetic acid to prevent precipitation of phosphates: long before the urine boils a dense milky precipitate appears, suggesting at first sight either phosphates or coagulated albumin; it attracts attention at once from the fact that on further warming it begins to clear up again, and after boiling it almost or completely goes. It will be realized that the precipitate cannot be albumin or phosphates, for not only would neither of these clear up at boiling-point in this way, but also the acidulation of the urine has been sufficient to prevent phosphates from coming down, whilst the temperature at which the dense sticky precipitate appears (about 60° C.) is far lower than that at which albumin coagulates. If any albumin is present at the same time the clearing at boiling-point will be but partial; the albumin should then be removed by boiling and filtration, when nitric acid added to the filtrate will give a white ring which redissolves on warming, to reappear on cooling, like that of albumose. This Bence-Jones proteid, when present, generally occurs in much larger amounts than ordinary albumose ever does, so that it is seldom overlooked unless it is mistaken for albumin. It may amount to anything between 1 and 20 parts per thousand, or more. It may be present on some days and not on others. It indicates, almost with certainty, that there is some affection of the bone-marrow; it might be due, for instance, to secondary deposits of malignant disease in bones, or to leukæmia; but in the great majority of cases it has occurred in connection with multiple myelomata—Kahler's disease or myelopathic albumosuria of Bradshaw. Unless there is other evidence to the contrary, abundance of Bence-Jones proteid in the urine indicates multiple tumours involving the bone-marrow. *Herbert French.*

ALKAPTONURIA.—(See Urine, Abnormal Coloration of, p. 746.)

ALLOCHEIRIA—Literally means 'other handness.' It sometimes happens that when a patient is touched upon, say, the back of his *right* foot, and is then asked where he has been touched, he says, "Upon the back of my *left* foot." This reference of sensations to exactly corresponding parts of the limbs or body on the wrong side is known as allocheiria. Experiments have shown that complete allocheiria results from transverse hemisection of the spinal cord. It seems that sensory impulses travel much the more readily up their own side of the cord, but can also pass by the opposite side if necessary; when they are compelled to do so, the brain interprets them as coming from that side of the body which usually sends impulses up this particular side of the cord. When a patient exhibits allocheiria, therefore, it generally indicates that there is a lesion affecting one side of the spinal cord, or the upward extensions of the tracts which convey sensory impulses from the cord to the brain, more than the other. It is necessarily a rare symptom. It might result from a stab or a bullet wound damaging the cord unilaterally; or from a gumma or neoplasm of the spinal meninges; it may be functional; rarely it may result from the cord becoming compressed more on one side than on the other by spinal caries, a new growth, callus, or a fracture-dislocation; and occasionally it may be noticed when there is a cord disease which, though usually bilateral, happens to have advanced more rapidly on one side than upon the other, as in exceptional cases of disseminated sclerosis, locomotor ataxy, or softening from syphilitic endarteritis and thrombosis. Except in functional cases, allocheiria will seldom be the only, or even the chief, feature in the case; paresis, pain, or some other symptom present will afford greater assistance in the diagnosis than will the allocheiria itself. *Herbert French.*

ALOPECIA.—(See Baldness, p. 70.)

AMAUROSIS.—(See Vision, Defects of, p. 759.)

AMBLYOPIA.—(See Vision, Defects of, p. 759.)

AMENORRHŒA.—The time at which menstruation first appears is very variable within certain limits, being influenced largely by climatic and racial peculiarities; in this country about fourteen may be taken as the average. When the menstrual flow has not become established it is usual to speak of primary amenorrhœa, whilst cessation of the flow after it has once been regularly established is known as secondary amenorrhœa. From the table of the causes of amenorrhœa below, it will be seen that some of them must of necessity give rise to primary amenorrhœa, whilst others more commonly produce the secondary variety. In investigating a case, therefore, it is important to ascertain first whether the condition is primary or secondary, and next whether it is real or only apparent. The latter condition, known as cryptomenorrhœa, implies that the menstrual flow takes place but is unable to escape externally because there is some closure of a part of the genital canal. The congenital form of cryptomenorrhœa is the only variety met with at all commonly, acquired closure of a part of the genital canal being exceedingly rare. Stenosis of the vagina is not uncommon as a result of injury and infection, but a small sinus is usually left which suffices for the escape of the menstrual fluid. We are led to suspect cryptomenorrhœa when the patient volunteers the statement that she has pelvic pain, headache, and possibly vomiting, of monthly occurrence, in fact the usual menstrual molimina, unaccompanied by any visible flow. A physical examination should be made at once in such a case, including abdominal palpation, inspection of the vulva, and a recto-abdominal bimanual examination. The common form is that in which the lower end of the vagina is imperforate, the hymen usually being visible on the outer side of the occluding membrane. The complete examination in such a case will reveal a fluctuating swelling reaching from the vulva to the pelvic brim, above which the uterus can often be palpated and moved about. It is further of considerable importance to make out whether the uterus and Fallopian tubes are distended with menstrual products along with the distended vagina, for in the presence of hæmatosalpinges the treatment is considerably modified. Abdominal section is required in such a case to avoid rupture of the tubes when the vagina collapses after incision of the occluding membrane. Distention of the vagina or hæmatocolpos is complete in this case, but may be partial where the lower part of the vagina is absent, and then is likely to be accompanied by distention of the uterus (hæmatometra) and hæmatosalpinx. Complete absence of the vagina can only be inferred from physical examination, when the distended organ appears to be only the uterus.

Although a secondary phenomenon, acquired cryptomenorrhœa produces the same symptoms and requires the same kind of investigation as the congenital cases. It must not be forgotten that acquired closure of the vagina following the vaginitis of specific fevers may occur in infancy, and will then, of course, produce primary amenorrhœa.

CAUSES OF APPARENT AMENORRHŒA.

Congenital :

- Imperforate vagina
- Imperforate hymen
- Absence of the vagina
- Imperforate cervix
- Double uterus with retention
- Hæmatocolpos
- Hæmatometra
- Hæmatosalpinx

Acquired :

- Closure of the vagina :
  - Due to specific fevers
  - Due to injury
- Closure of the cervix :
  - Due to injury
  - Following operations

CAUSES OF REAL AMENORRHŒA.

Physiological :

- Before puberty
- After the menopause
- During pregnancy
- During lactation

Pathological :

- Generative System :
  - Absence of essential organs
  - Infantile uterus
  - Small adult type of uterus
  - Deficient ovarian activity
  - Destruction of both ovaries :
    - By double ovarian growths
    - By pelvic inflammation
  - Superinvolution of the uterus
- Circulatory System :
  - Chlorosis
  - Anæmia
  - Leucocythæmia
  - Hodgkin's disease
  - Wasting diseases :
    - Malignant growths
    - Tubercle
    - Prolonged suppuration
    - Diabetes
  - Late stages of nephritis
  - Late stage of some forms of heart disease
  - Late stage of cirrhosis of the liver
- Nervous system :
  - Imbecility
  - Cretinism
  - Various forms of insanity
  - Cold just before or during menstruation
  - Suggestion—fear of pregnancy
  - Anorexia nervosa
- Altered internal secretions :
  - Myxœdema
  - Exophthalmic goitre
  - Addison's disease
  - Acromegaly
  - Obesity
  - Change of habits
- Toxic :
  - After specific fevers
  - Chronic poisoning by lead, mercury, morphia, alcohol

NOTE.—Real amenorrhœa may be (1) Primary with delayed onset ; (2) Primary and permanent ; (3) Secondary.

In considering the diagnosis of the causes of real amenorrhœa, the primary and secondary forms afford us an important clue to the possible causation. Suppose, for instance, that menstruation has once been established regularly, it is clear that there cannot be any serious congenital anomaly of the generative system ; the uterus and ovaries must at least be present and functional. We then must make a systematic examination of the generative, circulatory, nervous, and ductless gland systems, in order to learn by a process of exclusion which group of causes we have to deal with. If, however, the amenorrhœa is primary and real, that is, the patient has no molimina, our examination must first be directed towards finding out whether the essential organs, namely, uterus and ovaries, are present, and are normal in size and shape as far as a bimanual examination can ascertain. If necessary, an anæsthetic may be given for this purpose, for it is often a matter of considerable difficulty to decide the question. If the fact of absence of the essential organs can be established, we are clearly justified in considering the amenorrhœa to be permanent, and the patient or her friends should be told of this.

Apart from congenital anomalies, it is remarkable how few lesions of the generative organs there are which produce amenorrhœa; only those diseases which destroy both ovaries completely or render the uterus functionless can cause amenorrhœa, and under this heading we find only double ovarian growths, the late stages of pelvic inflammation (salpingo-oöphoritis), and superinvolution of the uterus. A tumour destroying one ovary as a rule has no effect on menstruation at all, provided the other is present and functionally perfect. It is possible for one ovary only to be functional; for instance, that on the same side as the undeveloped half of a unicornuate uterus may be quite atrophic and functionless. The presence of two tumours in the abdomen symmetrically arranged with regard to the uterus will sometimes permit of the diagnosis of double ovarian destruction, but quite commonly one tumour is much larger than the other, and the double nature of the lesion cannot be established until the abdomen is opened. Superinvolution of the uterus is not difficult to recognize when we remember that it always follows pregnancy, and the small size of the uterus can be made out readily by bimanual examination and the passage of the uterine sound. The organ sometimes measures only 1½ inches by the sound. It must not be forgotten that even in these cases the primary lesion may be an ovarian atrophy, but very little is known on this point. The term "deficient ovarian activity" is a time-honoured one, and must be taken to mean the absence of the internal secretion of the ovary. It is obvious that this condition cannot be diagnosed by any physical examination, and its presence can only be inferred when absolutely no other lesion of any system can be found to account for amenorrhœa, either primary or secondary.

It is impossible in the space at our disposal to draw up any detailed method by which the various diseases under the circulatory, nervous, etc., systems, can be diagnosed; these are discussed under the headings of other symptoms that they produce. It is, however, not out of place to note here that amenorrhœa caused by general diseases, unconnected with the generative system, depends upon: (1) Alterations in the blood itself; (2) Alterations in blood-pressure; (3) Altered relation of the nerve impulses which form part of the stimulus for menstruation; (4) Altered relations between the internal secretions of the ovary and the thyroid glands on the one hand, opposed to the suprarenal and pituitary glands on the other. Finally, with regard to pregnancy, which is the commonest of all causes of secondary amenorrhœa, it may be formulated as an axiom that an otherwise healthy woman who has had perfectly regular menstruation is probably pregnant if she has a period of absolute amenorrhœa. Nevertheless, the presence of pregnancy must never be assumed without a most careful consideration of the history, combined with a complete physical examination. The diagnosis of pregnancy must always be made upon a complex of symptoms rather than upon any one; the combination of amenorrhœa, secretion to be squeezed from the breasts, morning sickness, vaginal discoloration, and an abdominal tumour, can only mean pregnancy in the vast majority of cases. The addition of fœtal movements and the fœtal heart-sounds make the diagnosis absolute.

T. G. Stevens.

AMNESIA (Loss of Memory).—Memory is one of the higher functions of the brain, and presents wide variations in its degree of development in different individuals. The physiological range being so extensive, it is almost impossible to say whether an apparently poor memory is pathological or not, when the condition is of long standing and stationary. Slight degrees of impairment of memory are of interest to the psychologist, but to the majority of medical men the loss must be considerable or of peculiar character before it is of diagnostic importance. In some forms of excitement there may be exaltation of memory (hypermnesia); events are recalled and magnified in importance, which in normal states would never have reached the surface of conscious memory. In all forms of dementia, on the other hand, memory becomes impoverished (hypomnesia), and may eventually fail altogether (amnesia). Reference can be made to only a few states in which the condition of memory may be of service in diagnosis.

Dementia.—In all forms of dementia—senile, general paralytic, toxic, etc.—memory is impaired, and it is the rule to find that recent events are lost before those belonging to distant years. Even when memory is obliterated almost completely, a few isolated events in the past may be recalled distinctly without their surroundings, and may take a prominent place in the patient's personality. These traits characterize senility, but are also to be found, when looked for, in other demented states.

Epilepsy.—Amnesia is an important feature of the epileptic seizure ; in the majority of epileptics no memory of the convulsion is preserved, although events immediately preceding it may be retained clearly, as well as those which follow the return of consciousness ; in other cases the amnesia may cover a period preceding the attack (retrograde amnesia), while in others, actions are performed after the attacks, in an apparently conscious state, which the patient is quite unable to recall later on. To this phenomenon may be applied the term antegrade amnesia in association with post-epileptic automatism. Epileptic amnesia is often important in connection with medico-legal questions and criminology. In addition to temporary lapses of memory, the majority of epileptics suffer from the progressive hypomnesia common to all forms of dementia. It is one of the first signs of their intellectual deterioration, and not the result of the administration of bromides to which it is generally attributed.

Trauma.—Severe falls or blows on the head often cause complete amnesia ; the latter may cover not only a period of unconsciousness, but also a period preceding or following it, or both. As in cases of epilepsy, the amnesia may be retrograde, anterograde, or antero-retrograde.

Korsakow's Syndrome.—This condition, generally the result of alcoholism, is characterized by hypomnesia, disorientation, and pseudo-reminiscences. The patient loses memory for recent events, has no appreciation of time or place, talks freely and often plausibly about events which have never occurred, and yet may retain a very natural attitude of mind towards his surroundings. So natural may be his manner of talking and his behaviour, that the above-mentioned mental deficiencies may escape notice unless the medical man applies himself to their discovery.

Toxæmia.—In many infective diseases, such as enteric fever, the return of health may reveal a state of amnesia covering a considerable part of the patient's illness, and this blank, the result of intoxication of the higher cerebral centres, may be permanent.

Hysteria.—Amnesia is probably quite complete in connection with some forms of hysterical 'fits.' The patient in the interval between attacks has no recollection of the latter, although they are not associated with loss of consciousness. This fact underlies the theory which assumes a double consciousness; the person in one state of consciousness has no memory for events which occur in the other. *E. Farquhar Buzzard.*

ANÆMIA is a general and inexact term which may include one or more, or even all, of several different changes in the blood, but of which the main criterion clinically is diminution in the amount of hæmoglobin contained in a given volume, usually but not invariably associated with a decrease in the number of red cells per c.mm. of blood. Changes in the leucocytes are not essentially related to anæmia, though their behaviour affords means of diagnosing some forms of anæmia from others. Various terms have been used to denote different ways in which the blood may depart from the normal, and these may be defined shortly, though they are seldom important in practice.

Oligocythæmia or *hypocythæmia* both signify a diminution of the number of red cells below the normal 5,000,000 per c.mm. of blood in a man, 4,500,000 in a woman. *Oligæmia* means a diminished total amount of blood in the body ; *hydræmia*, an increased percentage of water in the blood ; *polyplasmia*, an increase in the volume of the plasma of the blood such as occurs in chlorosis ; *oligochromæmia*, a diminution in the amount of hæmoglobin per c.mm. of blood.

For purposes of comparison of one case with another, one speaks of the red cells and of the hæmoglobin as being normally 100 per cent in health. An anæmia may be such that the hæmoglobin is greatly diminished without so great a diminution in the red corpuscles ; it is also possible for the hæmoglobin and the red cells each to be diminished in equal proportions ; and thirdly, it is possible for both the hæmoglobin and the red corpuscles to be diminished, but for the hæmoglobin to be relatively less so than are the red cells. The red corpuscles contain relatively less hæmoglobin than they ought to in the first variety of anæmia, which is probably the commonest of all ; in the second group, although there is anæmia, each red corpuscle contains its full quantity of hæmoglobin ; whilst in the third group, although there is anæmia, each corpuscle contains more hæmoglobin than it normally should. As a means of expressing these facts shortly, one speaks of the *colour index* : this is the ratio of the hæmoglobin to the red corpuscles. If the red corpuscles and hæmoglobin are each 100 per cent of normal, the colour index is $\frac{100}{100}$, or 1. If the hæmoglobin were diminished to 40 per cent of normal, whilst the red cells were only diminished to 80 per cent of normal, the colour index would be $\frac{40}{80}$, or 0·5—the *chlorotic type*, in which the index is less than 1. If the hæmoglobin and the red cells were both diminished to 50 per cent of normal, there would be anæmia with a normal colour index of $\frac{50}{50}$, or 1. If the hæmoglobin were diminished to 30 per cent of normal, whilst the red cells were diminished to 20 per cent of normal, the colour index would be $\frac{30}{20}$, or 1·5—that is to say, greater than 1, a condition which is spoken of as the *pernicious type* of colour index, because it is seen best in pernicious anæmia.

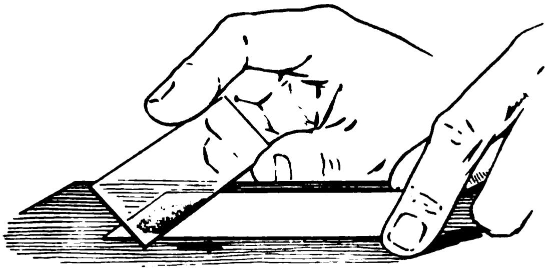


Fig. 2.—A GOOD METHOD OF MAKING A BLOOD-FILM. A drop of blood is received at one end of a microscope slide: a second slide is laid transversely across the first, as shown; the blood being in the acute angle *behind* it, the transverse slide is carried steadily forward over the surface of the first, *drawing the blood after it* in an even film.

Pallor may or may not indicate anæmia. Many persons look almost white, and yet their blood is not in an abnormal condition. Pallor is normal in night-workers and in those who work underground. Even in some daylight workers the distribution of the cutaneous capillaries seems to be such that the superficial skin has little, if any, of the normal colour of blood, and yet the individuals are not anæmic in the sense of having any diminution of the hæmoglobin or the red cells. The error of mistaking mere pallor for anæmia is avoided by means of a blood-count, which in all cases should include estimation of the percentage of hæmoglobin, and of the total number of red cells per c.mm.; and in most cases determination of the number of leucocytes per c.mm., a differential leucocyte count and an examination of the characters of the red corpuscles in stained blood films also.

Corpuscles are best counted by means of the Thoma-Zeiss or Thoma-Leitz hæmocytometer; hæmoglobin is measured most accurately by the Haldane-Gowers hæmoglobinometer; and blood films are best fixed and stained simultaneously by means of Jenner's stain, or, when hæmatozoa are to be looked for, by Leishman's stain. Full directions as to the use of these instruments and stains are generally issued with them, or one of the many small handbooks on laboratory methods may be referred to.

Having proved that the patient is suffering from real anæmia, that is to say from diminution in the percentage of hæmoglobin, and probably from a diminution in the red cells also, the next step in the diagnosis is to determine what is its nature. Attempts are sometimes made to fit all cases of anæmia into one or other of two main groups, labelled *primary* and *secondary* respectively; but this is not really very helpful clinically. In many cases the nature of the anæmia is obvious at once—it may be secondary to post-partum hæmorrhage or other blood loss, or the later stages of phthisis, syphilis, cancer, or malarial cachexia, and so on. Sometimes, however, even though anæmia is really due to a cause which in some patients is obvious, it is not obvious in the patient with whom one happens to be dealing, and then the diagnosis has to be arrived at by a process of exclusion. One need but mention as examples, perhaps, the difficulties that arise sometimes in diagnosing between fungating endocarditis, gastric carcinoma, and pernicious anæmia; or between anæmia due to blood-loss and blood-loss due to anæmia. In arriving at the diagnosis it is important first to exclude those conditions in which the blood picture is itself positive. A division of all cases of anæmia into (*A*) *Anæmias with a positive blood picture*; and (*B*) *Anæmias with an indeterminate or negative blood picture*, is probably more helpful clinically than any other classification. The only anæmias in which the blood picture can be described as itself positive—that is to say in which the diagnosis is indicated directly by the results of blood examination—are (*a*) pernicious anæmia, (*b*) spleno-medullary leukæmia, (*c*) lymphatic leukæmia, (*d*) mixed varieties of leukæmia, (*e*) parasitic anæmia associated with eosinophilia, and (*f*) parasitic anæmia associated with parasites in the blood.

Blood Changes common to all Severe Anæmias.—In any severe anæmia there are certain blood changes which are almost always to be found, which are not characteristic of any one variety of anæmia, but which, seeing that pernicious anæmia in its later stages is probably the profoundest of all the anæmias, are perhaps better seen in it than in any other disease. These are :—

(*a*). A very great diminution in the *number* of red corpuscles, down even to so low a figure as 600,000 per c.mm.

(*b*). Great variation in the *shapes* of the red cells—poikilocytosis; poikilocytes (*Plate II, Fig. E*) always retain a smooth, curved outline, but instead of being flat circular discs, like normal corpuscles, they may be oval or pear- or hour-glass-shaped, and so on. It is important not to mistake crenated corpuscles (*Plate II, Fig. D*), or red cells that have become polygonal by reason of mutual moulding when fixed in too close apposition with one another (*Plate II, Fig. C*), for poikilocytes.

(*c*). Alterations in the *sizes* of the corpuscles. In normal blood the red cells are almost all of the same diameter, about 7μ; in any severe anæmia they may vary considerably in size, many being much smaller than normal—*microcytes* (*Plate II, Fig. B*); some larger than normal—*macrocytes* or *megalocytes* (*Plate II, Fig. B*).

(*d*). The presence of *nucleated red corpuscles*. Normally none are present in the blood even in infancy; in any severe anæmia they may appear in varying numbers, and according to their sizes they are termed *microblasts*, *normoblasts*, *megaloblasts*, or *gigantoblasts* (*Plate II, Fig. F*)—the latter containing more than one nucleus, the others only one. It has sometimes been stated that the greater the number of nucleated corpuscles the less favourable the prognosis, but this is not necessarily the case, except in so far that it is unusual for nucleated forms to appear until a severe stage of the anæmia is reached.

None of the above changes, one must repeat, are diagnostic of any particular variety of severe anæmia, though they are perhaps most marked in the later stages of pernicious anæmia.

Normal Varieties of White Corpuscles.—It often happens that variations in the relative proportions of the different leucocytes in the blood afford means of differential diagnosis. Before changes from the normal can be understood, it is necessary to say a word or two about the normal varieties of white cells; these number anything from 5,000 to 10,000 per c.mm., the total changing considerably at different times of the day. When films are made it is found that four easily distinguishable varieties are to be seen. These have received different names at the hands of different observers, but they are so distinct that names hardly matter, and they might be termed quite well types A, B, C, and D respectively. If, however, one has to choose between the different names that have been given to them, the following may perhaps be selected as the most frequently employed :—(1) *Small lymphocytes*; (2) *Large lymphocytes*; (3) *Polymorphonuclear cells*; (4) *Coarsely granular eosinophile corpuscles.*

1. *The small lymphocytes* (*Plate II, Fig. H*) stain blue with Jenner's stain, both as to nucleus and protoplasm. The nucleus is round, and the protoplasm is relatively small in amount and free from granules.

2. *The large lymphocytes*, or *hyaline corpuscles* (*Plate II, Fig. J*), stain blue, both as to nucleus and protoplasm. The nucleus is more or less kidney-shaped, and the protoplasm relatively large in amount and free from granules.

3. *The polymorphonuclear cells* (*Plate II, Fig. K*) stain blue as to the multilobed nucleus, red as to the relatively abundant protoplasm, which under the high power is seen to be speckled with very fine red granules.

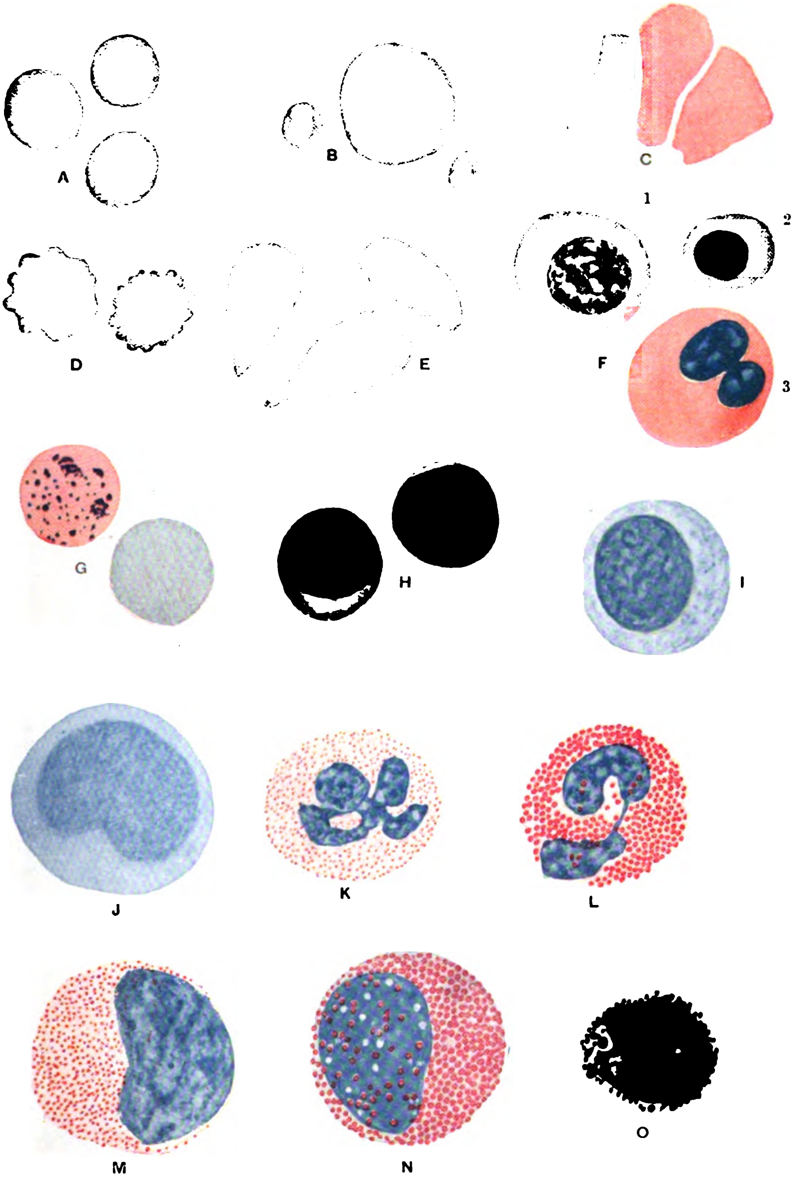
4. *The coarsely granular eosinophile corpuscles* (*Plate II, Fig. L*) stain blue as to the multilobed nuclei, red as to the protoplasm, the amount of which is approximately the same as in the polymorphonuclear cells, but differs from the latter in that it is studded with very striking large eosinophile granules.

The only difficulty that arises in making a differential leucocyte count in normal blood is that whereas the small lymphocytes usually become fixed in such a way as to cover relatively small areas, so that the cells seem to consist mainly of nucleus, at other times they spread out flatter over larger areas, and then the rounded nucleus seems to be surrounded by much protoplasm (*Plate II, Fig. I*). A small lymphocyte flattened out in this way is apt to be called either a large lymphocyte by those who do not insist upon the reniform nucleus of the latter, or a *transitional lymphocyte* by others. There is no deduction

PLATE II

RED AND WHITE BLOOD-CORPUSCLES

As seen under the $\frac{1}{12}$th-inch oil-immersion lens.



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A, Normal red corpuscles; **B**, Megalocytes and microcytes; **C**, Normal red corpuscles made angular by imperfect fixation; **D**, Crenated red corpuscles; **E**, Poikilocytes; **F**, Nucleated red corpuscles; (1) Normoblasts, (2) Megaloblasts; (3) Gigantoblasts; **G**, Punctate basophilia and polychromasia; **H**, Small lymphocyte; **I**, Indeterminate lymphocyte; **J**, Large hyaline lymphocyte; **K**, Polymorphonuclear corpuscle; **L**, Coarsely granular eosinophile corpuscle; **M**, Myelocyte; **N**, Eosinophile myelocyte; **O**, Basophile corpuscle.

INDEX OF DIAGNOSIS—*To face p. 22.*

of particular clinical value to be obtained by distinguishing these cells from small lymphocytes: it is better that they should be grouped with the small lymphocytes for clinical purposes at any rate, only undoubted large hyaline cells with reniform nuclei being included in the group of large lymphocytes or hyaline corpuscles.

The relative proportions of these cells differ according as the individual is a child or a grown-up person; for an adult one may say that, roughly speaking, out of 100 leucocytes

About 25 will be small lymphocytes
8 will be large hyaline lymphocytes
65 will be polymorphonuclear cells, and
2 will be coarsely granular eosinophile corpuscles
—
100

In children the tendency is for the small lymphocytes to be relatively more numerous in health, and still more so in any illness—up to 40 per cent or even more—whilst the polymorphonuclear cells are correspondingly diminished.

Some observers prefer to represent the different varieties of white corpuscles not as percentages but as total numbers per c.mm. of blood.

Abnormal Varieties of White Corpuscles.—Whereas the above are the only kinds of white cells in healthy blood, in certain diseases the following abnormal forms are met with:—

Myelocytes.—These are large corpuscles (*Plate II, Fig. M*), comparable in size to the polymorphonuclear cells, but differing from the latter in having either a perfectly round, an oval, or possibly a slightly kidney-shaped nucleus, rather than a multilobed one. There are all gradations of them, and at the two extremes it is difficult to differentiate some from large lymphocytes and others from polymorphonuclear cells. They are to be distinguished from the latter by the roundness of the nucleus, and from large lymphocytes by the granularity of the protoplasm. The granules in question are sometimes stained brightly with eosin—*eosinophile myelocytes* (*Plate II, Fig. N*), distinguishable at once from the ordinary eosinophile corpuscles by their nuclei being nearly spherical; more often, however, the granules stain blue, or some colour between blue and red—ordinary or *neutrophile myelocytes.* No useful clinical information can, so far as is at present known, be obtained by laying stress upon these differences in the staining reactions of different myelocytes, so that they are usually counted together simply as myelocytes. There is only one condition in which they are very numerous, and that is spleno-medullary leukæmia; but they may occur in small numbers in various other affections also, particularly in lymphadenoma, Hodgkin's disease, pernicious anæmia, and aplastic anæmia.

Basophile Corpuscles (*Plate II, Fig. O*).—These are much smaller than myelocytes, their size being comparable to that of small lymphocytes; they differ from the latter in that the protoplasm, instead of being homogeneous, contains from 2 or 3 to perhaps 20 or more very large granules which stain deep blue with Jenner's stain. They are unmistakeable. No definite clinical deductions can be drawn from their presence beyond the fact that, if there are more than 1 or 2 per 1,000, the blood is abnormal. They may be present in many different varieties of anæmia, but they are not characteristic of any; they seldom amount to more than 2 or 3 per cent, and often to no more than 0·5 per cent, even in disease.

Punctate Basophilia.—There are certain conditions, particularly pernicious anæmia in its later stages, leukæmia, and lead poisoning, in which the red cells, instead of staining uniformly pink with the eosin of Jenner's stain, present large numbers of small blue specks or granules in their protoplasm (*Plate II, Fig. G*), a condition known as *punctate basophilia.* In a case of doubt, when pernicious anæmia has been excluded by there being a low colour index, and when leukæmia is contra-indicated by the fact that there is a normal leucocyte count, the presence of extensive punctate basophilia is said sometimes to afford confirmative evidence of plumbism.

We may now pass on to consider the commoner varieties of anæmia, dealing first with anæmias with positive blood pictures.

(*A*).—ANÆMIAS WITH POSITIVE BLOOD PICTURES.

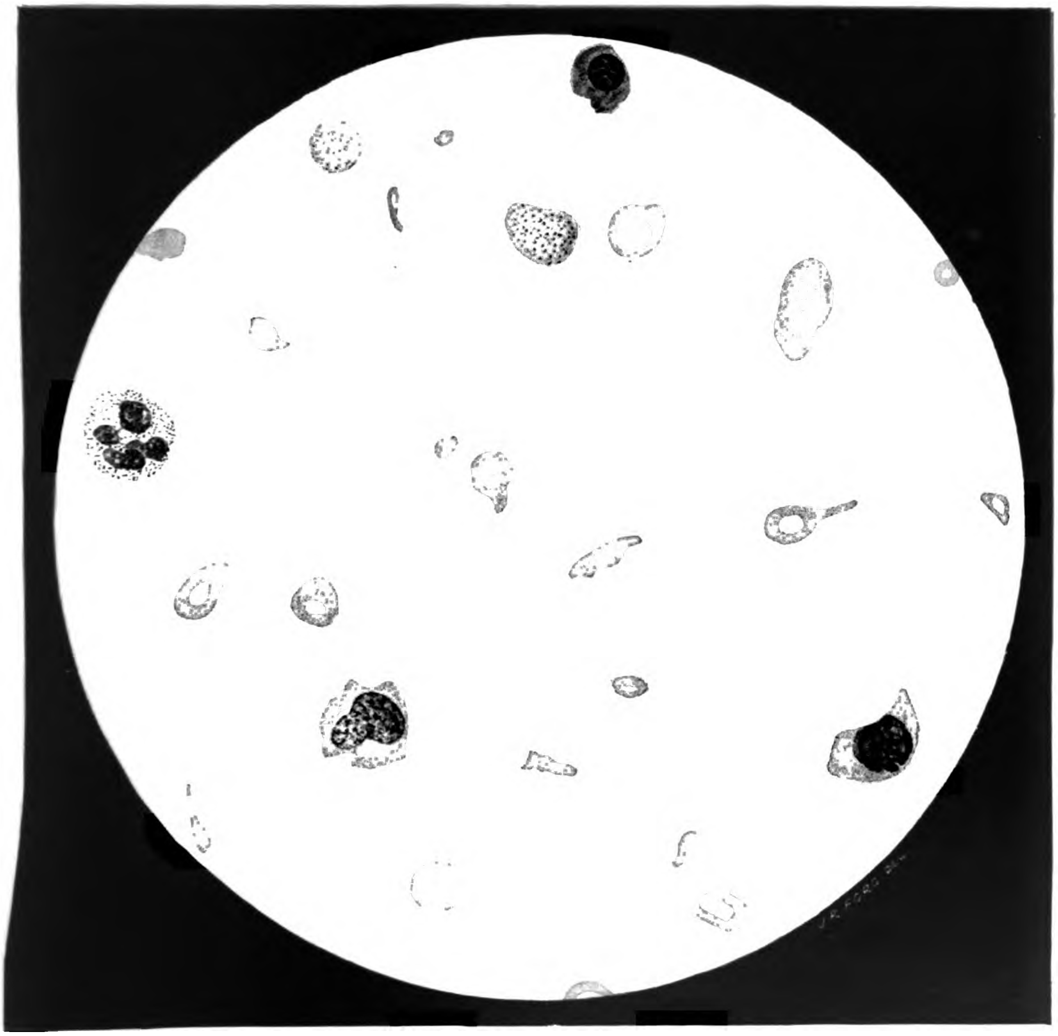
Pernicious Anæmia is a disease of insidious onset in adults, the main symptoms being progressive loss of muscle-power and increasing pallor, with loss of weight, but with relatively less loss of body volume. Various other symptoms may be associated with these, or no others may be present. The diagnosis is seldom made until a relatively late stage of the malady has been reached, by which time there is a great diminution in the hæmoglobin, down perhaps to 30 per cent of normal or less, and a still greater diminution of the red cells, down perhaps to 25 per cent, 20 per cent, or even 10 per cent of normal ; consequently the colour index is high, and this is the pathognomonic sign of the disease. There is no leucocytosis, but rather leucopenia (p. 331): the differential leucocyte count shows a relative increase in the small lymphocytes, a corresponding diminution in the polymorphonuclear cells, normal numbers of eosinophile corpuscles and large lymphocytes, occasional basophile corpuscles, and one or two myelocytes. Blood films also show all the changes described above (p. 22) as common to any severe anæmia, but with particularly large relative numbers of megalocytes. When these blood changes are all present there can be no doubt about the diagnosis, and we need not enter here into all the other symptoms that may be presented by the patient. It is important to remember, however, that there is one group of the cases in which nerve symptoms predominate before the anæmia is pronounced. The diagnosis of pernicious anæmia cannot be made without a blood-count, and it can be made absolutely with one ; one word of warning is required, and that is that the colour index is not continuously high in every case of pernicious anæmia, so that perhaps several blood-counts may be required at intervals. It should also be noted that the power of temporary recuperation is considerable, and when the patient's condition improves the blood may return partly or wholly to normal ; during such remission the colour index, instead of remaining greater than 1, becomes 1 or less than 1.

There are certain cases of very severe anæmia which some would include under the heading of pernicious anæmia, although the colour index is persistently less than 1. It is more useful, however, from a clinical point of view to leave these cases unlabelled, or at any rate not to call them pernicious anæmia, which has so characteristic a blood picture. One variety has recently become separated from the rest under the title of *aplastic anæmia*, the chief characters of which are a profound, progressive, and ultimately fatal anæmia for which no cause can be found, which seems in many respects to simulate pernicious anæmia, but which is persistently associated with a low instead of a high colour index. It is, moreover, unaccompanied by a positive Prussian blue reaction in the liver—Perl's test with potassium ferrocyanide and hydrochloric acid—post mortem ; this, when positive, is strongly confirmatory of pernicious anæmia, for very few other conditions give it, and they are rare—sprue, for example, is one such, and bronzed diabetes another.

Spleno-medullary Leukæmia.—In the earlier stages of this disease there is no anæmia at all, though later diminution both in the hæmoglobin and in the red cells may be profound. The essential point in the diagnosis is the occurrence of a very great increase of the total number of leucocytes, not at all uncommonly up to such a figure as 200,000, and sometimes up to 600,000 or even 1,000,000 per c.mm. There is only one other condition which can produce so extreme an increase in the total number of leucocytes, and that is *lymphatic leukæmia*. The two are immediately distinguishable from one another by the differential leucocyte count, the characteristic point about which, in spleno-medullary leukæmia, is the large number of myelocytes present. These may amount to 30, to even 50 per cent, or more, of all the leucocytes present, with the consequence that there is a relative but not an absolute diminution in the other varieties of white cells. Occasional basophile cells are seen ; but whatever may be the proportion of these or other leucocytes, the main point in the diagnosis is the large relative number of myelocytes in association with an enormous increase in the total leucocyte count. When anæmia ultimately ensues it is of the chlorotic type ; that is to say, the hæmoglobin falls before, and to a greater extent than, the red cells. The disease generally lasts from one to three years before ending fatally, and in the later stages all the blood-changes characteristic of severe anæmia may be found. Clinically, the other main feature of the complaint is the enormous enlargement of the spleen, which here reaches dimensions bigger than in any other disease, the viscus often extending right across the middle line to the right iliac fossa or down into the pelvis. It is note-

PLATE III

PERNICIOUS ANÆMIA



Part of a blood film from a case of severe pernicious anæmia, showing poikilocytes, microcytes, megalocytes, nucleated red cells, and punctate basophilia.

worthy that in patients treated with the x-rays the spleen very often becomes greatly reduced in size, and the blood picture may return nearly to normal, though it seldom if ever happens, even when the number of leucocytes per c.mm. has reached the normal, that there is an absence of myelocytes in the differential leucocyte count. Notwithstanding this apparent improvement in the blood and in the spleen, the length of time the patient survives does not seem to be increased. The splenic enlargement is not associated with enlargement of the lymphatic glands.

Lymphatic Leukæmia.—There is no age at which any form of leukæmia may not occur; but upon the whole the spleno-medullary form affects adults rather than children, whereas the lymphatic affects children rather than adults. Its course is usually rapid and invariably fatal, death resulting, as a rule, within three or four months from the first definite symptoms. Anæmia is much more rapid in its development in the lymphatic than in the spleno-medullary form. The first symptoms may be either anæmia, or lymphatic glandular enlargement in the neck, axillæ, and groins, or the occurrence of purpura, epistaxis or other forms of hæmorrhage, or in certain cases a complete change in the child's temperament in the direction particularly of excessive irritability of temper, with loss of appetite and obvious and progressive illness. There are cases in which no glands are enlarged, the diagnosis not being at all obvious without a blood-count. More often there is general enlargement of the lymphatic glands, visceral and peripheral, sometimes associated with similar increase in the size of other glands, particularly the salivary and lachrymal—Miculicz's syndrome—and the spleen is nearly always palpable and sometimes large, though seldom so big as it is in spleno-medullary leukæmia. Serous inflammations are common, and there is apt to be pyrexia, as in other severe anæmias, especially in spleno-medullary leukæmia (*Fig.* 4), Hodgkin's disease (*Fig.* 247, p. 570), and pernicious anæmia (*Fig.* 246, p. 569). The diagnosis is afforded at once by the blood-count in the majority of cases. There is a varying degree of increase in the leucocytes, sometimes

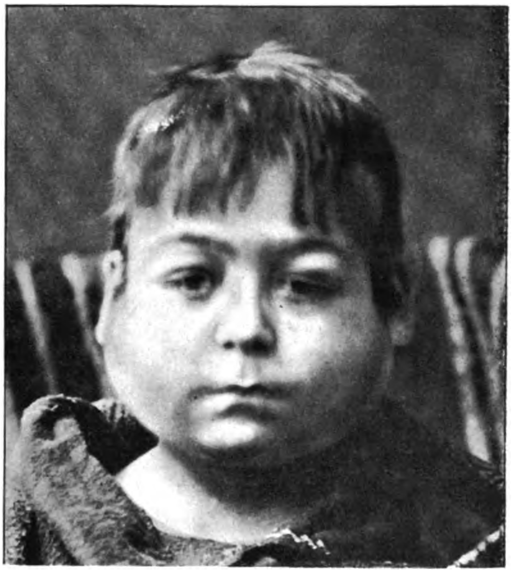


Fig. 3.—A case of chronic enlargement of the salivary and lachrymal glands (Miculicz's syndrome) in a case of lymphatic leukæmia. (*Kindly lent by Dr. Frederick Taylor.*)

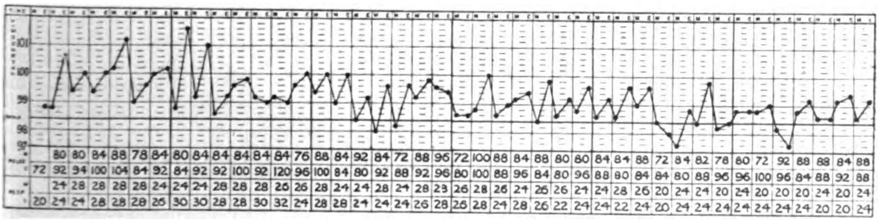


Fig. 4.—Temperature chart (morning and evening) in a case of spleno-medullary leukæmia in a woman, age 11, who improved very markedly under x-ray treatment while in hospital.

reaching no higher than 20,000 or 30,000, more often 80,000 to 100,000, and sometimes, but more rarely, to much higher figures, such as 200,000, 600,000, 800,000, or even 1,500,000 per c.mm. Whatever the total leucocytes count, however, the striking feature is the enormous relative increase in the small lymphocytes in the differential leucocyte count. Out of every hundred leucocytes it is not uncommon to find that 90, or even

95 or 98 are lymphocytes; so that there is an enormous relative and sometimes absolute reduction in the other white corpuscles. Amongst them will be found an occasional myelocyte and one or two basophile corpuscles. The red cells and the hæmoglobin become diminished progressively, and the former may exhibit all the other changes described above (p. 22) as characteristic of any very severe anæmia. Whereas in most cases the colour index becomes less than 1 as the disease progresses, in a few instances, especially some time before the end, the colour index has been found to be greater than 1, as it is in pernicious anæmia. There is no likelihood of mistaking one condition for the other on account of the changes in the white cells.

Some authorities describe two types of lymphatic leukæmia according as the lymphocytes seen in the films are of relatively large or small size; as has been explained above, however, there is always difficulty in deciding whether differences in apparent size of the lymphocytes constitute differences in kind, and there is no very great clinical purpose served in drawing the distinction here, unless perhaps that upon the whole the larger the lymphocytes present the greater the number of months the patient is likely to survive.

The chief difficulties that arise in the diagnosis occur in two ways: first, there are a few instances in which lymphatic leukæmia has run its course without any actual increase in the number of leucocytes per c.mm. of blood, the diagnosis being afforded only by the enormous relative increase in the small lymphocytes; and secondly, children normally have a relatively high leucocyte count, from which it happens that lymphatic leukæmia may sometimes be suspected in them when it is not really present. Suppose, for instance, a child suffers from an obscure illness associated with anæmia of the chlorotic type with an increase in the leucocytes up to 25,000 per c. mm. and a relative increase of the small lymphocytes up to 55 per cent, would one be justified in diagnosing lymphatic leukæmia? One might be if there was general enlargement of the lymphatic glands and enlargement of the spleen; but otherwise both the leucocytosis and the relative increase in the lymphocytes might be due to some other complaint, and the only means of arriving at the diagnosis might be by awaiting developments. It is not safe to insist upon a diagnosis of lymphatic leukæmia unless there is either a very large increase in the total number of leucocytes, or a relative increase in the small lymphocytes up to 90 per cent or over, or both these changes at the same time.

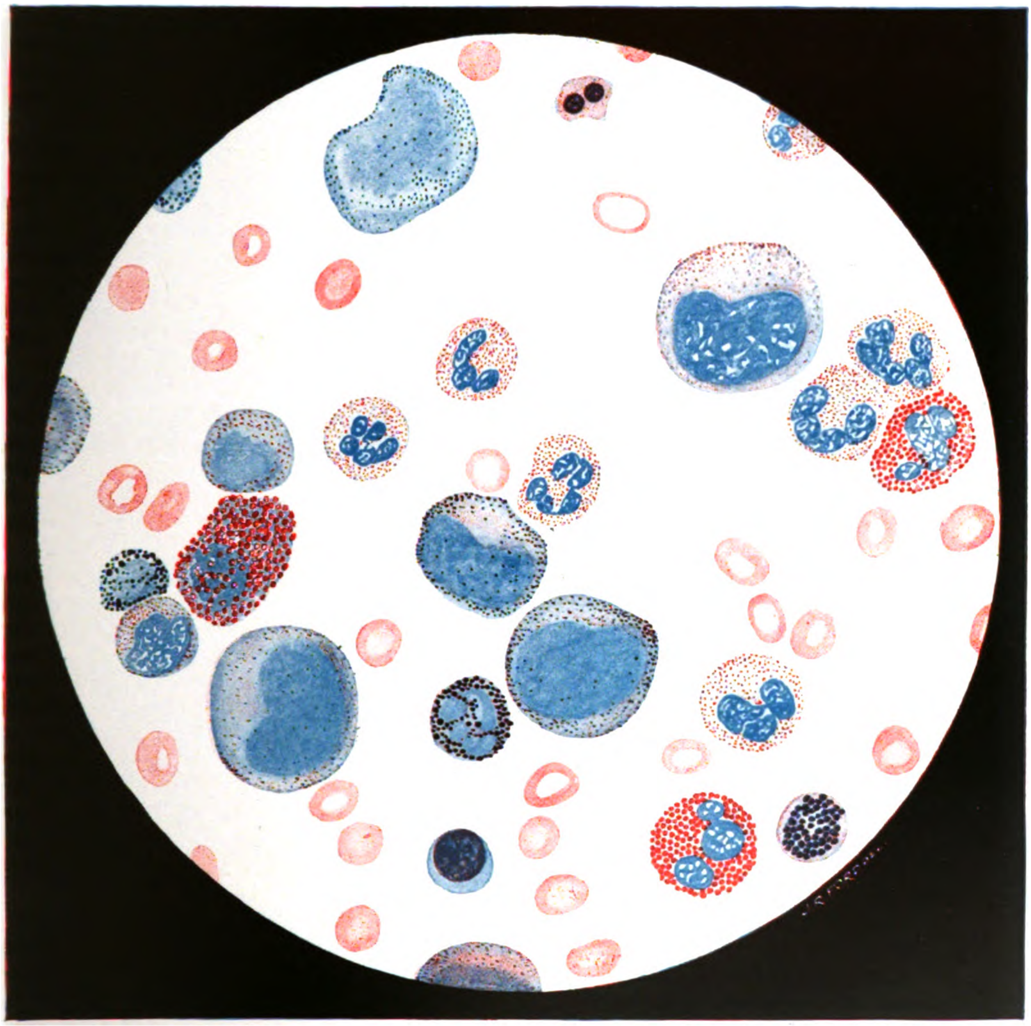
Mixed Forms of Leukæmia.—Although the majority of cases of leukæmia belong either to the spleno-medullary or the lymphatic form, there are cases in which the symptoms and the blood changes partake of the characters of both. Either the splenic or the lymphatic glandular enlargement, or both, may be marked; there may be no anæmia until the disease has passed its earlier stages, when the red cells and hæmoglobin pass through the chlorotic type of changes until they reach those severe alterations characteristic of all anæmias in their last stages; the white corpuscles show more or less increase in their total numbers, and the differential leucocyte count shows not only considerable numbers of myelocytes, such perhaps as 20 per cent or more, but also a great relative increase in the lymphocytes up to, it may be, 60 per cent or over. The occurrence of these cases of ‘ mixed ’ leukæmia would seem to indicate that there is really no absolute difference in kind, but rather only a difference in type, between the lymphatic and the spleno-medullary forms already described.

Parasitic Anæmia associated with Eosinophilia.—Many varieties of the parasites that affect man produce hardly any blood changes at all—*Trichocephalus dispar*, *Oxyuris vermicularis*, *Ascaris lumbricoides*. Other parasites, however, produce very marked changes in the blood, and one may mention in particular *Bothriocephalus latus*, *Ankylostomum duodenale*, *Trichina spiralis*, *Bilharzia hæmatobia*, *Filaria sanguinis hominis*, and not a few cases of *hydatid* disease. The anæmia which results may be very profound, and the blood may exhibit all the changes described above as common to the severest anæmias. The colour index is usually low, but sometimes it is greater than 1, simulating pernicious anæmia; but whatever the total leucocytes, the differential count very commonly presents a considerable increase in the coarsely eosinophile corpuscles, and this EOSINOPHILIA (p. 219), in association with severe anæmia, is suggestive of the presence of some toxic parasite. It does not indicate which parasite is present, however, this being determined by careful examination of the fæces, urine, and so forth (see PARASITES, INTESTINAL, p. 519).

Parasitic Anæmia associated with Parasites in the Blood.—The four best known

PLATE IV

SPLENO-MEDULLARY LEUKÆMIA



Part of a blood film from a case of spleno-medullary leukæmia, showing five neutrophile myelocytes, one eosinophile myelocyte, three basophile cells, and one binucleated red cell in addition to normal corpuscles.

diseases in which human beings have parasites in the blood are : malaria, filariasis, trypanosomiasis, and relapsing fever. In all these there may be much destruction of red cells with consequent anæmia of the chlorotic type. In most cases the history, particularly of residence in some tropical country where the disease in question is likely to occur, will suggest the diagnosis, and the examination of the blood, either fresh or in films, will be confirmative.

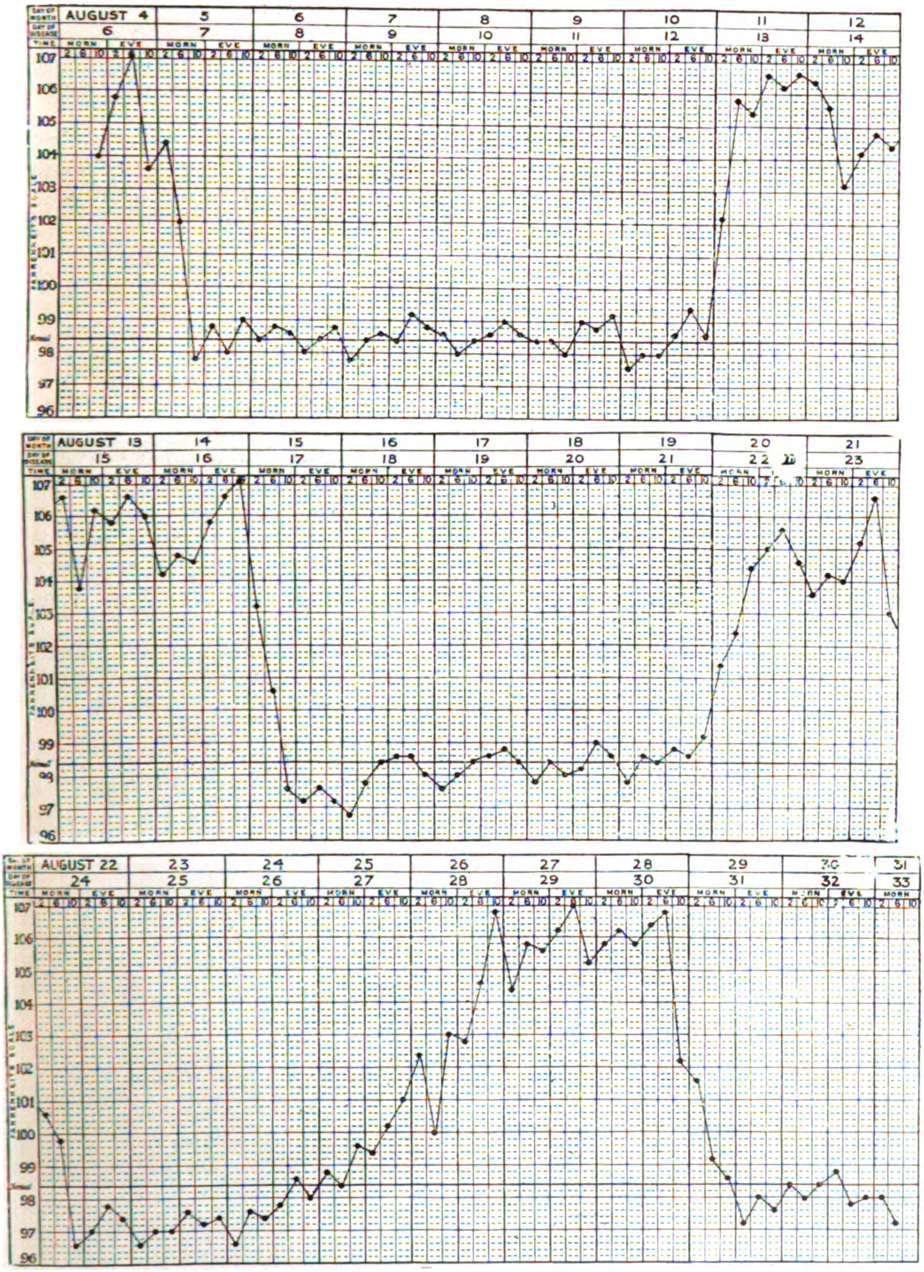


Fig. 5.—Four-hourly temperature chart in a case of severe relapsing fever.

Relapsing fever used to be prevalent in Great Britain, and it still occurs in epidemic orm in times of famine in association with uncleanness. It is commoner abroad. It is due to infection by the spirochæte of Obermeier (*Plate XXVIII, Fig I*, p. 614) introduced into the body by the bites of bugs. It is a long spiral organism, 40 μ long and 1 μ broad, actively motile in fresh blood, but best seen in films stained with Leishman's stain. They first appear a day or two before the paroxysms of fever (*Fig.* 5), and may reach large numbers In the intervals they are not to be found. The course of the disease usually suggests the diagnosis, outbursts of pyrexia associated with extreme prostration and severe illness, lasting about a week or rather less, alternating with intermissions of about the same length. There may be an indefinite number of relapses before the patient either dies or recovers.

Filariasis may be latent for a long time before it produces symptoms. Its best known effects are elephantiasis of the legs or genital organs, with or without chyluria. It occurs in many parts of the tropics, particularly in some of the Pacific Islands, such as Fiji; and in certain parts of China. The elephantiasis and chyluria are due to mechanical obstruction to the pelvic lymphatics by the mature worms. The blood exhibits more or less anæmia of the chlorotic type, with a varying degree of eosinophilia, whilst at certain times of the day or night the peripheral blood also contains the long but narrow filarial embryos (*Plate XXVIII, Fig. F*, p. 614). There are probably different varieties of the organism, but they cannot be distinguished easily by the appearance of these embryos alone. Without laying stress upon generic differences, it is important that in most cases they are to be found in the peripheral blood only at night (*Filaria bancrofti nocturna*); during the day they seem to retreat into the deep vessels; there are other cases, however, in which embryos, very similar in appearance, occur in the peripheral blood in the daytime and not at night (*Filaria diurna*); whilst in *Filaria perstans* they are present in the blood both day and night. Roughly speaking, one may say that each embryo when stretched out is 200 μ long and 4 to 5 μ wide, and they stain by Leishman's method. They may be found in the blood of patients who have returned to England after contracting the disease abroad.

Trypanosomiasis—the cause of *sleeping sickness*. Trypanosomes of many different kinds are known to affect various animals, birds and fish, but the only one which is important in man is the *Trypanosoma gambiense* (*Plate XXVIII, Fig. G*, p. 614). It is to be found in blood films stained by Leishman's method months or years before it finds its way into the cerebrospinal fluid to produce sleeping sickness. It has a large and definite nucleus about its middle, surrounded by protoplasm which becomes prolonged into a relatively long undulating membrane terminating in a flagellum. It is an extra-corpuscular organism readily distinguishable when seen in its mature stage. It occurs particularly in people who have been resident in Uganda or other district in which *Glossina palpalis*, the fly which spreads the disease, abounds. The diagnosis is much less easy when the blood contains only immature forms. It is sometimes easier to find the embryos in fluid obtained by puncturing the enlarged inguinal or other lymphatic glands often present in these patients. It is

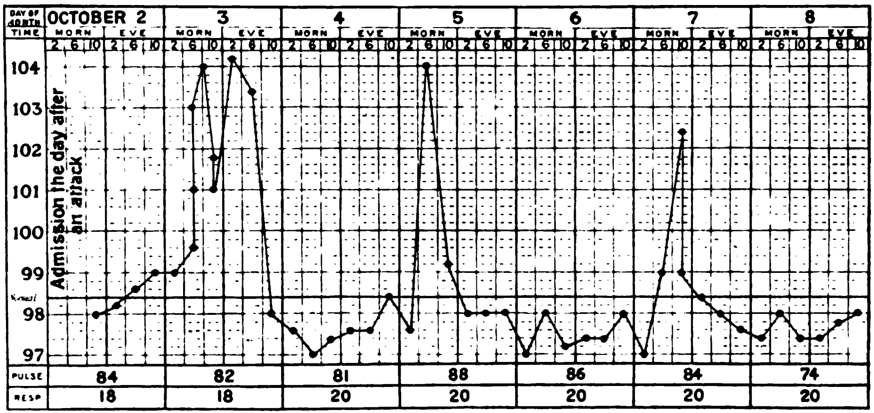
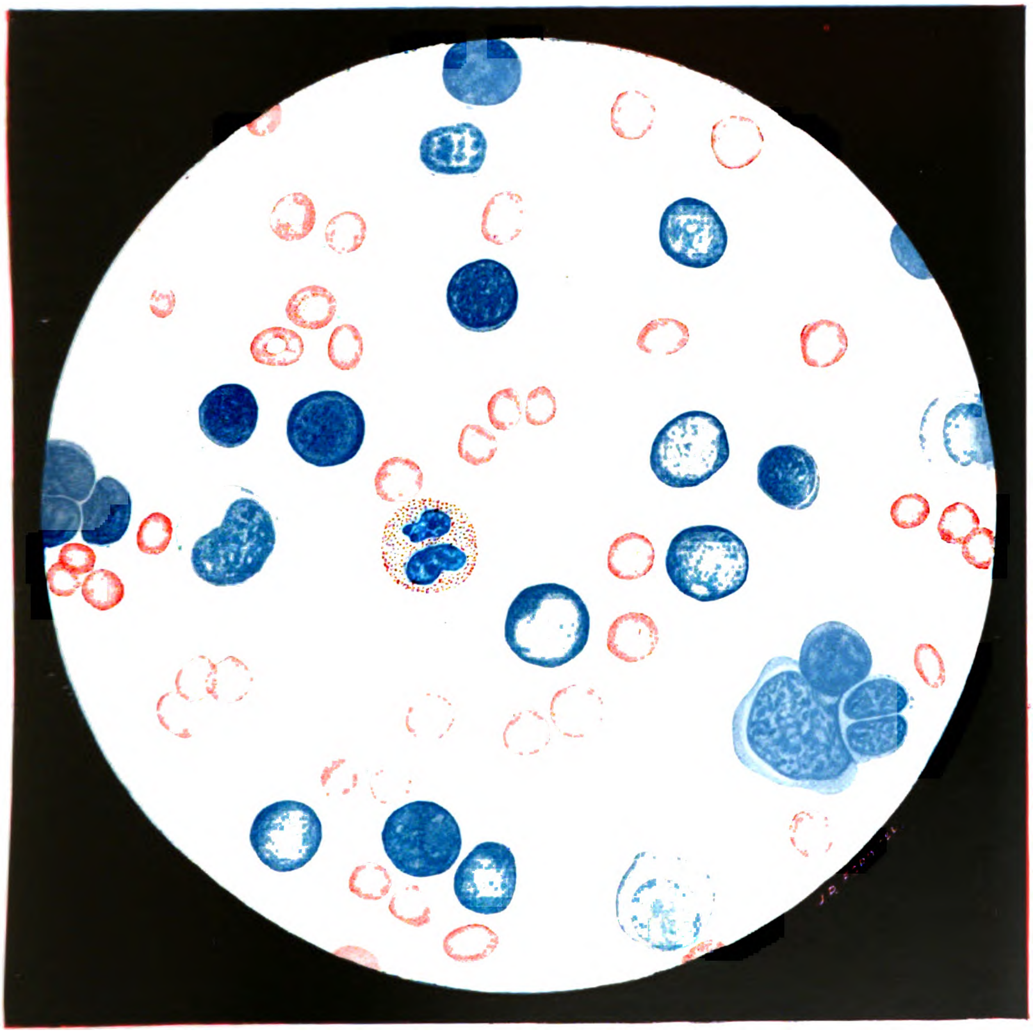


Fig. 6.—Case of simple tertian malaria, showing the attacks occurring every third day.
(*Chart supplied by the London School of Tropical Medicine.*)

PLATE V

LYMPHATIC LEUKÆMIA



Part of a blood film from a case of lymphatic leukæmia, showing a large increase in the small lymphocytes.

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worthy of note that one variety of severe anæmia occurring in Assam, associated with pyrexia and enlargement of the spleen, and formerly thought to be a variety of malaria, is due to a variety of trypanosomiasis in which only immature forms of the parasite (Leishman-Donovan bodies) have been found (*Plate XXVIII, Fig. H*, p. 614); and here not in the general blood stream, but in the fluid obtained by splenic puncture. The disease is termed ***Kala-azar***.

Malaria is not essentially associated with anæmia; but in patients who have had recurrent attacks blood destruction by the parasites leads to considerable reduction both in the red cells and in the hæmoglobin, the colour index generally being of the chlorotic type. The changes in the white corpuscles are described on p. 361. The diagnosis can often be surmised when a patient who is, or has been, resident in a malarial district suffers from periodic rigors with pyrexia. Theoretically there are two main types of the disease,

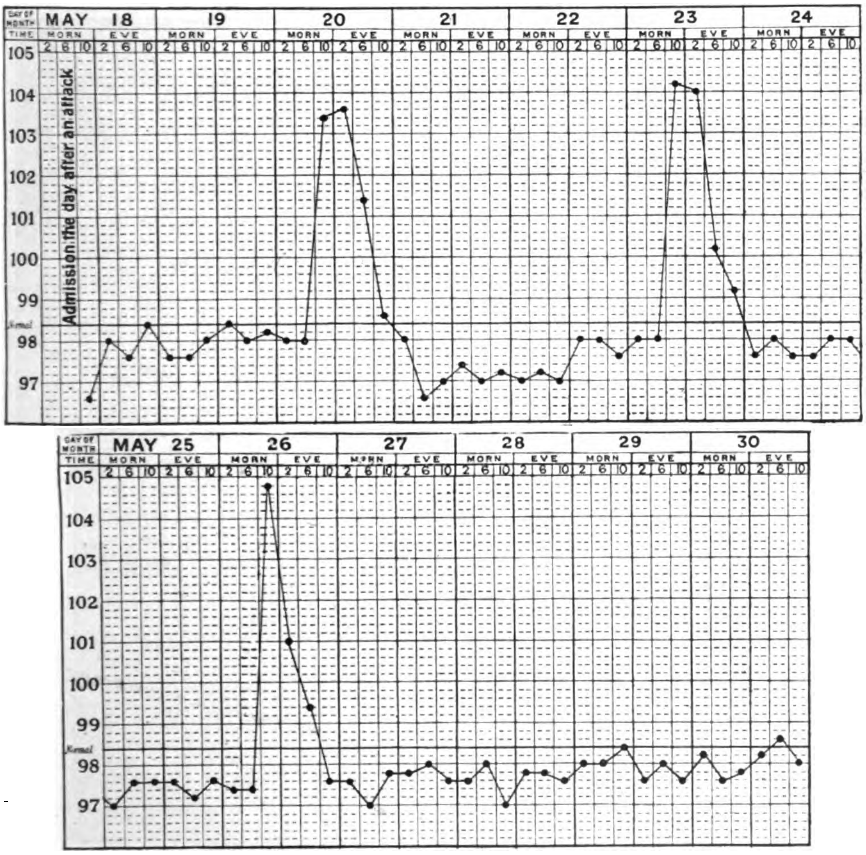


Fig. 7.—Case of quartan malarial fever, the attacks recurring every fourth day.
(*Chart supplied by the London School of Tropical Medicine.*)

—the *tertian*, in which the paroxysms come on every alternate day with complete freedom every intermediate day (*Fig.* 6); and the *quartan*, in which there are two-day intervals, so that the paroxysms occur every fourth day (*Fig.* 7). What happens in a malarial district, however, is that after a patient has been infected by one set of mosquito bites with a tertian or quartan ague, he becomes infected subsequently upon different days by other mosquitoes with other tertian or quartan parasites, so that there is a mingling together of the effects of different sets of hæmatozoa. For instance, if a patient had become infected by two tertian parasites, the one producing rigors upon Monday, Wednesday, Friday, and Sunday, and the other attacks upon Tuesday, Thursday, Saturday, and Monday, this patient would have a paroxysm every day, the type being then spoken of as *quotidian* (*Fig* 8). If he were infected by two quartan parasites, the one producing attacks upon Monday, Thursday, and Sunday, and the other upon Tuesday, Friday, and Monday, the occurrence of the paroxysms becomes less regular, for the patient would have a rigor upon Monday, another on Tuesday, none on Wednesday, a rigor upon Thursday and Friday, but none on Saturday, and so on. Each infection by a fresh brood of malarial parasites complicates the clinical picture, until finally in those who have been long in malarial districts the attacks of pyrexia may be quite irregular or even almost continuous. Each paroxysm has three characteristic stages, any one of which may last from half an hour to two or three hours, or even more. During the first or *cold stage*, the patient shivers with a severe rigor, feels cold, looks blue and pinched, but nevertheless has a rise of temperature to 102° F. or 103° F. The teeth chatter and the patient wraps himself up to try and keep warm. This is followed by the *hot stage*, which begins with flushing of the face, severe headache, pains in the back, further rise of the temperature to 104° F. to 106° F., and a sensation of such heat that the patient throws off the clothes and calls for cooling drinks. This ends in the third or *sweating stage*, during which the skin, previously dry, breaks out into perspiration so severe that all the bedclothes may be wringing wet. The temperature now falls, and the patient, more or less exhausted, sleeps, and on waking feels comparatively well except for a sense of weakness; he may be able to do his ordinary work until the next paroxysm comes on. Only in a few cases do much severer symptoms supervene if proper treatment be adopted. In the absence of treatment, however, malaria may lead to hyperpyrexia (107° F.–112° F.); to coma; or to a condition of algidity and collapse; any one of which may end in death.

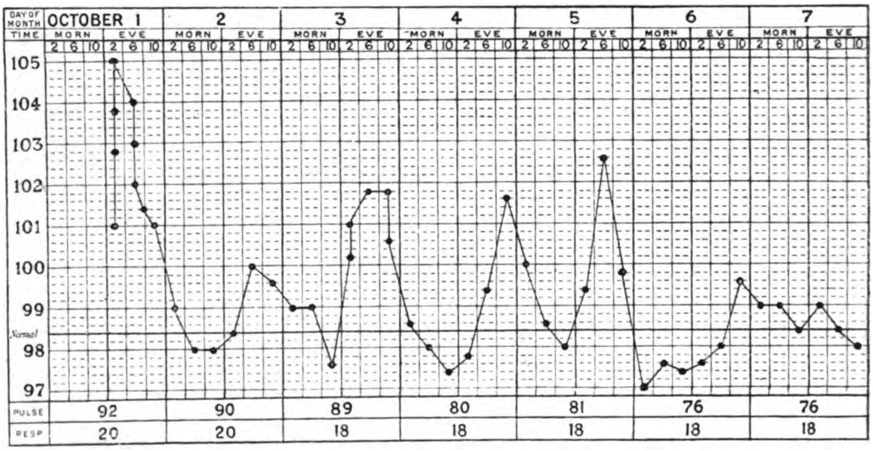


Fig. 8.—Case of malaria to illustrate quotidian fever due to double tertian infection. (*Chart supplied by the London School of Tropical Medicine.*)

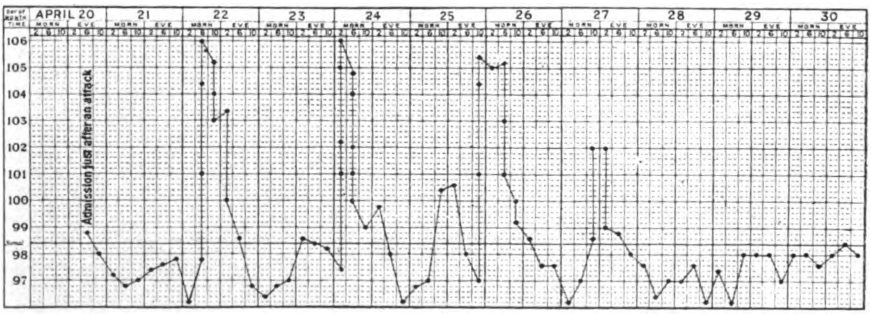


Fig. 9.—Case of malarial fever illustrating severe tertian attacks alternating with mild tertian attacks due to double infection. (*Chart supplied by the London School of Tropical Medicine.*)

The diagnosis may be confirmed to some extent by finding that the pyrexial outbursts diminish or cease altogether under the administration of quinine, but the only real proof of the nature of the complaint is the discovery in the blood of the malarial parasites (*Plate XXVIII, Figs. A, B, C, D,* and *E,* p. 614). It is important to note that the administration of quinine renders it difficult or impossible to find these in blood films, and then the behaviour of the leucocytes (p. 361) may be very helpful. Albuminuria is common, and the urine generally contains urobilin during active malaria, ceasing to do so when the latter becomes latent; microscopically, golden brown pigment granules are often to be found in the centrifugalized deposit; these and the urobilin together may point to the diagnosis when no parasites can be found in the blood. For a detailed account of all the stages and appearances of various malarial parasites, text-books of tropical medicine should be consulted. There are two main types to be seen in films stained by Leishman's method—the *ring-form* and the *crescent-form*. The latter are perhaps the rarer, though the severer types of malaria, particularly the æstivo-autumnal form met with on the West Coast of Africa, are generally due to it; the crescentic parasites cannot be mistaken for anything else. The ordinary tertian and quartan agues are due to the ring form of parasites, which,

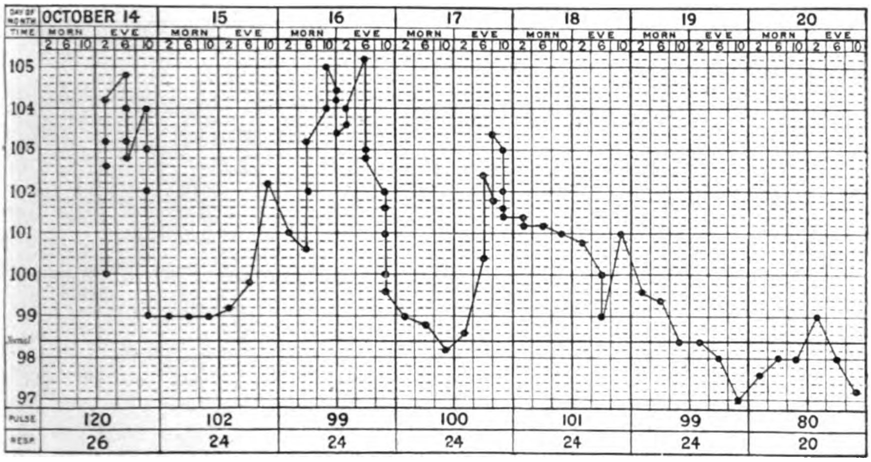


Fig. 10.—Case of malarial fever becoming complex from multiple malarial infection.
(*Chart supplied by the London School of Tropical Medicine.*)

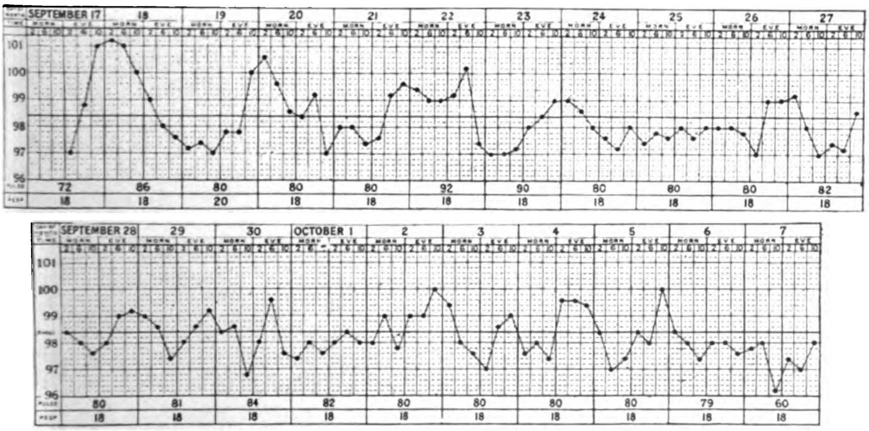


Fig. 11.—Chart to illustrate irregular pyrexia in chronic malaria.
(*Chart supplied by the London School of Tropical Medicine.*)

though the two types are distinct from one another, are sufficiently similar not to be distinguishable in films except by experts. If blood is examined at the beginning of the rigor the stage most commonly seen is that of *Plate XXVIII, Fig. B*, p. 614. The two chief points of morphological distinction between tertian and quartan parasites are, first, that the pigment granules are much blacker and fewer in number with the quartan than the tertian, and secondly, that in the rosette stage the quartan segments are fewer than the tertian. One remarkable feature about malaria is that it may remain latent for many years, and yet recur in those who have long since returned to Great Britain from the tropics. What has happened to the parasites in the interval is not known, but their re-appearance is brought about by such conditions as general depression of health from overwork or worry, or as the result of some intercurrent malady.

(*B*).—ANÆMIAS WITH AN INDETERMINATE OR NEGATIVE BLOOD PICTURE.

The diagnosis of the fact of anæmia is made by means of a blood-count, but in the great majority of cases the cause of the anæmia itself is not indicated by the blood condition. The differential diagnosis has to be made on other grounds. One may subdivide *Group B* into four sub-groups, namely, (1) Those cases in which the anæmia is slight and in itself not a very prominent symptom ; e.g., in an indoor worker or a convalescent : (2) Those cases in which, though the anæmia may be severe, the routine examination of the patient discovers some more or less obvious and not absolutely uncommon cause for it ; e.g., chronic tubal nephritis : (3) Those cases in which, though the anæmia may be severe, no obvious lesion can be discovered, but in which there is nothing about the case to suggest that the condition is a rare or unusual one ; e.g., chlorosis : (4) Those cases in which the anæmia may be more or less severe, in which there may or may not be obvious lesions to account for it, but in which the circumstances of the case suggest that the disease is unusual or rare ; e.g., chloroma.

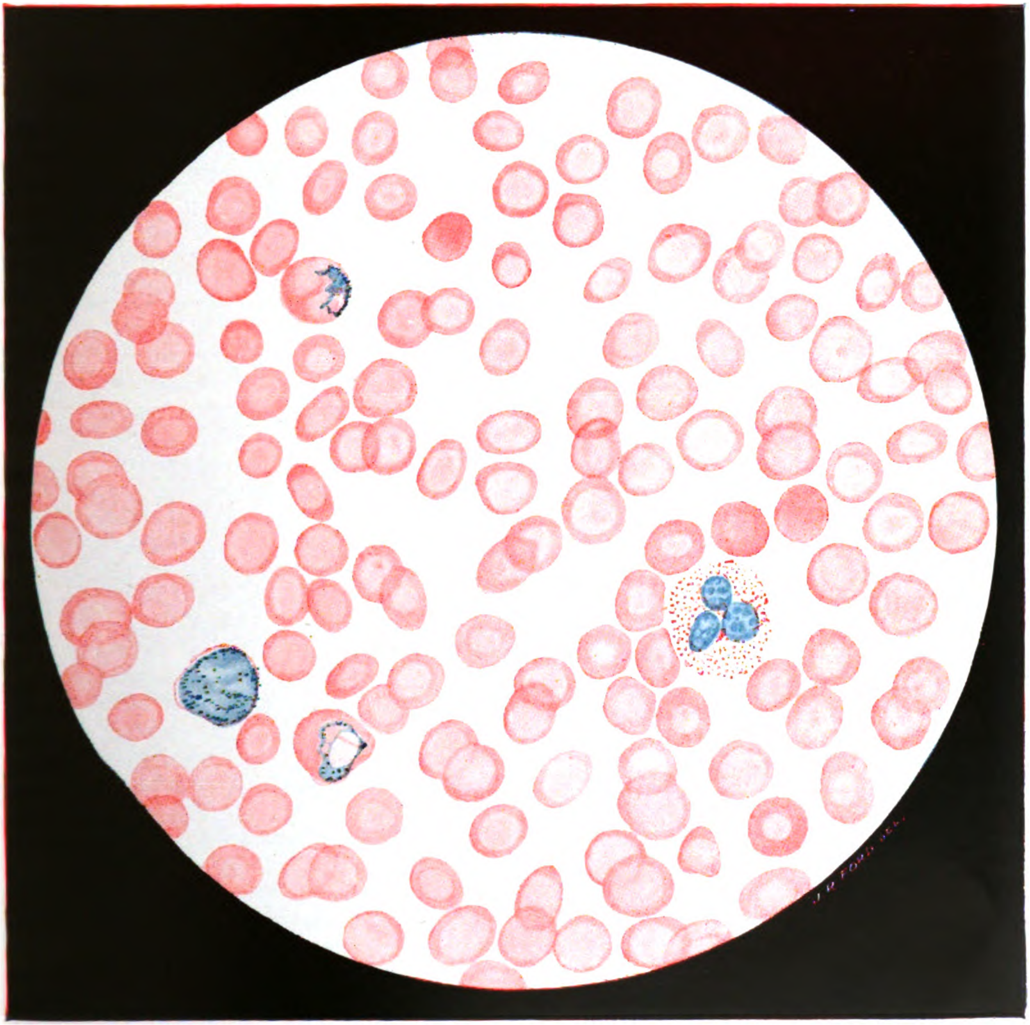
Cases in which the Anæmia is slight and in itself not a very prominent symptom.—It is clear that before any anæmia that is not due to acute blood loss from internal or external hæmorrhage reaches a severe stage, it must pass through a phase in which it may be regarded as slight or mild. This group therefore really includes all the other groups at some stage of their development, and the diagnostician will often label a case to start with comparatively mild or unimportant, when the course of events ultimately shows that this was wrong. For instance, a case of pernicious anæmia may exhibit what seems to be unimportant symptoms for months or years before the anæmia reaches so definite and severe a stage as to be diagnosed correctly. The group now under discussion is meant to include only such slight degrees of anæmia as are themselves not important in the matter of diagnosis ; for instance, in people who live too much indoors, in those who are convalescent from some illness, in those who suffer from chronic indigestion, constipation, obesity, some forms of chronic intoxication by microbial products, due to such things as infective synovitis or arthritis, pyorrhœa alveolaris and oral sepsis, uterine or ovarian disease, the earlier stages of phthisis, empyema, latent or deep-seated caseous glands or tuberculous affection of joints, vertebræ, or peritoneum in children, the milder cases of plumbism, and so on ; in all these cases there may be a sufficient degree of anæmia to attract some attention, but the diagnosis will rest upon other symptoms and signs than those connected with the blood, and in most cases the anæmia will not be extreme.

Cases in which, though the Anæmia may be severe, a routine examination of the patient discovers some more or less obvious and not absolutely uncommon cause of it.

Hæmorrhage.—Some of the most striking cases of anæmia in this group are those in which there has been recurrent or severe loss of blood. When the latter has been lost by epistaxis, hæmoptysis, hæmatemesis, hæmaturia, menorrhagia, metrorrhagia, metrostaxis, purpura, or by the escape of blood per rectum, the nature of the anæmia will generally be obvious, and the differential diagnosis will depend upon the cause of the particular hæmorrhage in question (see EPISTAXIS, etc.). One should insist upon a complete blood-count in all these cases, however, in order to exclude pernicious anæmia, leukæmia, and the other conditions in which the blood-picture is positive, lest the bleeding be due to the blood state and not the blood state to the bleeding. The possibility of melæna should also be borne in

PLATE VI

MALARIA



Part of a blood film from a case of malaria, showing three malarial parasites of the ring type.

mind, for without examination of the fæces the extreme pallor resulting from loss of blood from such a lesion as a duodenal ulcer may not be diagnosed correctly. *Hæmophilia* should not be forgotten ; the way the patient bleeds excessively from slight scratches or cuts will generally point to the diagnosis, especially if there is a family history of a similar condition, males being affected more than females. The blood-picture in hæmophilia is entirely negative, the anæmia that results from the bleeding being of the chlorotic type. It is sometimes stated that the result of blood-loss is to produce an anæmia in which the red corpuscles and the hæmoglobin are equally reduced, so that the colour index remains more or less normal. This may be true of an acute bleeding such as venesection or post-partum hæmorrhage, but the effect of recurrent blood-loss is to produce the chlorotic type of anæmia, in which the red corpuscles are less diminished than is the hæmoglobin.

Cachexia.—A similar blood picture, namely an anæmia of the chlorotic type more or less severe, but without anything which may be called pathognomonic, either as to the red cells or the leucocytes, is to be found in almost all forms of cachexia, whether due to syphilis, tuberculous or malignant disease, malaria, beri-beri and other tropical illnesses, œsophageal stenosis, or starvation. A careful physical examination of the patient and enquiry into his symptoms may point to the correct diagnosis ; but it is to be borne in mind how difficult it sometimes is to detect phthisis, or some cases of carcinoma or sarcoma, even when far advanced. Sputum analysis should not be omitted ; rectal examination should not be forgotten ; the *x*-rays may serve to detect lesions within the thorax, and Wassermann's serum reaction may be employed when syphilis is suspected. It is remarkable how little anæmia may result from some varieties of cancer, particularly carcinoma of the breast : whilst other varieties, especially carcinoma of the stomach, produce progressive anæmia comparatively early. It is noteworthy that, whereas in former times the absence of free hydrochloric acid from the gastric juice at the proper interval after a test meal was regarded as good evidence in favour of a carcinoma ventriculi, it has now been established firmly that the hydrochloric acid may be very deficient or entirely absent in a great many other conditions also ; it is absent in almost all cases of advanced carcinoma, whether of the stomach or not ; and in many chronic maladies associated with ill-health all the secretions of the body suffer, and amongst them the hydrochloric acid of the gastric juice. It follows, therefore, that it is only when the diagnosis has been narrowed down to there being some lesion of the stomach, that the discovery that the hydrochloric acid is very deficient or absent affords evidence that the lesion is a carcinoma.

Parasitic affections sometimes escape recognition, even when they have led to sufficient anæmia to attract attention (see PARASITES, INTESTINAL, p. 519). The two varieties most apt to be associated with anæmia are *Ankylostomum duodenale* and *Bothriocephalus latus*. *Bilharzia hæmatobia* may also lead to severe anæmia, but generally does so on account of the HÆMATURIA (p. 282) that it produces. Eosinophilia (p. 218) may suggest a parasitic infection.

Certain *drugs* are apt to produce anæmia of the simple chlorotic type if their administration is continued over a long period ; particularly *mercury*, *arsenic*, *lead* and *salicylates*. Acute mercurialism is commonly associated with stomatitis and salivation, but in chronic cases, in addition to anæmia, there is apt to be a motor type of peripheral neuritis affecting the limbs and associated with a remarkable tremor (p. 726), particularly of the hands. The diagnosis is generally arrived at from the fact that the patient has been receiving mercury medicinally, or is employed in some work in which mercury is used, for instance, the making of thermometers or mirrors, or the curing of rabbit skins for the manufacture of hats. *Arsenical poisoning* seldom gives rise to anæmia as its sole symptom ; but it is noteworthy that although liquor arsenicalis is an admirable remedy for the relief of pernicious anæmia, arsenic itself is also a cause of anæmia amongst those who work in it. As a rule, in addition to anæmia there is marked pigmentation of the skin (*Plate VII*), and Addison's disease may be simulated. In the latter, however, the pigmentation occurs on the mucous membranes, particularly of the lips and cheeks, as well as upon the skin, and this—though in very exceptional cases a similar pigmentation within the mouth has been observed in pernicious anæmia (see *Plate XXII*. p. 528), and perhaps after taking arsenic for long periods—is always very suggestive of Addison's disease, and the diagnosis may be confirmed by finding a slight degree of eosinophilia, a remarkably low blood-pressure, down even to 80 mm. Hg or less, attacks of vomiting, syncope, and remarkable asthenia.

If there is active tuberculosis of the suprarenal capsules, Calmette's or von Pirquet's reactions with tuberculin (*Plate XXXVII*, p. 770), may be positive, but these two tests are now less relied on than formerly. In arsenical cases there may also be evidence of peripheral neuritis and of hyperkeratosis of the soles and palms. Analysis of the hair will discover an abnormally high percentage of arsenic. The chlorotic type of anæmia in *lead poisoning* may be extreme, but the diagnosis will depend upon other symptoms, of which any or all of the following may occur :—a blue line upon the gums ; constipation ; nausea ; vomiting ; epigastric pains ; abdominal colic ; a tendency to repeated abortion in women ; peripheral neuritis, particularly of the wrist-drop type ; various cerebral symptoms of any degree, from mere headache or insomnia to epileptic convulsions or acute mania, or other serious mental signs summarised by the term saturnine encephalopathy ; impairment of sight ; optic neuritis ; ophthalmoplegia, chiefly affecting the sixth cranial nerve ; a tendency to gout, albuminuria and granular kidney, and the secondary effects of the latter. The absence of a blue line on the gums does not exclude lead poisoning in those whose teeth are clean ; nor does its presence prove lead poisoning, for most workers in lead exhibit a blue line whether they have other symptoms or not. In cases of doubt, it may be necessary to collect an abundance of urine, evaporate it, and apply the ordinary tests for inorganic lead. The occupation of the patient will often suggest the diagnosis. *Salicylates* are said to produce anæmia if their administration is continued for a long period ; but it is also possible that the anæmia may be due to the condition for which the salicylates are being given, namely *acute rheumatism*. The diagnosis is generally obvious.

In addition to the anæmia that may result from acute rheumatism itself, there is apt to be pronounced anæmia in some forms of valvular heart disease, pa ticularly *affections of the aortic valves*, whether rheumatic or syphilitic. Mitral disease, particularly mitral stenosis, is more likely to cause polycythæmia (p. 533), unless there is *fungating* or *infective endocarditis*. The occurrence of a progressive anæmia in chronic heart cases always arouses suspicion of the latter ; most cases of fungating endocarditis present symptoms of failing compensation which are often very difficult to distinguish from those due to the mechanical effects of chronic valvular disease, so that it is often difficult to distinguish a heart case without fungating endocarditis from one in which fungating endocarditis has supervened. In addition to anæmia the following points would be in favour of the latter : sudden and radical changes in the character of the heart bruits, for instance from musical to blowing, and vice versa ; enlargement of the spleen ; the occurrence of hæmorrhages, particularly subcutaneous or retinal ; optic neuritis ; pyrexia (*Fig.* 243, p. 566), whatever its type, provided it cannot be explained by any intercurrent affection such as tonsillitis or pleurisy —though the absence of pyrexia does not exclude the disease ; rigors, though these are often absent ; and symptoms of infarction or embolism in the spleen, kidney, brain, intestine, retinal or peripheral vessels resulting in convulsions or paralysis ; cessation of pulse in one or other of the accessible arteries such as the radial, posterior tibial or dorsalis pedis ; acute gangrene of some part whose circulation has thus been cut off suddenly—a toe, or the tip of the nose for example ; the development of a spontaneous peripheral aneurysm ; sudden hæmaturia ; sudden acute pain over the spleen, associated perhaps with a peritonitic rub. It is noteworthy that there is but little leucocytosis in infective endocarditis. Cultivations from the blood obtained by aseptic venesection may serve to clinch the diagnosis, and also to indicate what serum or vaccine treatment should be employed ; though it is remarkable how often blood cultures are negative in these cases, even when the blood is obtained during a period of high pyrexia.

It is in some cases easy, but in others relatively difficult, to be sure of the diagnosis of *subacute nephritis*. Anæmia is a prominent symptom in the chronic nephritis of young people, though the reverse is generally the case in the red granular kidney of later life ; for the differential diagnosis, see ALBUMINURIA (p. 9). The old aphorism of " the large white person with the large white kidney " may sometimes suggest the malady.

Many subacute or chronic maladies associated with continued absorption of microbial toxins have anæmia as a prominent symptom. One may mention, for instance, *chronic colitis*, whether muco-membranous, 'simple' ulcerative, or tropical dysenteric (see DIARRHŒA, p. 172) ; deep-seated *suppuration* acts in the same way, and one is familiar with the pallor of patients suffering from empyema ; the development of this anæmia after the crisis of lobar pneumonia, or in connection with broncho-pneumonia, in children, not infrequently suggests that an empyema has developed; the diagnosis may be confirmed by the physical signs, but it will be clinched by finding pus when the chest is needled. Leucocytosis or a relative increase in the polymorphonuclear cells does not help in determining the presence of empyema so much as in other cases of suppuration, because empyema is nearly always secondary to lobar or lobular pneumonia, and in each of these there is also a polymorphonuclear leucocytosis. Other examples of chronic sepsis which may produce severe anæmia are chronic appendicular abscess; pyosalpinx; hepatic abscess; the breaking down of ovarian or uterine tumours; chronic endometritis; pyorrhœa alveolaris; infection of sinuses connected with bones or joints, particularly unclean tuberculous hip or knee joints; psoas abscess; suppurative periostitis or osteomyelitis, with necrosis of bone; secondary coccal infections in phthisis with cavitation, or in bronchiectasis. Chronic sepsis may produce *lardaceous disease*, which itself is also a cause of profound anæmia, with a peculiar pale yellowish or transparent appearance of the skin, though its diagnosis is exceedingly difficult in any but advanced cases. It is guessed at, as a rule, on account of there being a chronic purulent discharge from lung, joint or limb, or else severe tertiary syphilis. There may be enlargement of the liver and spleen, albuminuria, and a tendency to diarrhœa; but even when all these symptoms are present, it not infrequently happens that the post-mortem examination shows that there was no lardaceous disease at all.

Rheumatoid arthritis is an indefinite group of joint diseases which differ essentially from osteo-arthritis (p. 243), in that with the former there are more or less severe constitutional symptoms, including slight pyrexia, loss of appetite and weight, pigmentation of the skin, and anæmia. The nearest lymphatic glands, e.g., the epitrochlear when the hands are affected, are often enlarged and tender. The diagnosis seldom depends upon the anæmia, however. Probably there are many varieties of rheumatoid arthritis which will some day be classified upon a bacteriological basis into those due to gonococci, streptococci, staphylococci, pneumococci, *Bacillus coli communis*, *Spirochæta pallida*, and so on. There are two types that are particularly prone to anæmia, and these are, first, the form in which there is marked spindle-shaped enlargement of all the first interphalangeal joints in adults, whatever other joints may be affected at the same time (p. 342); and secondly, a general destructive affection of the joints in children, associated with emaciation, anæmia, enlargement of the spleen and of the lymphatic glands, and known as *Still's disease* (*Fig.* 169, p. 377). (See JOINTS, AFFECTIONS OF, p. 337.)

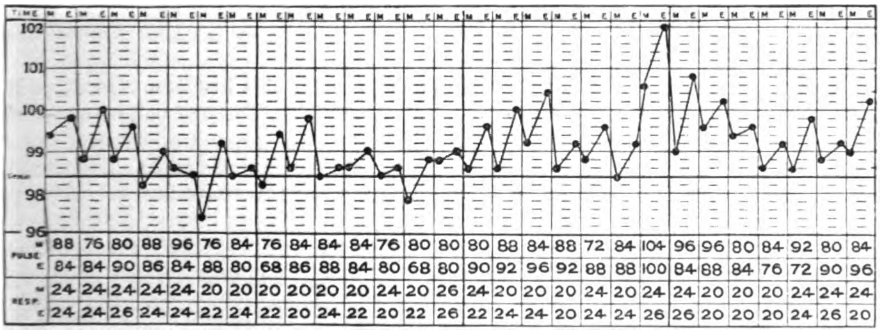


Fig. 12.—Temperature chart (morning and evening) from a case of cirrhosis of the liver, showing the tendency to slight pyrexia, especially in the evening.

Cirrhosis of the liver sooner or later leads to anæmia of the chlorotic type, although in the earlier stages the alcoholic patient may have a rubicund complexion; by the time the anæmia is pronounced there will almost certainly have been other symptoms of the complaint, particularly HÆMATEMESIS (p. 265), JAUNDICE (p. 324), or ASCITES (p. 43). Patients with cirrhosis of the liver often have some evening pyrexia (*Fig.* 12), and they tend to undue pigmentation of the skin.

Hyperlactation is a prominent cause of anæmia and general ill-health, especially in women in towns. The cause for prolongation of the period of lactation is often an idea that pregnancy will not recur whilst the last infant is being suckled. The diagnosis is generally obvious if its possibility is borne in mind.

Gastric ulcer, or rather the symptoms which are often stated to be those of gastric ulcer, is frequently associated with anæmia; the latter in a few cases is the result of direct loss of blood by HÆMATEMESIS (p. 268), or, in the case of duodenal ulcer, MELÆNA (p. 75) A duodenal ulcer may sometimes simulate gastric ulcer, but more often it produces symptoms which are apt to be mistaken for gall-stones, the pain being referred to a spot about an inch below the tip of the ninth right rib. As a rule the pain in cases of duodenal ulcer bears a definite relationship to food, being greatest when the patient is beginning to be hungry, and relieved by the taking of food. Gastric ulcer, on the other hand, is much more difficult to diagnose, for even when the patients have suffered from epigastric pain coming on immediately after food, from vomiting which relieves the pain, and from one or more attacks of hæmatemesis, it is possible for the latter to be due to generalized oozing from the gastric mucosa—" gastrostaxis "—rather than a definite measurable ulcer. When there has been no hæmatemesis the diagnosis is still more difficult, though it is noteworthy that in nearly half the cases in which the presence of an ulcer has been proved by operation there has been no history of hæmatemesis. It was formerly stated that gastric ulcers are common in the female sex between the ages of fifteen and thirty, especially in the unmarried and the anæmic; notably amongst the servant class; operative demonstrations of gastric ulcers, however, seem to show that they are really commoner in later life, and affect men as often as women, so that there is a very decided possibility that the gastric symptoms of anæmic women are not in fact due to ulcer. One meets with patients who have pain the moment they take food, in whom vomiting after meals is persistent, in whom the diagnosis of gastric ulcer would certainly have been made in former years, but in whom that diagnosis is made now only with considerable caution. It has become increasingly recognized that the vomiting and the gastric signs are often due to the anæmia itself, and result from anæmic dilatation of the heart. In diagnosing between this condition and that of true gastric ulcer, one of the best plans is to put the patient to bed, and when she has been recumbent for twenty-four or thirty-six hours, to see what is the effect of giving her full diet. Full diet will be borne quite well in cases of severe anæmia associated with gastric symptoms without ulcer so long as the patient remains in bed; but if she gets up and returns to work before the anæmia is cured the gastric symptoms come on again directly. The vomiting and the epigastric pain seem to be related not so much to food as to work in these cases. When there is an ulcer, however, an attempt to adopt full meat and vegetable diet on the second day of resting in bed nearly always fails if there have been severe symptoms up to that time.

Conditions in which, though the Anæmia may be severe, no obvious Lesion can be discovered, whilst at the same time there is nothing to suggest that the case is a rare or unusual one.

Chlorosis is almost the only malady which comes under this heading, if one includes the milder anæmias of girls and young women as well as the severe cases of yellow-green sickness to which the term should strictly speaking be limited. The cases of anæmic vomiting just discussed might also come under the same heading. Chlorosis and simple chlorotic anæmia, without obvious organic lesions, are affections of the female sex—absent before puberty and common immediately after, seldom lasting after thirty years of age, and generally not so long; cured as a rule by marriage; never fatal even when severe; an affection of all classes, but mostly of indoor workers such as servant girls, and not often affecting those who are employed in outdoor pursuits. The diagnosis is generally easy. The patients are comparatively well covered though they often eat very little. Emaciation is rare in chlorosis, and this is probably due to the fact that the blood is less deficient in quantity than diluted by excess of water. The leucocytes are normal both in total number and in differential count. The red corpuscles are often much less diminished than might be expected from the appearance of the patient, the chief feature of the complaint being the great reduction in the hæmoglobin, so that the colour index may fall to 0·5, 0·4, or even less. As the condition improves the red cells return to normal fairly quickly, and the hæmoglobin rises steadily but less rapidly. The way in which the patients react to treatment by rest in bed, by the giving of iron, by keeping the bowels open, and by living in a sunny atmosphere, is remarkable, and helps to clinch the diagnosis in any case of doubt. It has been mentioned above that there are many blood changes which are common to severe anæmias; it should be noted that even when the hæmoglobin has fallen to 30 per cent of normal in a severe case of chlorosis, the changes in the blood-cells enumerated on page 22 seldom appear. Chlorosis, more often than any other form of anæmia, leads to hæmic cardiac bruits, particularly a blowing systolic bruit in the pulmonary area and a *bruit de diable* in the neck. The patients are often constipated, are apt to suffer from menstrual irregularity, particularly amenorrhœa, which may last for months, and a tendency to œdema of the feet. The viscera are generally normal. Chlorosis, unlike many other forms of severe anæmia, seldom produces albuminuria.

Cases in which the Anæmia may be more or less severe, in which there may or may not be obvious lesions to account for it, but in which the circumstances of the case suggest that the disease is unusual or rare.

Hodgkin's disease is often spoken of as though it were an affection in which the blood-count indicates the diagnosis. This is not the case, however, the blood changes being merely negative, though a blood-count is essential in order to exclude leukæmia by finding that there is no leucocytosis. At first there is no anæmia ; later there is progressive anæmia of the chlorotic type, with finally all the changes in the red cells common to the severe anæmias (p. 22). There is no leucocytosis, or none of moment. The differential leucocyte count may be normal; more often, however, there is some relative increase in the lymphocytes with proportionate relative diminution in the polymorphonuclear cells, and when a large number of white corpuscles are examined occasional myelocytes and one or two basophile corpuscles will be detected. The diagnosis is made from the enlargement of the LYMPHATIC GLANDS (p. 377) and of the SPLEEN (p. 635).

Splenic anæmia is a malady in which there is considerable enlargement of the spleen, progressive anæmia of the simple chlorotic type, and no other very obvious evidence as to what is wrong with the patient. It is probable that more than one condition is at present labelled splenic anæmia ; a considerable number of the cases turn out ultimately to be cirrhosis of the liver (p. 433), in which enlargement of the spleen happens to have been the first symptom to attract attention, very likely years before the other effects of cirrhosis manifested themselves. When splenic anæmia is the original diagnosis in a case which ultimately proves to be cirrhosis of the liver, the condition is often spoken of as *Banti's disease*.

Aplastic anæmia has been mentioned above (p. 24), and there are a considerable number of obscure cases of severe anæmia to which up to the present no definite labels can be attached. Some of these simulate pernicious anæmia, but all differ from the latter in having a colour index persistently less than 1. One can only refer to them as severe and even fatal un-named anæmias.

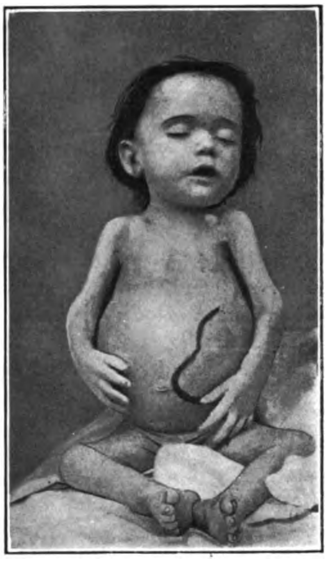


Fig. 13.—Pseudo-leukæmia infantum (von Jaksch's disease). The black line demarcates the spleen.

Pseudo-leukæmia infantum is a condition in which enormous enlargement of the spleen takes place in a young child or infant (*Fig.* 13), associated as a rule with more or less ascites and enlargement of the abdomen. So great is the splenic enlargement sometimes that the condition at first suggests leukæmia ; but when a blood-count is made, although the red cells may be very much diminished and exhibit all the changes characteristic of severe anæmia, there is no extreme leucocytosis, so that the condition cannot be classified as a leukæmia, and hence is termed 'pseudo-leukæmia infantum ;' it has also been called '*von Jaksch's disease*.' It generally begins at an age of less than two years. The liver is enlarged, but less so than the spleen. There may be severe hæmorrhage from the mucous membranes, and there is often periodic pyrexia. The disease may be mistaken for rickets or for congenital syphilis ; indeed some authorities think that it is really due to one or other or both of these causes in an exaggerated degree. Whether this is so or not, the prognosis is fair even when the anæmia has reached a severe degree. The ascites may disappear, the huge spleen may become restored to its normal dimensions, and the patient recover completely in the course of months.

Myxœdema is a condition which may be mistaken for simple anæmia, and consequently it is apt to be overlooked, particularly at that stage which merits the term 'hypothyroidism' rather than myxœdema. It is an affection of women rather than of men; it comes on very slowly, and sometimes it can be diagnosed only by watching the beneficial effects of thyroid treatment. There is generally excess of gelatinous subcutaneous tissue, which gives the patient a puffy or œdematous appearance, especially in the face (*Fig.* 15), hands (*Fig.* 16), and lower limbs, so that not a few cases are mistaken for nephritis. The urine is copious and of low specific gravity, but usually does not contain albumin; though in some cases there is sufficient albuminuria to make the case still more like one of Bright's disease. The apparent œdema does not pit on pressure, or pits far less easily than it would if it were ordinary œdema; the skin becomes thickened, and the hair decreases in quantity and becomes brittle. Physical movements are lethargic, and the intellect dull, so that there is slowness of action both of body and of mind, symptoms that disappear in a remarkable way under thyroid treatment. In some cases the mental symptoms predominate to such a degree that some form of delusional insanity or dementia may be diagnosed, or even a cerebral tumour. The chlorotic type of anæmia which accompanies myxœdema may be masked by a local flush over the malar bones, not unlike that of mitral stenosis.

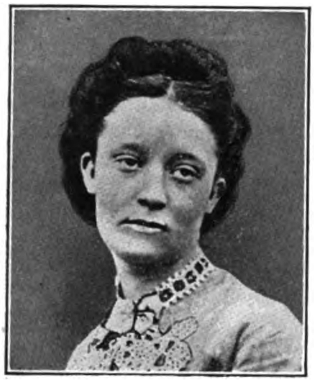


Fig. 14. The same patient as *Fig.* 15, previous to the development of myxœdema.

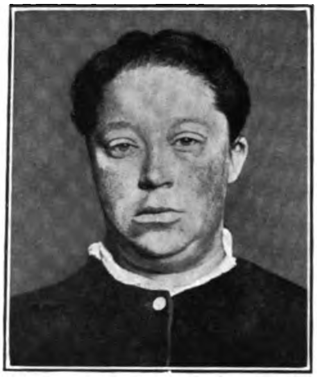


Fig. 15.—Myxœdema: the characteristic facies, illustrating the broadening of the features and the malar flush. (Compare *Fig.* 14.)

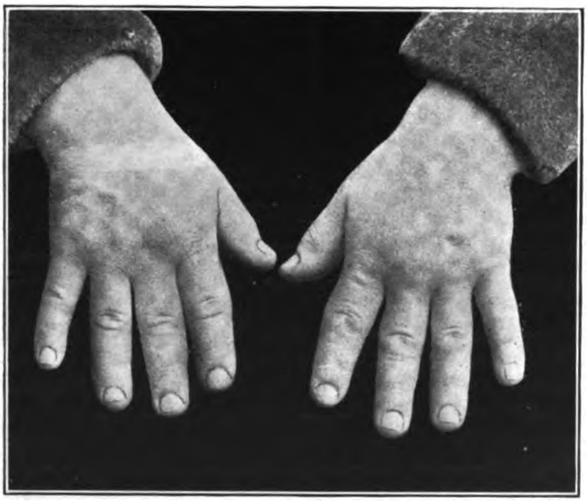


Fig. 16.—Hands of a patient suffering from myxœdema, illustrating the swelling of the soft parts, the broadening of the fingers, and their consequent stumpy or podgy appearance.

Scurvy is a rare disease which may lead to the most profound anæmia, though it seldom does so without also producing extensive hæmorrhage into the skin, beneath the periosteum of the tibiæ or other bones, from mucous membranes, and especially from the spongy and fœtid gums. It is not common now-a-days, except in a mild form in children,—*scurvy-rickets* or *Barlow's disease*—in which tenderness of the bones associated with anæmia, often mistaken for rickets, is the main symptom. The tenderness in question is due to local sub-periosteal hæmorrhage, and the way in which the complaint rapidly gets better under suitable treatment with fresh vegetable diet helps in clinching the diagnosis. The severer forms of scurvy are due to prolonged deprivation of fresh food, such as is rare in modern practice, though it used to be common on board ships.

Chloroma is a very rare affection, related to lymphatic leukæmia on the one hand and to lymphosarcoma on the other. It is associated with the formation of multiple tumours, especially in connection with bones, and a progressive and severe anæmia of indeterminate type. The condition is fatal, and the diagnosis is at once suggested by the green colour of the neoplastic deposits. *Herbert French.*

ANÆSTHESIA.—(See SENSATION, ABNORMALITIES OF, p. 604.)

ANALGESIA.—(See SENSATION, ABNORMALITIES OF, p. 604.)

ANASARCA.—(See ŒDEMA, p. 411.)

ANKLE-CLONUS is best elicited when, the patient lying on his back, with his knees slightly flexed, the observer quickly, but not violently, dorsiflexes the foot, the hand being applied along its outer border in such a way as to keep it well outwardly rotated. The result, when ankle-clonus is present, is a series of rhythmical jerks at the ankle-joint, at the rate of about 7 per second—*the contractions continuing as long as the pressure is maintained.* The last proviso is important, because it often happens that a few ankle-jerks are obtained, varying in number from two or three to as many as twenty or thirty, but gradually tailing off and ceasing, although the pressure on the sole is maintained. This is sometimes spoken of as a "tendency to ankle-clonus," but for clinical purposes it is not ankle-clonus at all, and indicates nothing more than hypersensitiveness of the nervous system, and not organic disease. Ankle-clonus, on the other hand, denotes changes in the corresponding crossed pyramidal tract, and it is to be expected in association with increased knee-jerk and extensor plantar reflex. Its chief value lies in determining between functional and organic exaggerations of the knee-jerk ; the latter may be very brisk as the result of pure nervousness, but if it is associated with either an extensor plantar reflex or ankle-clonus, or both, the exaggeration is due to organic disease of the upper neuron, hemiplegic or paraplegic as the case may be. Whereas, however, the presence of maintained ankle-clonus is conclusive proof of an upper neuron affection, the absence of such clonus does not exclude such lesion ; ankle-clonus is not met with until there is a relatively large amount of lateral column change ; it comes later, as a rule, than the extensor plantar reflexes. *Herbert French.*

ANOSMIA.—(See SMELL, ABNORMALITIES OF, p. 611.)

ANURIA—or suppression of urine—must be distinguished from *retention of urine,* in which urine is secreted from the kidneys, but is retained in the bladder from some lesion causing obstruction to the urethra, such as urethral stricture or prostatic obstruction in the male, or pressure or drag upon the urethra by a large pelvic tumour or a retroverted gravid uterus in the female. Retention of urine may also occur without urethral obstruction in various forms of disease of the spinal nervous system affecting the lumbar centres. In retention of urine there is pain above the pubes, constant and urgent desire to pass urine, and the distended bladder forms a tense, oval, dull tumour above the pubes in the middle line. In many cases a previous history of obstruction to the urinary flow will be obtained, whilst in others the involuntary dribbling of urine from the urethra from an over-distended bladder at once distinguishes the case from one of anuria.

CAUSES OF ANURIA.

A.—Obstructive :—
- Calculus in kidney or ureter
- Vesical carcinoma involving the ureteric orifices
- Uterine carcinoma
- Large pelvic or abdominal tumours.

B.—Non-obstructive :—
- Toxic, hæmatogenous or ascending.

In renal disease, nephritis, lardaceous disease, tuberculosis, polycystic disease, suppurative pyelonephritis
Reflex, after operations or trauma. or the sudden emptying of an over-distended bladder.
In poisoning from mercury, lead, phosphorus, oxalic acid, cantharides, or turpentine.
In severe collapse.
Hysteria.

Anuria may occur and be complete without any other symptom, and it is remarkable that in the obstructive forms, especially with calculus, anuria may be complete for several days without any other symptom—latent uræmia. In the non-obstructive forms, anuria may be accompanied from the onset by the various symptoms of uræmia, such as vomiting, convulsive muscular twitchings, dyspnœa, and headache. In the obstructive form there may be total absence of any urine secreted, or a small quantity may be passed of low specific gravity, and containing very little urea or solids. Albumin is absent unless there be hæmaturia or cystitis, when pus may be present also. The patient may complain of aching in one or both lumbar regions, but, with the exception that no urine is passed, seems to be in ordinary health. The appetite is good and the mental state clear; but after a variable period, from seven to ten days, the patient becomes drowsy, the tongue dry, temperature subnormal, appetite deficient, and pupils small. There may be muscular twitching; the drowsiness gradually becomes deeper, without any true uræmic convulsions, and death may be postponed for as long as twenty days from the onset of the anuria. This sequence is very different from that seen when anuria occurs from non-obstructive causes, when there is frequently marked disturbance of the nervous system; headache and giddiness are followed rapidly by convulsions, delirium, and dyspnœa, with vomiting and small pupils, the patient rapidly becoming comatose and dying in a few days.

A.—OBSTRUCTIVE ANURIA.

Calculous Disease is the most frequent cause of obstructive anuria. It may occur at any age, but is commonest in men of about forty. Suppression of urine may arise from the impaction of a small calculus in the ureter of a kidney which is practically normal, or may be due to the total destruction of the renal secreting substance, which has progressed gradually and without marked symptoms. Between these two extremes there may be many stages, and the two conditions, namely, ureteric impaction and renal destruction, may exist at the same time. Clinically, it is rare for calculous anuria to arise from simultaneous blockage of both ureters by calculi; it is less uncommon to find that one kidney has been destroyed by previous disease, the ureter of the remaining organ then becoming obstructed by a stone. Exceptionally, the blockage of one ureter may cause reflex suppression of urine in the other kidney, especially if the function of the latter is impaired already by disease; but in these cases the anuria is usually temporary. Calculous anuria may occur suddenly, and in patients who are apparently in good health, for it is no uncommon thing for a patient to go on in good health when he possesses only one functionally active kidney, the other having been destroyed by slow disease, or being absent; or there may be a history of previous lumbar pain, hæmaturia, pyuria, or the passage of calculi. At the onset of anuria there is usually pain in the lumbar region along the course of the ureter of the side most recently affected; it commonly lasts a day or so and then subsides, or it may last throughout the period of anuria. In addition, there is frequently a constant desire to micturate, although no urine is passed, or if the anuria is intermittent, urine of pale colour and low specific gravity, sometimes blood-stained, may be passed. If the anuria remains complete, no other symptoms may occur for several days, a feature which is common to the obstructive forms of anuria, but is in marked contrast to the non-obstructive variety. After a period of anuria lasting from seven to ten days, the patient becomes drowsy, the tongue is dry, there is disinclination for food, and the general symptoms of uræmia may come on; but in many cases the patient may die before any symptoms of uræmia occur. Thus, it is usual to speak of a *tolerant* and a *uræmic period* in obstructive anuria. The tolerant stage of obstructive anuria may be even further prolonged if the functional kidney be already hydronephrotic from previous intermittent obstruction, even to twenty days. The sudden obstruction to the urinary flow in a comparatively normal kidney causes complete suppression, whilst a partial or intermittent obstruction causes dilatation of the kidney. If such a kidney be the functionating organ, and become completely obstructed, the dilatation will increase; and a lumbar tumour may be palpable. If there is pain on pressure over the kidney, or along the course of the ureter, the diagnosis is strengthened, or it may be decided to settle the diagnosis by immediate operation. In some cases in which one kidney has been destroyed gradually without pain, and anuria occurs, there may be great difficulty in determining which of the two kidneys is the functional organ which has recently become obstructed; in these cases it is a good rule to operate upon the side on which the pain has occurred most recently. If the patient is not too stout, palpation may detect a distinct area of pain over a calculus impacted in the course of the ureter; or on careful rectal or vaginal examination a calculus impacted in the vesical end of the ureter may be felt. If the case is seen early, evidence of ureteric calculus may be obtained by the cystoscope, when the ureteric orifice of the obstructed side may be seen to be congested or ecchymosed; or a ureteric bougie impermeable to the Röntgen rays may be passed into the ureter and a skiagram obtained, though it is only exceptionally that this can be carried out. Operation upon the side of the recent pain may be urged strongly, when the kidney can be opened and drained, and opportunity taken to explore as much of the ureter as can be felt by the parietal incision and by catheterization from above.

Anuria from Vesical Carcinoma implies that either both ureteric orifices are involved in the disease, or that the ureteric orifice of the only functional kidney is implicated. The condition is uncommon as a pure obstructive anuria, for in most cases the kidneys are already the seat of changes due in part to the back pressure and in part to sepsis, so that when anuria terminates a case of vesical carcinoma, it is more often due to renal disease than to ureteric obstruction. If the bladder has remained uninfected by septic organisms, the gradually increasing ureteric obstruction may first cause hydronephrosis, so that when the obstruction becomes complete the renal distention may increase quickly, and the symptoms of uræmia be delayed. In cases arising from vesical carcinoma, it is very rare for the anuria to occur before symptoms of vesical growth are apparent, such as hæmaturia, pyuria, increased frequency and pain on micturition; but in the infiltrating type of carcinoma, hæmaturia and frequency of micturition may be absent for a long time. In all cases, careful vaginal or rectal examination will detect infiltration and thickening of the base of the bladder, and the growth can be seen through the cystoscope (*Plate XVI*, *Fig. F*, p. 284).

Uterine Carcinoma.—Anuria is frequent in the terminal stage of uterine carcinoma, when the growth has extended into the cellular tissues of the broad ligament and involved the terminal portions of the ureters, or when the orifices of the latter are implicated in the direct infiltration of the growth into the bladder base. In the majority of cases dying from uterine cancer in the inoperable wards of the London Cancer Hospital, the kidneys are found to be hydronephrotic, the renal pelvis dilated, or the renal secreting tissue sclerosed, apart from the frequent infection with septic micro-organisms. In all cases the growth has reached an advanced stage, and the disease has been apparent, but it has been recorded that anuria has occurred before the patient has complained of any symptom pointing to the uterine condition. These cases might simulate other forms of obstructive anuria, but the diagnosis would be apparent upon vaginal examination.

Pelvic or **Abdominal Tumours,** such as uterine fibromyomata or ovarian carcinomata, may cause anuria from direct pressure on the ureters, especially if a part of the tumour is impacted in the pelvic cavity. The cause of the anuria will be apparent on examination of the abdomen and of the pelvic organs.

B.—NON-OBSTRUCTIVE ANURIA.

Marked diminution in the amount of urine or complete anuria may occur without obstructive lesion of the urinary apparatus, due in many instances to disease of the renal secreting tissues. In many of these cases the symptoms differ remarkably from those seen in obstructive anuria, in that the anuria is accompanied by symptoms of uræmia in a short time, and not after an interval of days as in the obstructive cases. Anuria may occur under certain toxic conditions, as in acute fevers, or in acute poisoning by mercury, lead, phosphorus, or turpentine; the history and accompanying symptoms of such cases are usually sufficient to point to the nature of the urinary suppression.

Anuria in Renal Disease.—In *acute nephritis*, anuria may occur early or after the disease is well established, and is usually accompanied by marked disturbance of the nervous system. The sudden onset of the disease after exposure to cold, or in the course of an acute specific fever such as scarlet fever, enteric, or pneumonia, or in hæmatogenous renal infections, associated with pallor, backache, puffiness of the face and ankles, and slight pyrexia, together with the small amount of urine passed before the suppression becomes complete, are points all suggesting acute nephritis. If the urine has been tested before the onset of anuria, it is often of reddish-brown colour from the presence of blood, and contains abundant albumin, together with renal, epithelial, and blood casts. In *chronic nephritis*, anuria may occur as a late symptom in the disease, and is occasionally preceded by a period in which polyuria is marked. Anuria in chronic nephritis is accompanied by prominent symptoms of uræmia, such as headache, giddiness, convulsions, stertor, and coma, and unless the flow of urine is re-established quickly, death ensues. The previous history of the case, high arterial tension, cardiac hypertrophy, retinal changes, and signs of back-pressure, with or without ascites and anasarca, will point to the nature of the anuria. In other diseases of the kidney, such as *lardaceous disease*, *suppurative pyelonephritis*, or *bilateral tuberculosis*, anuria may be preceded by general failing health, with loss of appetite, subnormal temperature, a dry brown tongue, headache, increasing pulse-rate, hiccough, and attacks of dyspnœa ; frequently there may be polyuria before suppression occurs. In these cases the anuria is terminal, the condition of the kidneys having been known previously. With the occurrence of anuria there may be great restlessness, with muscular twitching, loss of sphincteric control, convulsions, and a gradual lapse into coma.

Polycystic disease of the kidneys frequently terminates in anuria and uræmia, but the diagnosis of the disease has probably been arrived at previously. The symptoms resemble in a great measure those of chronic nephritis, with the exception that ascites and œdema of the extremities are uncommon. Headache, flatulence, and digestive troubles, sickness, and general lassitude are symptoms of renal inefficiency, whilst arteriosclerosis, a bilateral renal tumour, and a low-specific-gravity urine in increased quantity, would suggest polycystic disease. Hæmaturia is the first symptom in not a few of these cases.

Anuria following Operations or Trauma.—Anuria may occur in patients who have undergone an operation and who are the subjects of renal disease, or may occur occasionally even when no renal disease is present. Any extensive operation which involves a good deal of shock in a patient with renal disease, or in whom the kidneys have been subjected to back-pressure, as in uterine myomata, may succumb to anuria unless appropriate measures are undertaken ; even an apparently trivial operation on the urinary organs may cause acute suppression of urine. This must be differentiated carefully from the retention of urine in the bladder often seen after operations such as for hæmorrhoids or for hernia. Acute suppression of urine may follow operations upon the lower urinary tracts, such as the passage of instruments, or the performance of internal urethrotomy. Anuria is particularly liable to occur when a catheter is passed to relieve an over-distended bladder in a case of prostatic enlargement or urethral stricture, the kidneys being already distended from back-pressure or infected with septic processes, and it must be laid down as a golden rule, that if a catheter is passed in these cases, the urine must be withdrawn very gradually. Anuria following operations upon the lower urinary tract is diagnosed by the direct relationship between the operation and the onset of symptoms ; by the rigors, pyrexia, and the profound prostration, rapidly followed by convulsive movements and coma.

Anuria may also occur in the severe *collapse* following an injury, in the late stages of *cholera* or *yellow fever*, and occasionally as a manifestation of *hysteria*. It may be simulated by a *malingerer*.

R. H. Jocelyn Swan.

APHASIA.—(See Speech, Abnormalities of, p. 624.)

APHONIA.—(See Speech, Abnormalities of, p. 628.)

APPETITE, ABNORMAL.—Appetite may be : (1) *Increased* ; (2) *Diminished* ; (3) *Perverted*.

Increase of Appetite sometimes occurs in cases of *hyperchlorhydria*. The general condition is then well maintained, there is usually pain or discomfort in the later period of digestion, relieved (temporarily) by the taking of more food. A test meal shows excess of hydrochloric acid.

In *diabetes*, especially in its earlier stages, there is often an abnormal craving for food; but in spite of large meals the patient wastes. Examination of the urine will establish the diagnosis.

Intestinal parasites (round-worms and tape-worms) are believed to be a cause of excessive appetite in some cases. This is doubtful; but in any case the point can always be cleared up by giving an anthelmintic.

In some cases of *hysteria* an excessive appetite is present (bulimia). The patient is usually a young woman, and other stigmata of hysteria are present.

Diminution of Appetite occurs in many forms of dyspepsia, especially when associated with a lessened gastric secretion. Thus it is almost constantly present in *gastritis*, except, perhaps, in the acid form. If renal disease, advanced mitral disease, or cirrhosis of the liver be present, secondary gastritis may be diagnosed. If there be a history of the abuse of alcohol or tobacco, or of indiscretions in diet, or if there be a marked defect of the chewing apparatus, there is probably primary gastritis. The tongue will probably be furred, and a test meal shows diminished acidity and probably an excess of mucus, but the examination of the stomach is otherwise negative. (See also INDIGESTION, *p.* 317.)

Loss of appetite is also an early symptom in cases of *gastric carcinoma*, and should lead, especially in elderly subjects, to careful examination for other signs of that disease. There is frequently a special distaste for meat in such cases. (See INDIGESTION, p. 316.) In children a profound anorexia is sometimes an early symptom of *tuberculosis*.

In hysterical young women complete disinclination for food (*anorexia nervosa*) is sometimes met with. The diagnosis is based upon the absence of other causes of the symptom, the presence of other signs of hysteria, and the history of mental or emotional shock. The loss of appetite in such cases may amount to a complete refusal of all food, and the patient may emaciate to a dangerous degree. Obstinate constipation is usually present as well. Allied to these cases is the loss of appetite which occurs in melancholic forms of *insanity*. In such a case delusions may be present.

Perverted Appetite may occur in the course of *pregnancy*, and is of no special significance. It is met with, too, in nervous, anæmic children, in whom it often takes the form of dirt-eating (*pica*). Here, also, it is not a sign of any diagnostic value. Perverted appetite is also a common occurrence in insanity; but other evidence of mental disturbance is always present as well.

Robert Hutchison.

ASCITES, or the accumulation of serous fluid in the peritoneal cavity, is not a disease in itself, for it may be produced by a great variety of conditions. It is easy to determine its precise cause in some cases; in others it may be almost impossible to say during life what is the primary condition. One may discuss (1) *Its physical signs*; (2) *How to distinguish it from other conditions which may simulate it*; (3) *A classified list of its causes*; (4) *The chief points which will help in arriving at a correct differential diagnosis in a particular case.*

1. **PHYSICAL SIGNS.—Inspection.**—The abdomen is distended uniformly, the degree varying with the amount of fluid. If the quantity is large, and its accumulation has been rapid, the abdomen is more or less globular, the umbilical region being the most prominent. The skin is tense and shiny, and there may be lineæ albicantes. If the quantity of fluid is large but its accumulation has been gradual, bulging of the flanks is more marked; the lower ribs may be pushed outwards, and the epigastric angle widened. If the quantity of fluid is small, only a slight bulging of the flanks may be noticed. The appearance of the abdomen depends a good deal on the position of the patient. If lying on one side, the most dependent part is the most prominent, owing to the fluid gravitating to that side of the abdomen. If the patient stands or sits upright, the hypogastric and iliac regions will be most bulged. The umbilicus becomes stretched transversely and flush with the surface, or even protruded; it retains its position in the median abdominal line, and remains nearer to the pubes than to the ensiform cartilage. In tuberculous peritonitis the skin in its immediate neighbourhood may be reddened and œdematous, or there may be a fæcal fistula here. In cirrhosis of the liver the veins around the umbilicus are said to be dilated, but the so-called ‘caput medusæ’ is rare. The superficial veins all over the abdomen and

lower part of the chest may be dilated, the blood flowing in an upward direction, this reversal of the stream occurring mainly when the inferior vena cava is obstructed, either by the tension of the ascites or by something related to its cause. (See VEINS, VARICOSE ABDOMINAL, p. 748.) The abdominal respiratory movements may be absent or much diminished. The cardiac impulse may be displaced upwards and outwards. The legs, thighs, and scrotum may be œdematous, and so may the loins.

Palpation.—The abdomen may be anything between quite flaccid and very tense. A fluid thrill may be obtained by placing the hand flat against one flank and gently flicking the other with the fingers of the other hand ; the possibility of a thrill being transmitted in the abdominal wall should be eliminated by getting the patient or an assistant to place the side of his hand on the front of the abdomen, so as to stop the mural thrill at the point of contact with the abdominal wall. If the above precaution is taken and a thrill is obtainable, it denotes the presence of fluid.

If the liver or spleen has enlarged it sinks backwards, so that between these organs and the abdominal wall a layer of fluid is present ; if the hand placed on the abdomen, in the right or left hypochondriac region as the case may be, is suddenly depressed, this fluid is displaced, and the surface of the enlarged organ can then be felt. This phenomenon of 'dipping' is almost pathognomonic of ascites.

Percussion.—When the patient lies flat on his back the fluid gravitates to the posterior part of the abdomen, and the air-containing viscera float to the anterior part, so that the percussion note is resonant in front and dull in the flanks. As the fluid increases in quantity, the line of dullness creeps forward from the flanks and upwards from the pubes, and keeps a concave upper border ; in extreme cases the abdomen may be dull all over, particularly in children.

One of the most prominent physical signs of ascites is the effect produced on the percussion note by a change in the posture of the patient. If, after examining him lying on the back and finding dullness in the flanks and resonance in the front, he be turned on one side, the uppermost flank becomes resonant and the line of dullness on the other side rises nearer to the median abdominal line, owing to the fluid gravitating to the most dependent part. If only a very small quantity of fluid is present, the abdomen may be resonant all over when the patient lies on his back ; but if he is percussed in the knee-elbow position, the umbilical region may be found to be dull.

In some cases, especially of tuberculous peritonitis, shortening of the mesentery is apt to be associated with the ascites ; the intestines cannot then rise, and the result is dullness all over the abdomen, or in very exceptional cases dullness in front with resonance in the flanks. Chronic peritonitis may cause the fluid to be loculated, through matting together of the intestines. The abdominal distention may then not be uniform, and change of posture may not alter the character of the percussion note.

Mensuration.—The abdomen should be measured, fixed points being taken in front and behind, e.g., the umbilicus in front and the tip of the third lumbar spine behind. This is important in order to watch the effect of treatment. The distance of the umbilicus from the ensiform cartilage, pubes, and anterior superior iliac spines should also be noted. In ascites, the navel is nearly always nearer the pubes than the ensiform cartilage, and equidistant from the two anterior superior iliac spines when the patient lies flat on his back.

It is always important to examine the abdomen carefully after paracentesis ; the cause of the ascites can often be discovered in this way, in the shape of tumours, or enlargements of organs, which were previously obscured by the tenseness of the abdominal wall.

2. **DIAGNOSIS.**—Ascites has to be distinguished from other conditions which may give rise to general abdominal distention, especially from :—*Tympanites* ; *Ovarian and parovarian cysts* ; *Gravid uterus with hydrops amnii* ; *Distended bladder* ; *Distention associated with obesity* ; *Phantom tumour* ; *Large abdominal cysts and solid tumours.*

Tympanites is distinguished from ascites by the following signs :—The outline of distended coils of intestine may be visible, and peristaltic movements may be noticed. There is no fluid thrill if precautions are taken to prevent a thrill being transmitted by the abdominal wall. The abdomen is resonant all over, both in front and in the flanks.

Ovarian Cyst.—There may be a history of the enlargement of the abdomen having been noticed at an early date to be more on one side than the other, and to have arisen from the pelvis. The umbilicus may be nearer to the ensiform cartilage than the pubes, and nearer to one anterior superior iliac spine than the other. A fluid thrill may not be obtained far back in the flanks, but only in front of the mid-axillary lines. There is usually dullness in front, with resonance in the flanks. The outline of the cyst may possibly be noticed during the respiratory movements. On measuring the abdomen the greatest circumference is usually below the umbilicus; whereas in ascites it is generally at the umbilicus. A vaginal examination may reveal that the uterus is drawn upwards and that its mobility is impaired; whereas in ascites it is low down and movable. If paracentesis has been performed, the nature of the ovarian fluid is characteristic, being usually thick, tenacious, viscid, and of a brownish or greenish colour; whereas ascitic fluid is yellowish, limpid, and clear. Much difficulty arises when ovarian cyst and ascites are both present, owing to infection of the peritoneum by secondary deposits from the ovary. Even without this, however, it is by no means always easy to distinguish between ovarian cyst and ascites when the abdominal distention has become extreme.

Gravid Uterus with Hydrops Amnii.—In this condition it may be possible to make out the outline of the enlarged uterus; the tumour may vary in consistency as the uterine wall contracts and relaxes; on vaginal examination the cervix is soft and patulous and the uterus enlarged. There will be other signs of pregnancy, the characteristic condition of the breasts, fœtal movements and heart sounds, and the history of amenorrhœa. There will be dullness in the front of the abdomen, resonance in the flanks.

Distended Bladder.—This may reach well above the umbilicus, most frequently in women as the result of a retroverted gravid uterus, or in men over sixty from enlargement of the prostate. The most important symptoms are: incontinence of urine from over-distention and overflow, and abdominal distention. There is generally a globular mass to be palpated in the middle line above the pubes and reaching up to the umbilicus or higher; it is dull to percussion in front, with resonance in the flanks. The passage of a catheter should clear up all doubt.

General Obesity may cause much abdominal distention. The mesentery, omentum, and abdominal wall may be so loaded with fat that it is difficult to make a satisfactory examination, and it may be almost impossible to determine with certainty the presence of even a moderate amount of fluid.

Phantom Tumour.—The abdomen may occasionally be so distended in women, especially at the time of the climacteric, that ascites, ovarian tumour, or pregnancy may be simulated when there is merely a phantom tumour. If an anæsthetic is administered it often disappears, the rigid abdominal wall becomes flaccid, and it can be determined whether fluid in the peritoneal cavity or any abdominal tumour is present or not.

Large Abdominal Cysts may occasionally simulate ascites, e.g., hydronephrosis, pancreatic cyst, and hydatid cyst; they do not, however, cause uniform distention of the abdomen as a rule. They are most likely to be mistaken for simple chronic peritonitis in which local collections of fluid have arisen from matting together of the intestines. Hydronephrosis may be distinguished by its position and by the fact that it may vary in size, a decrease being associated with an increase in the amount of urine passed. Pancreatic cyst may be differentiated by its position in the upper part of the abdomen and by its more or less circular outline. If paracentesis abdominis has been performed, the character of the fluid and its ferments would point to the nature of the disease.

3. **CAUSES.**—Having made up one's mind that the general abdominal distention is due to fluid in the peritoneal cavity, one must next differentiate the cause of the ascites. The following is a classified list:—

i. **Diseases of the Peritoneum**:—
  Non-suppurative acute peritonitis
  'Simple' chronic peritonitis
  Tuberculous peritonitis
  Malignant peritonitis, generally secondary to a primary growth elsewhere
  Hydatid cysts in the peritoneal cavity.

ii. **Obstruction to the main Portal Vein** by:—
  Non-suppurative thrombosis
  Enlarged portal lymphatic glands:—
    Malignant
    Lymphadenomatous
    Tuberculous
    Lymphatic leukæmic

Tumours of adjacent organs, such as :—
- Liver
- Pancreas
- Kidney
- Stomach
- Duodenum
- Colon
- Suprarenal capsule

Rarities such as aneurysm of the hepatic artery

iii. **Diseases of the Liver** :—
- Cirrhosis
- Perihepatitis, really part of chronic simple peritonitis
- Carcinoma
- Sarcoma
- Syphilis
- Hydatid disease

Doubtful causes if the lesions are confined to the liver ; i.e., if there is ascites, it is probably not due to the carcinoma, etc., in the liver, but to simultaneous affection either of the peritoneum or of the portal lymphatic glands

iv. **Obstruction of the Inferior Vena Cava above the Hepatic Veins** by :—
- Thrombosis
- Chronic mediastinitis
- Mediastinal growth

v. **Chronic Failure of the right Heart** (‘**backward pressure**’) the result of :—
- Valvular disease :—
  - Mitral stenosis
  - Mitral regurgitation
  - Aortic stenosis or regurgitation with secondary mitral regurgitation :—
    - Rheumatic or syphilitic
  - Congenital pulmonary stenosis (rarely)
- Chronic myocardial affections :—
  - Fatty degeneration
  - Fatty infiltration
  - Fatty superposition
  - Fibroid heart
  - Primary alcoholic heart
- Adherent pericardium
- Chronic lung affections, especially :
  - Emphysema
  - Recurrent bronchitis

    Generally associated
  - Fibroid lung
- Chronic high blood pressure :—
  - Red granular contracted kidneys
  - Pale granular contracted kidneys
  - Arteriosclerosis

vi. **Bright's Disease.** In Bright's disease ascites may be caused in at least four different ways,—namely, as the result of :—
- Part of a general dropsy
- Acute peritonitis
- Chronic peritonitis
- Secondary to hypertrophy and dilatation of the heart, followed by failure of compensation

vii. **Severe Anæmias,** in which the ascites is usually the result of acute, subacute, or chronic intercurrent peritonitis, as in :—
- Splenomedullary leukæmia
- Lymphatic leukæmia
- Hodgkin's disease
- Pseudo-leukæmia infantum
- Splenic anæmia
- Pernicious anæmia
- Aplastic anæmia
- Malaria

4. **DIFFERENTIAL DIAGNOSIS.**—If ascites is the only fluid accumulation present in the patient; if, although there is also swelling and œdema of the legs, the ascites is known to have appeared first ; or if the ascites is out of proportion to dropsy elsewhere : it is most probably due either to some form of peritonitis, to portal obstruction from thrombosis of or pressure on the portal vein, or to cirrhosis of the liver.

If it is associated with general anasarca, that is to say, with œdema of the legs, body, and face, perhaps even of the scalp, and possibly with other serous effusions, the probable cause is acute, or acute on chronic, Bright's disease.

If swelling and œdema of the legs were noticed first and the ascites followed, heart failure from one of the causes in *Group* V, or obstruction of the inferior vena cava above the hepatic veins, would be the most likely cause ; it is important to remember, however, that in the slighter cases, or in those of long standing, the patient is often uncertain which swelled first, his legs or his abdomen, and his statements on the point may be misleading.

If jaundice is associated with the ascites, it points to some form of portal obstruction as the cause, either cirrhosis of the liver, or, if the jaundice is intense, to some actual pressure on the portal vein and common bile-ducts, generally due to malignant disease.

If enlargement of the liver is associated with the ascites this may be due to carcinoma, sarcoma, cirrhosis, perihepatitis, syphilis of the liver, or to nutmeg change the result of backward pressure from chronic heart or lung disease.

If the ascites is associated with multiple abdominal tumours it suggests tuberculous or malignant peritonitis, or in rarer cases hydatid disease.

i. Diseases of the Peritoneum.

Acute Non-suppurative Peritonitis is an acute inflammation of the peritoneum analogous to acute 'simple' pleurisy with serous effusion. One seldom speaks of ascites, however, in connection with acute infective peritonitis such as would lead to pus formation if laparotomy were not resorted to; and it is difficult to draw a decided line between acute peritonitis in which the fluid should be called ascites, and other conditions of acute generalized peritonitis to which the term would not be applied. There are, however, cases in which acute serous effusion due to non-suppurative peritonitis occurs in acute and chronic Bright's disease; or acute tuberculous peritonitis almost simulating general suppurative peritonitis; whilst pneumococcal and gonococcal peritonitis may be acute in onset, and yet take the form of an ascitic effusion, recovery occurring without the necessity for laparotomy. It is probably a question of the dose of the micro-organism that affects the peritoneum, and it is by no means impossible that, whereas the perforation of a gastric ulcer, duodenal ulcer, dysenteric, typhoid, or tuberculous ulcer of the intestines, or leakage from a pyosalpinx, an appendicular abscess, stercoral ulcer of the colon, or a perirectal or prostatic abscess, generally gives rise to acute general peritonitis which would prove suppurative if it were not operated on, the same conditions may in some cases lead to a slighter affection with a severe but non-suppurative ascitic effusion ending in spontaneous recovery. Whether laparotomy is indicated or not in any given instance must depend upon the individual circumstances of the case; but it is much safer for the patient to be operated upon for acute non-suppurative peritonitis of the type of which we are now speaking than for general suppurative peritonitis to escape operation.

Simple Chronic Peritonitis is a chronic inflammation that is not tuberculous or malignant. It may follow simple acute peritonitis, but its two commonest causes are: a former tuberculous peritonitis from which the tubercles have disappeared; and the chronic inflammation which results from repeated paracentesis abdominis for any other variety of ascites. The latter is important; it sometimes happens, in a heart case for instance, that both œdema of the legs and ascites have been prominent symptoms, paracentesis abdominis being indicated on account of the cardiac distress; the tapping of the abdomen may have had to be repeated several times, and yet ultimately the cardiac compensation has been restored, the patient's general condition becoming quite good and the œdema of the legs disappearing; yet in spite of this general improvement, ascites may still persist and require further tapping at intervals. In such a case, whereas at first the ascites was due to backward pressure from the failing heart, it ultimately becomes due to chronic peritonitis, the result of the repeated tappings. It is usually associated with perihepatitis, which indeed is only one of the local manifestations of chronic peritonitis. Even when all inflammation has ceased, the great thickening of the peritoneum over the diaphragm, liver, and spleen may have blocked up those pores through which the peritoneal secretions naturally drain away, so that the fluid keeps on re-accumulating, and necessitates repeated tapping, which in some cases has been performed over three hundred times. The peritoneum becomes thickened generally, and the intestines bound down and matted together. There may be local or general abdominal distention, depending on whether loculi are formed or not by the adhesions. On account of the shortening of the mesentery and matting together of the intestines there may be dullness all over the abdomen, so that this form of ascites is particularly liable to be mistaken for ovarian cyst or tumour. Albuminuria is frequent on account of interference with the renal circulation, and there may even be a few tube casts; there may or may not be actual renal disease, but this should not be diagnosed from the albuminuria unless there is also high blood-pressure, retinitis, or other confirmatory sign. Abdominal pain is generally slight, and although there may be vomiting or constipation, there is usually neither.

Tuberculous Peritonitis.—This is the most common cause of ascites in children. There are several varieties, of which the following may be distinguished:—

1. The acute ascitic form, which may simulate acute general peritonitis due to perforation of a viscus (see above).
2. The peritoneum may be studded all over with miliary tubercles without any caseous

masses. The physical signs are those of ascites without any abdominal tumour, and it is not difficult to mistake it when it occurs in an adult for cirrhosis of the liver or for malignant peritonitis, especially that form which is secondary to ovarian tumour. In a child, the occurrence of ascites without œdema of the legs at once suggests tuberculous peritonitis ; in an older person tuberculous peritonitis is much less common.

3. The omentum may be contracted and thickened from infiltration with caseous or fibro-caseous material, and a hard abdominal tumour simulating an enlarged liver may be felt. It may be distinguished, however, by the resonant percussion note between it and the costal margin, and the liver edge may be palpable above and distinct from the omental mass which simulates it. Ascites in cases of this kind is generally less in amount than in the miliary tuberculous form.

4. The intestines may be matted together and the adhesions thickened and infiltrated with tuberculous deposits, so that the peritoneal cavity may be divided into several loculi of fluid, the abdominal distention being not uniform, and paracentesis only removing part of the ascites.

5. The mesentery may be thickened and contracted, and the intestines bound down to the posterior parts of the abdominal cavity, so that if there is ascites there will either be dullness all over the abdomen, or dullness in front with resonance in the flanks, suggesting ovarian cyst rather than tuberculous peritonitis. After paracentesis, a more or less defined irregular deeply situated tumour may often be felt.

6. When the caseation affects the mesenteric glands in particular, multiple irregular tumours are felt, sometimes but not always associated with ascites.

7. Occasionally local thickenings in the abdominal wall are to be felt as the result of subperitoneal inflammatory deposits, a condition which may often be mistaken for rigid contraction of the recti muscles or for disease of the parietes rather than of the peritoneum ; if, however, there is ascites at the same time, tuberculous peritonitis would be very probable, particularly in a child.

It will naturally depend upon the acuteness of the tuberculous process whether there will be pyrexia or not, and whether there will be abdominal pain and tenderness. In the caseous varieties, whether of the glands, omentum, mesentery, or abdominal wall, pain and tenderness are the rule, and the temperature generally rises to 101° F. to 104° F. each evening. It is not at all uncommon in such cases for redness and œdema to develop round the umbilicus, and for a purulent discharge to occur from the latter, or for a fæcal fistula to develop. The commonest cause for spontaneous fæcal fistula of the umbilicus is tuberculous peritonitis. When the active tuberculous process has become quiescent there may still be ascites, though the temperature is subnormal. When paracentesis is performed, it is advisable to inject some of the fluid into a guinea-pig, to see if the latter develops general tuberculosis. The nature of the case may sometimes be suggested by the presence of tuberculous lesions elsewhere in the patient's body ; for instance, in the spine, kidney, a joint such as the hip or knee, glands in the neck, or lupus, though very often tuberculous peritonitis is the only objective lesion.

Ascitic Fluids.—It has been stated that chemical analyses of ascitic fluid often afford material assistance in arriving at a diagnosis of its cause ; but in practice only the broadest conclusions can be drawn. The higher the specific gravity, the larger the percentage of albumin, and the greater the tendency to spontaneous coagulation, the more definitely can one conclude that the condition is an inflammatory exudate—e.g., specific gravity 1,025, twenty parts per thousand of albumin, with a spontaneous coagulation. The lower the specific gravity, the smaller the percentage of albumin, and the more definite the absence of spontaneous coagulation, the more likely is the condition to be a non-inflammatory transudate—e.g., specific gravity 1,005, five parts per thousand of albumin, and no coagulation. There are, however, many intermediate cases in which chemical investigation of the fluid leaves one in doubt as to whether the condition is inflammatory or not.

It has also been stated that differential analyses of the proteids are helpful, notably as to whether there is more globulin or more albumin present ; but it is doubtful whether this really is so.

Microscopical examinations are more valuable than chemical ; the centrifugalized deposit should be examined under the high power ; it may exhibit many leucocytes in inflammatory conditions, polymorphonuclear cells predominating in acute conditions, small lymphocytes in subacute or chronic affections such as tuberculous peritonitis ; peritoneal cells in cases of inflammation ; and occasionally the diagnosis is clinched by finding actual fragments of new growth or hydatid hooklets (*Fig.* 18). The deposits may also be stained for bacteria, and sometimes tubercle bacilli, streptococci, staphylococci, gonococci, or pneumococci may be found. When investigating ascitic fluid bacteriologically, however, it is probably better to resort to cultural or inoculation methods than to rely solely upon films prepared from the deposit.

Cancerous Peritonitis usually occurs in patients over forty, and the growth is practically always secondary. Primary carcinoma of the peritoneum is very rare, and it is usually colloid and not associated with ascites. In secondary cases the omentum may be thickened and infiltrated, the umbilicus fixed, the urachus palpably infiltrated, and nodules and masses may develop all over the peritoneum. Rapid emaciation and cachexia are the rule. A large quantity of fluid may be present, and if it is blood-stained at the first tapping this is very suggestive of malignant disease. Ascites may be the first and only evidence of growth, and it may be mistaken for that of tuberculous peritonitis or cirrhosis of the liver, especially when the abdominal distention is so marked that no nodules can be felt. Evidence of a primary growth should always be looked for with care, especially in connection with the stomach, pancreas, colon, rectum, or ovaries. Rectal examination should never be omitted, and if need be the sigmoidoscope may be used. It should not be forgotten that useful indication of intra-abdominal malignant disease is sometimes afforded by enlargement of the left supraclavicular lymphatic glands by secondary deposits (*Fig.* 17).

There is one variety of secondary malignant peritonitis which merits special mention—namely, that which may result from a proliferating papillomatous ovarian cyst. The malignancy of the latter is sometimes relative, so that although there may be thousands of papilloma deposits on the peritoneum, causing ascites that may need tapping scores of times at short intervals, there may be no other secondary deposits anywhere. The diagnosis may be made as the result of careful vaginal examination, or by finding fragments of the malignant papillomata in the ascitic fluid, or perhaps the case may be regarded as chronic 'simple' peritonitis until the abdomen is opened.

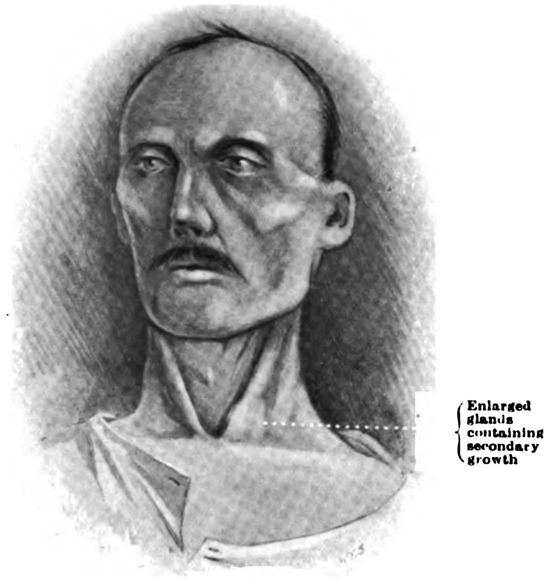


Fig. 17.—Enlargement of the left supraclavicular glands in a case of abdominal malignant disease (carcinoma of the sigmoid colon).

Hydatid Cysts in the peritoneal cavity may be primary, but more often they are secondary to hydatid disease of the liver. The malady is rare in this country, though commoner in Australasia and elsewhere. The patient is generally an adult and the diagnosis is often obvious, though sometimes it may be very obscure. There may be a large globular tumour in the liver, rarely giving the typical hydatid thrill; there may be EOSINOPHILIA (p. 219), and an investigation of the blood serum in special laboratories may show the specific hydatid serum reaction. In some cases in which there are hydatid cysts associated with ascites it is possible to make the diagnosis by rectal examination; one has felt globular bodies about the size of grapes in front of the anterior rectal wall, and when these have been pressed upon to investigate them more fully, they have slipped away from under one's finger through being pushed up into the ascitic fluid; after waiting a moment the finger has felt them come back into Douglas's pouch. Similar mobility of spherical masses in the ascitic fluid may be noted elsewhere—for instance, in an iliac fossa. The ultimate diagnosis depends upon the detection of hooklets (*Fig.* 18) in the fluid obtained by paracentesis or by laparotomy. It is important to bear in mind, however, that the absence of hooklets does not exclude hydatid disease, the cysts sometimes being sterile, and in that case not producing hooklets.

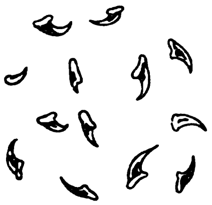


Fig. 18.—Echinococcal hooklets.

Chylous Ascites is not in itself a specific malady, for there is more than one condition in which the ascitic fluid may appear like milk. This may result from obstruction to the main abdominal lymphatics, particularly the receptaculum chyli and thoracic duct ; or from their rupture after injury to the abdomen ; more often the condition is associated in this country, in some way which is not fully understood, with the peritonitis of chronic *Bright's Disease*, or of *leukæmia*. The best known tropical cause for chylous ascites is *Filaria sanguinis hominis* with elephantiasis. In rare cases the secondary deposits of malignant disease may be such as to obstruct the thoracic duct, and so produce the chylous condition of the ascitic fluid. Chyluria may or may not occur at the same time.

There are two types of chylous ascites, one in which actual chyle accumulates in the peritoneal cavity as the result of direct leakage from the thoracic duct or receptaculum—true chylous ascites ; the other in which the condition is in the main one of ascites, but the fluid becomes milky-looking from little-understood chemical changes taking place in it, particularly in the proteids. This is termed chyliform ascites, or pseudochylous ascites. There is much more real fat in the former condition than in the latter ; but chyliform ascites is commoner than true chylous ascites. The diagnosis between the two is afforded by chemical and microscopical analyses of the fluid obtained by tapping, the chief points of distinction being as follows :—

Chylous Ascites.	*Chyliform Ascites.*
1. The fluid tends to accumulate very rapidly, and in consequence large volumes are removed at paracentesis.	1. Collects more slowly, the volume of the fluid varying with the exciting pathological condition.
2. Generally yellowish-white in colour and less perfectly emulsified.	2. In colour a pure milky-white solution in the form of an almost perfect emulsion.
3. Degree of opalescence more or less constant at successive tappings.	3. The opalescence generally increases or diminishes at successive tappings.
4. Possesses an odour corresponding to the odour of the food ingested.	4. Odourless.
5. Microscopically the fluid contains fine fat globules, but very few cellular elements.	5. Microscopically the quantity of free fat is variable ; often numerous fine, highly refractile granules are present, and these do not give the reactions for fat. Cellular elements may be numerous and often contain fat ; sometimes very scanty.
6. Generally shows a distinct creamy layer on standing.	6. A cream may or may not form, but does not affect the opalescence ; a sediment frequently settles out.
7. Specific gravity generally exceeds 1012.	7. Specific gravity less than 1012.
8. Depression of freezing point about 0·51° C. and approximating that for chyle.	8. Depression of freezing point ranges from 0·56° to 0·61°, and thus corresponds to the figures for blood serum.
9. Total solids vary considerably, but usually exceed 4 per cent.	9. Total solids rarely exceed 2 per cent.
10. The total protein content generally exceeds 3 grams per cent, and of this the serum-albumin is the largest fraction, globulin occurring only in traces.	10. The protein constituents vary between 1 and 3 grams per cent, and of these the serum-globulin occurs in appreciable quantities.
11. Mucinoid substances absent.	11. Mucinoid substances present.
12. The fat content is generally high, varying from 0·4 to 4 per cent. The fat corresponds in all its properties to the fat contained in food.	12. The fat content is generally low, and it may be present in traces only ; in its melting and chemical composition it proves to be pathological fat.
13. Of the lipins cholesterol is invariably found, and lecithin only occurs in traces.	13. The most characteristic lipin is lecithin, though cholesterol is occasionally present.
14. No evidence of the presence of a lipin-globulin combination.	14. The lecithin is mainly combined with the globulin, and when present is the cause of the opalescence of the fluid. Such fluids resist putrefaction.
15. The salts and the organic substances present approximate to the values found for chyle obtained from the thoracic duct.	15. The salts and organic materials correspond closely to those of lymph and serous fluids.

ii. **Obstruction to the Main Portal Vein.**—This is most commonly due to *enlargement of the portal lymphatic glands* by secondary deposits of malignant disease ; it is common for the main bile-ducts to be obstructed at the same time, so that an increasing depth of jaundice accompanies the ascites. When there are masses of secondary growth in the liver associated with jaundice, or ascites, or both, it is seldom that the hepatic masses are themselves respon-

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sible for the symptoms, these being more often due to the associated deposits in the portal lymphatic glands. The diagnosis is made on discovering a primary growth, more often a carcinoma than a sarcoma. It is much rarer for the lymphatic glandular enlargement to be lymphadenomatous, tuberculous, or due to lymphatic leukæmia. If ascites were a prominent symptom in any of these conditions, it would be regarded as consequent on affection of the peritoneum rather than on obstruction to the portal vein, unless there were deepening jaundice at the same time. In the latter case malignant disease would be simulated. General enlargement of the lymphatic glands in the axillæ, groins, and neck, with or without evidence of enlargement of those in the thorax or abdomen, together with enlargement of the spleen, would suggest either lymphadenoma or lymphatic leukæmia; the absence of positive blood changes would render the former more likely, for in lymphatic leukæmia there is more or less considerable leucocytosis with a great relative increase in the small lymphocytes up to 90 per cent or more (p. 25). Only in very rare cases do tuberculous portal glands cause ascites, and when they do the diagnosis must be one of guess-work only, unless in association with definite tuberculous peritonitis there were jaundice suggesting obstruction to the common bile-duct and to the portal vein at the same time.

Thrombosis of the portal vein may be suppurative, in which case there is no ascites, but a pyrexial condition with rigors and possibly jaundice, diagnosed as a rule only when there has been some definite inflammatory focus in the portal area, such as appendicitis, which might lead to infection of the portal vein. Primary thrombosis of the portal vein is rare, and its diagnosis can seldom be more than guessed at. It leads to marked ascites, possibly with simultaneous increase in any tendency there may be to piles, and without evidence of tuberculous or malignant disease of the peritoneum or cirrhosis of the liver. It is by a process of exclusion that the diagnosis of portal vein thrombosis might be arrived at, especially if the ascitic fluid withdrawn by paracentesis, when examined chemically, were found to contain a relatively very high proportion of coagulable proteids without any particular tendency to spontaneous coagulation, and without those polymorphonuclear cells or lymphocytes that would be found if the high percentage of proteid were due to the ascites being inflammatory.

Tumours of adjacent organs seldom obstruct the portal vein enough to cause ascites without presenting other symptoms which suggest the diagnosis. Sometimes, however, unless the tumour can be felt, great difficulty may be experienced in determining the nature of the case. Carcinoma of the pancreas may be accompanied by glycosuria and the passage of fatty stools, together with deepening jaundice, progressive enlargement of the gall-bladder, and a positive CAMMIDGE'S PANCREATIC REACTION (p. 100). On account of the relation of the tumour to the aorta, marked transmitted pulsation may be felt in it, and by inflating the stomach it may be demonstrated that the tumour lies posterior to the latter. Renal tumours may be difficult to distinguish from enlargement of the liver when they are big; but they are generally associated with ALBUMINURIA (p. 4), HÆMATURIA (p. 275), or PYURIA (p. 574). Carcinoma of the stomach, duodenum, colon, or suprarenal capsule would be suggested by the position of the mass, or by the gastric or intestinal symptoms; if there were ascites accompanying them, it would generally be due not to the primary tumour, but to secondary deposits either in the peritoneum or in the portal lymphatic glands.

Aneurysm of the hepatic artery is a pathological curiosity, though in recorded cases it has produced ascites and jaundice. The commonest cause of aneurysm of the hepatic artery is fungating endocarditis with embolism.

iii. **Diseases of the Liver.**—*Cirrhosis of the Liver.*—When ascites is due to this the diagnosis is sometimes easy on account of the history of chronic alcoholism, and possibly of former hæmatemesis, melæna, or jaundice. There may also be acne rosacea and telangiectases on the cheeks, or even a bottle-nose, a furred and tremulous tongue, a history of morning sickness, cramps in the legs at night, nausea, loss of appetite especially for breakfast, epistaxis, perhaps the presence of distended veins round the umbilicus, hæmorrhoids, enlargement of the liver, the surface of which is hard and rough and the edge irregular and perhaps beaded, enlargement of the spleen, icteric tinge of the conjunctivæ, and a peculiarly sallow, slightly pigmented facies, which is almost characteristic in the later stages of the malady. Cirrhosis is a slowly progressive disease sometimes extending over twenty years or more, producing a large, smooth, unilobular cirrhotic liver with jaundice and a tendency to hæmatemesis in its earlier stages; but later a small liver in which, in addition to the unilobular fibrous tissue, there has developed a much coarser multilobular meshwork which, by progressive contraction, has led to the previously large, smooth organ becoming smaller, rougher, and harder, until it may sometimes be so small as to be no longer palpable. Only in the very last stage does it produce ascites. People have been known to be total abstainers for as long as eighteen years or more after the first symptoms of cirrhosis have developed, and yet to die with a granular, contracted, ' hob-nail ' liver and ascites.

Perihepatitis.—A case of cirrhosis of the liver seldom survives long after it has first become necessary to tap the abdomen, and when paracentesis abdominis has to be performed more than once or twice in a case supposed to be cirrhosis, this points to the diagnosis being wrong, the case being one, not of cirrhosis, but of perihepatitis. This is not always so, however, for it happens sometimes that even when the ascites was originally due to cirrhosis, the repeated tapping produces perihepatitis, the greatly thickened capsule of the liver being the result of multiple tappings for what was at first cirrhotic ascites. It is exceedingly difficult to be certain of the diagnosis of simple perihepatitis ; the condition is really only part of a chronic peritonitis. The capsule of the liver becomes much thickened, and it contracts and distorts the organ, and rounds the edge, or else turns it up or under, in a way which is characteristic. It is only if this curled-under or turned-up edge can be detected that the diagnosis of perihepatitis can be made with certainty. Syphilis is possibly the cause of the malady in some cases.

Ascites associated with *carcinoma* or *sarcoma of the liver* is usually accompanied by intense jaundice, and there is always doubt as to whether these symptoms are not due rather to coincident affection of the portal lymphatic glands than to the deposits in the liver itself. The latter becomes much enlarged, very hard, the edge often coming well below the umbilicus. Probably the largest livers that occur are due to secondary carcinoma or sarcoma. They may reach a weight of 22 lb. or more. Besides being very hard, the liver may be tender, and umbilicated nodules may be felt on its surface. Primary growth of the liver is exceedingly rare, and though it leads to progressive and deepening jaundice, it does not often produce ascites. Secondary growth is so much more common, that it is important to look for the primary growth elsewhere with great care before primary growth in the liver is diagnosed. Retinal and rectal examination should not be omitted ; and Cammidge's pancreatic reaction (p. 100) should be tested, in case the primary growth be in the pancreas.

Syphilis may produce local peritonitis over a gumma ; it may also lead to general chronic peritonitis and thus to ascites. The diagnosis is made upon the history, upon the signs of syphilis elsewhere, and upon Wassermann's serum reaction.

Hydatid disease of the liver seldom of itself causes ascites, though it may be associated with coincident affection of the peritoneum with ascites (p. 49).

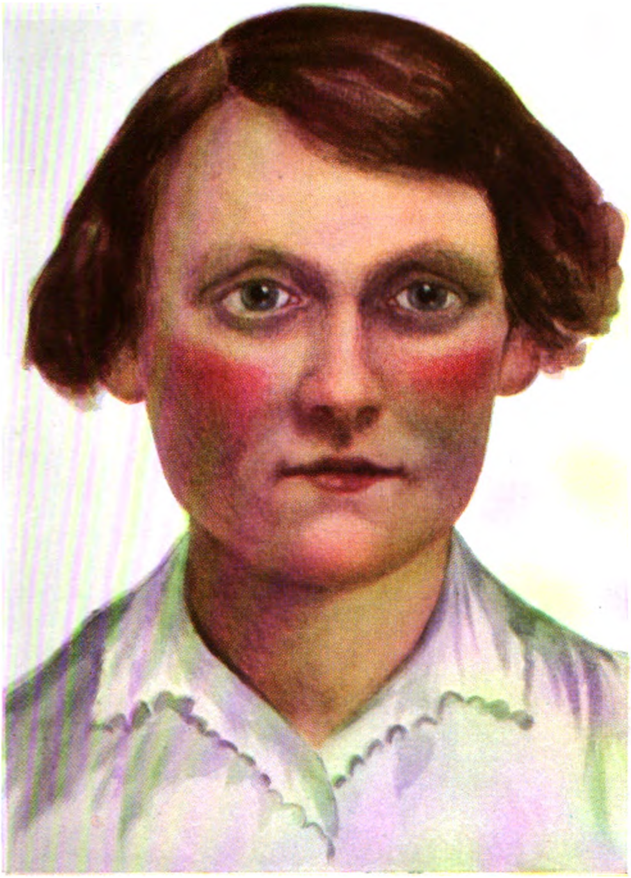
We may now pass on to consider those cases in which, if the history is correct, there has been swelling of the legs before, or at any rate not later than, swelling of the abdomen ; and if one follows the classification as given on pages 45 and 46, one comes next to :—

iv. **Obstruction of the Inferior Vena Cava above the Hepatic Veins.**—This is rare, and will seldom be diagnosed unless there is either (1) clear evidence of extension of *thrombosis* to the inferior vena cava from a previous thrombus in one leg, associated with extension of œdema up the back, followed by albuminuria and perhaps hæmaturia when the renal veins are involved, and then by ascites, together with varicose distention of the abdominal veins and reversal of the blood-stream in them ; or (2) a history or the physical signs of *chronic mediastinitis*, which generally results from recurrent attacks of pleurisy and pericarditis, especially rheumatic, or of *intrathoracic new growth*, which is distinguished from chronic mediastinitis by the shorter history and by the *x*-ray appearances. (*Fig.* 42, p. 105.) (See VEINS, VARICOSE THORACIC, p. 750; and VEINS, VARICOSE ABDOMINAL, p. 748.)

v. **Chronic Failure of the Right Side of the Heart (Backward Pressure).**—Ascites as the result of backward pressure in chronic heart and lung disease is nearly always preceded by swelling and œdema of the legs. Careful examination of the heart and lungs, a history of acute rheumatism, or of recurrent winter cough, or an abundant and offensive periodic expectoration, may suggest valvular disease of the heart, chronic bronchitis and emphysema, or fibroid lung with or without bronchiectasis, to account for the ascites. Nutmeg liver also results in these cases, the enlargement varying with the degree of heart failure, the surface of the organ being smooth, sometimes pulsating synchronously with the heart,

PLATE VIA

THE FACIES IN MITRAL STENOSIS



Showing the typical malar flush and deep-red lips, with yellowish pallor of the forehead, nose, and circum-oral region.

tender, with a well-defined edge which may reach below the level of the umbilicus in the right nipple line. The urine is apt to contain albumin, and when the heart failure has reached an advanced degree it may be exceedingly difficult to say whether it is due to primary valvular disease, primary lung disease, primary kidney disease, primary arterial disease, or to primary affection of the muscle of the heart. The importance of casts in the urine in the differential diagnosis has been referred to under ALBUMINURIA (p. 6), where the significance of the blood-pressure, of retinal changes, and so forth, are also discussed.

The valvular heart lesion most apt to be mis-diagnosed in connection with ascites is *mitral stenosis*; for by the time the heart failure has reached a sufficient degree to cause ascites, characteristic bruits, especially the presystolic, become no longer audible in many cases. The heart beats very rapidly and irregularly, no bruits may be audible at all. Mitral stenosis may still be suggested by the characteristic facies (*Plate* VIA), with its yellowish pallor of the forehead, and around the nose and mouth, with bright or dark red coloration of the lips and over the malar bones and upper portions of the cheeks; or by the history of acute rheumatism or chorea, though absence of such a history by no means excludes valvular heart disease. It may, however, be impossible to say whether there is mitral stenosis or not until the patient has been kept in bed, given digitalis, and watched for a week or more, until there is some degree of recovery of the cardiac compensation; by which time the characteristic bruits of mitral stenosis very often return with the increasing force of the heart's beat.

Some of the hardest of heart-failure cases to diagnose with certainty are those due to *chronic affections of the myocardium* or to *adherent pericardium*. In each case the diagnosis is arrived at mainly by a process of exclusion. Chronic myocardial degeneration seldom occurs in young people, or at any rate it is much commoner in middle life and later. The symptoms are those which are common in all varieties of chronic heart failure (p. 418), whatever the cause of the latter. There may or may not be the systolic bruit of mitral regurgitation, or an aortic systolic bruit due to atheroma of the aortic valves, but upon the whole the physical signs do not suggest valvular disease; the urinary changes and the absence of casts do not suggest nephritis or granular kidney; the blood-pressure may not suggest arteriosclerosis; the lung signs do not suggest bronchitis and emphysema, or fibroid lung: so that some myocardial affection is all that is left to diagnose. If there is a history of the drinking of much alcohol, particularly beer, *primary alcoholic heart* may be suspected, though this is less common in England than in Germany. *Fatty superposition* would be suggested if there were general obesity with shortness of breath on ordinary exertion; whilst overloading of the surface of the heart seldom occurs without some *fatty infiltration* at the same time. *Fatty degeneration* is more likely after a long febrile illness, or chronic poisoning by phosphorus, arsenic, or lead, or by the hypothetical toxins of severe anæmias, such as pernicious or aplastic anæmia. *Fibroid heart* is very difficult to distinguish from fatty heart, but it is the more likely in a syphilitic patient, particularly if the patient is not obese and if there is syphilitic aortic regurgitation or angina pectoris.

Adherent pericardium is not in itself an explicit term, for there are three different conditions which come under the one heading; there may be (1) Adhesions between the parietal and visceral layers of the pericardium; (2) Adhesions between the parietal pericardium and the structures around it, particularly the pleuræ, diaphragm, and chest wall; or (3) Adhesions both of the parietal to the visceral layer of pericardium and of the parietal layer to the structures outside it—really a form of chronic mediastinitis. It is clear that the physical signs will differ according to which of these three things has happened. That which ought to be implied strictly by the term adherent pericardium is adhesion of the parietal to the visceral layer, without any other adhesions whatever, and of this condition there are no positive physical signs at all, nor need there be any symptoms. The diagnosis is generally made by guess-work, the patient being known to have had pericarditis, or being suspected of having had it because of having suffered from acute rheumatism with severe complications, and the heart now being found much larger than it ought to be in proportion to the apparent valvular disease as indicated by the bruits. It is common, however, for the parietal and visceral layers of pericardium to be universally adherent without the heart being big, and without there being any ill effects at all, the condition being met with post mortem in patients who die of something quite different. It is only when the parietal layer has become adherent to the visceral layer when the heart was already dilated at the time of the pericarditis that symptoms subsequently accrue, the result rather of the inability of the already big heart to maintain sufficient hypertrophy than of any intrinsic interference with its action by the adherent pericardium itself. It quite often happens, indeed, that when there has been rheumatic myocardial affection without pericarditis, the big heart that results is out of all proportion to the valvular disease, and yet in the post-mortem room no abnormality of the pericardium is found.

The following points in connection with heart disease in children are as true as most aphorisms: mitral stenosis is almost unknown before puberty, whatever the bruits that suggest it; heart disease never proves fatal before puberty unless as the result either of the severity of the acute inflammation of valves, muscle, or pericardium, or else from adherent pericardium. Fatal mechanical failure of the heart before puberty in a patient who presents no symptoms of rheumatic reinfection points to adherent pericardium.

Adhesions between the parietal pericardium and the structures outside it, without any adhesion between the parietal and visceral layers within the pericardium, are exceedingly common, generally resulting from former pleurisy. The former inflammation must have extended outside both the pericardium and the pleuræ, so that it was really a mediastinitis; but clinically the condition is seldom spoken of as mediastinitis, because it is of very little importance, and in itself produces no symptoms; the physical sign which might suggest it is deficiency in the movement of the position of the cardiac impulse to the left or to the right as the patient rolls from one side to the other.

The third variety of adherent pericardium, namely that in which there are adhesions between the parietal and visceral layers and between the parietal layer and the chest wall, pleuræ, and other structures outside it, is really a combined condition of adherent pericardium and mediastinal adhesions which, when an extreme degree is reached, becomes what is known as chronic mediastinitis. Here again, it is possible for neither symptoms nor physical signs to present themselves, the condition being found unexpectedly in the post-mortem room. It is this condition which is generally diagnosed under the name of adherent pericardium. There will be a history of former pericarditis, pleurisy, or both, probably rheumatic. The heart will be large out of all proportion to any valvular disease that is present, without there being other obvious cause for its hypertrophy and dilatation, such as nephritis, arteriosclerosis, hard work, alcoholism, fatty or fibroid heart, or chronic lung disease. If the parietal pericardium is adherent both to the pleuræ and to the diaphragm—particularly the latter—there will very likely be retraction of the lower left ribs posteriorly, synchronous with the heart-beat; it is this physical sign—systolic retraction of the lower left ribs—which is generally regarded as pathognomonic of adherent pericardium; it is really evidence, of course, of adhesions outside rather than within the pericardium. The sign needs to be looked for with some care; the observer watching the posterior profile of the left chest from the patient's left side, small movements obviously due to cardiac and not respiratory action are to be seen in the ninth or tenth intercostal space in the line of the angle of the scapula, or just outside this; irregularity in the heart's action often rendering these visible only now and then—perhaps only when a strong heart-beat happens to coincide with the most favourable phase of respiration. The sign, however, is far from uncommon. Another physical sign which is regarded by some as indicative of general pericardial adhesions, is an ingoing impulse in the third or fourth intercostal space half-way between the left nipple and the left border of the sternum, synchronous with an outgoing impulse nearer the apex, giving an oscillating or see-saw appearance to the precordial region—some of the intercostal spaces moving inwards at the same time as others move out with the heart-beat. As a matter of fact, the probable explanation of the ingoing movement nearer the sternum when the part of the heart which is nearer the apex causes the ordinary outgoing impulse, is the visible withdrawal of the hypertrophied right ventricle as it contracts. This see-saw appearance in the precordial region is indicative therefore of great hypertrophy of the right ventricle; it does not indicate what is the cause of this hypertrophy, though amongst its causes would be adherent pericardium. A similar appearance is often seen in cases of extreme mitral stenosis of long standing, even when there is no adherent pericardium.

Bright's Disease may produce ascites in more ways than one; the effusion may, for instance, simply be part of a general anasarca, the accumulation of the ascitic fluid in the peritoneal cavity corresponding precisely with its accumulation in the subcutaneous tissues;

or the Bright's disease may lead to acute or chronic peritonitis of the types described above; or, especially in chronic cases associated with pale or red granular contracted kidneys, there may be failure of the dilated and hypertrophied heart, with ascites, which may be very difficult to distinguish from that of primary heart disease; especially as the greater part of the associated albuminuria is now the result of the heart failure rather than of the renal sclerosis; and casts may seem unduly few in proportion to the albumin. If the blood-pressure is very high the diagnosis is more likely to be arteriosclerosis or granular kidney than primary heart-failure, though, curiously enough, the blood-pressure is generally above normal in heart-failure from any cause, even when the pulse is as irregular and feeble as it often is in the late stages of mitral stenosis. This terminal rise of blood-pressure in heart cases probably results from the partial asphyxia.

Severe Anæmias often cause ascites, but they do not give rise to much difficulty in diagnosis, because the sub-acute or chronic peritonitis which is the cause of the ascitic exudate in these cases arises, as a rule, comparatively late in the disease, after the diagnosis has been made on other grounds, by blood-counts and otherwise. (See ANÆMIA, p. 20; SPLEEN, ENLARGEMENT OF, p. 628; LYMPHATIC GLAND ENLARGEMENT, p. 376.) One need not do more here than refer to the huge enlargement of the spleen without lymphatic glandular enlargement, and the great leucocytosis with a large portion of myelocytes, in *spleno-medullary leukæmia*; the considerable leucocytosis, the enlargement of the lymphatic glands and probably of the spleen, and the great relative increase of the small lymphocytes, in *lymphatic leukæmia*; the enlargement of the lymphatic glands and of the spleen, and the absence of any positive blood changes, beyond anæmia of the chlorotic type without leucocytosis, in *Hodgkin's disease*; the enlargement of the spleen, the absence of lymphatic glandular enlargement, and the occurrence of a progressive and ultimately severe anæmia, of the simple chlorotic type without leucocytosis, but with an occasional myelocyte and basophile corpuscle, in *splenic anæmia* (which often, as the course of the disease goes on, turns out to be cirrhosis of the liver); the profound anæmia and the high colour-index without leucocytosis, in *pernicious anæmia*; the severe anæmia suggestive of pernicious anæmia, but with a persistently low colour-index, in *aplastic anæmia*; and the splenic enlargement with profound chlorotic anæmia without leucocytosis, in *pseudo-leukæmia infantum*.

Herbert French.

ATAXY is the term used to describe voluntary movements which are imperfectly controlled or co-ordinated. It is displayed in its simplest form by infants under the age of one year. In pathological states, it is often a symptom of great diagnostic importance; but before its value as a localizing sign of disease can be utilized, it is necessary to appreciate broadly the physiological mechanism by which co-ordination is brought about, and the possible situations where a lesion is able to disturb the smooth working of that mechanism. For the proper co-ordination of voluntary movement, impulses from the muscles, tendons, joints, and skin of the part which is moved must reach the brain. These impulses are of two kinds:—

1. Sensory afferent impulses which are carried to the *cerebrum* by way of the peripheral nerves, the posterior columns of the cord, the fillet, and finally from the basal ganglia to the cortex in the neighbourhood of the motor area. These impulses cross from one side of the body to the opposite hemisphere, the crossing taking place in the medulla.
2. Non-sensory afferent impulses, so-called because they never reach consciousness, pass from the peripheral structures concerned in movement, by way of the peripheral nerves and the ascending cerebellar tracts of the cord, to the *cerebellum*, and principally to the cerebellar lobe of the same side of the body. In some manner which is not perfectly understood, but in which preservation of muscular tone is probably concerned, the co-operation of the cerebellum is required if movements initiated in the motor area of the cerebrum are to be carried out in a co-ordinate manner.

Not only must these two sets of impulses reach the brain, but the parts of the brain, cerebral and cerebellar, which form their destination, must also be intact if voluntary movement is to be carried out with accuracy and co-ordination.

From the clinical point of view it is necessary to ascertain in the first place whether a patient is ataxic, and in the second whether the ataxy can be attributed to the loss of the sensory or non-sensory afferent impulses. In some cases the ataxy is obvious; in others it can be detected only by the careful application of certain tests. For instance, a patient may walk into a well-lighted room with perfect ease and without anything remarkable in his gait, but if he is asked to walk along a line, placing one foot exactly in front of another, he may at once display his lack of co-ordination. Such ataxy is just as important from a diagnostic standpoint as the imperfect attempts of an advanced tabetic patient to walk even when supported by companions on either side. It is the quality and not the quantity of a defect which gives the needed information.

The co-ordination of movements performed by the upper extremities must also be investigated with the same care. The patient may handle his stick in quite a natural manner, but if asked to unbutton and button his coat, to touch the tip of his nose with the tip of his finger, to write, etc., he may fail to convince the observer that his control of fine movements is up to the normal standard.

Having ascertained the existence of ataxy, the next step is to decide whether it is dependent on the loss of sensory or non-sensory afferent impulses, or on the imperfect function of the cerebrum or cerebellum. If the ataxy is due to loss of sensory impulses, it will be increased by the loss of visual impulses brought about by closing the eyes. It will also be possible to demonstrate the loss of sensory impulses by asking the patient to describe the position of a limb with his eyes closed after it has been moved by the observer. When these two tests are positive, it may safely be assumed that the lesion affects the first set of impulses or their cerebral destination.

If, on the other hand, the ataxy is uninfluenced by closing the eyes and the patient is perfectly accurate in describing the position of his limbs, it is probable that the cerebellar tracts are at fault, or the cerebellum itself.

For further localization of the lesion in any particular case it will be necessary to take into account concomitant phenomena.

Interference with the passage of impulses necessary for proper co-ordination may be provoked by lesions in (1) *The peripheral nerves*; (2) *The spinal cord*; (3) *The brain-stem*; (4) *The cerebrum*; and (5) *The cerebellum*. Let us now consider the effect of lesions in these different regions, and the diagnostic evidence as to their localization afforded by ataxy.

1. **Peripheral Nerves.**—A severe lesion of a peripheral nerve must lead to ataxy of movements performed by the muscles to which it is distributed; a severe lesion will also paralyze the muscles, however, and thus prevent any ataxy being demonstrated. Less severe lesions, such as occur in slight cases of peripheral neuritis, allow of some voluntary movement, so that ataxy becomes demonstrable. Thus a case of peripheral neuritis of alcoholic or diphtheritic origin may show impaired strength, together with ataxy in all four limbs. The diagnosis of a peripheral nerve affection in such a case will depend on the following points: In the first place, the symptoms will be found to be symmetrical, and in the affected limbs the impairment of strength will be most marked in the extensors of the wrists and ankles. Secondly, slight anæsthesia to cotton-wool may be detected over the glove and stocking areas. With regard to pain (p. C06), there may be blunted cutaneous sensibility to the prick of a pin over the same area, but almost constantly, deep pressure on the affected muscles will establish the fact that these tissues are abnormally sensitive. This is a most important point in diagnosis, because it strikes an essential distinction between cases of ataxic peripheral neuritis, sometimes described as pseudo-tabes, and cases of true spinal tabes, in which it is an almost invariable rule to find diminution or loss of painful sensibility on squeezing the muscles. In the third place, the tendon reflexes will be markedly diminished or completely absent, while the plantar reflexes will probably be unobtainable. Finally, the use of electrical currents upon the muscles will show that the response to faradic currents is materially lessened or abolished, and that the contraction excited by the make and break of the galvanic current may be of the slow, worm-like type so characteristic of the reaction of degeneration (p. 582).

The ataxy of peripheral neuritis has in itself no reliable characteristic to distinguish it from ataxy due to spinal disease. That it is due to a lesion of the peripheral nerves is concluded not from the nature of the ataxy, but from the presence of other symptoms also referable to interference with the functions of the nerves. The gait is unsteady, and the patient keeps his legs apart in order to lessen the tendency to lose his balance. The clumsiness of the upper extremities may be demonstrated by his inability to bring the first finger of one hand accurately into apposition with that of the other, or to touch the tip of his nose with either. Both the unsteadiness of gait and the awkwardness of the fingers are exaggerated if he attempts to walk, or carry out movements with his hands when his eyes are closed. A tendency to high-steppage will be noticeable in walking if, in addition to the ataxy, there is well-marked paresis of the dorsiflexors of the ankles. In such a case the patient is obliged to lift the feet to an unusual height in order to clear the ground.

2. **Spinal Cord.**—The ataxy due to disease of the spinal cord is seen best in *tabes dorsalis*, in which malady degeneration of the posterior column ascending tracts occurs early, and in which, consequently, the patient does not receive the normal impulses from the muscles, tendons, and joints so necessary for the preservation of his sense of position and movement. Contrary to popular ideas, gross ataxy is met with only in a small proportion of the cases of this disease, and it is often necessary to apply delicate tests to demonstrate its presence. The patient's gait may not be remarkable in good daylight, but he may complain of its uncertainty in the dark, or he may be obviously ataxic with his eyes closed. Another patient may have noticed nothing amiss with his walking in the ordinary way, but if he is asked to follow a line on the floor, placing one foot exactly in front of the other, his impaired power of balance will become apparent, especially if he is directed to accomplish this test with his head raised and his eyes fixed on something in front of him instead of upon his feet.

In cases of moderate ataxy the gait and stance of the patient are remarkable for the wide base he assumes, and his tendency to guide his feet by means of his vision. Romberg's sign can be obtained easily. This sign is not diagnostic of tabes, as is so often assumed, but is merely used for the purpose of ascertaining whether the removal of visual impulses will convert a condition of stability into one of instability. Many if asked to describe Romberg's sign, reply, " You direct the patient to put his feet together and close his eyes ; if he sways or falls, the sign is present." This is obviously incorrect, because the patient may sway even before his eyes are closed. In order to test a patient for this sign, he must be directed to stand with his feet as near together as he is able to do with steadiness, and, having established his stability in that position with open eyes, he must be told to close the latter. If he sways or tends to fall, it is clear that he had been depending on his visual impulses, and that, without their aid, the impulses derived from his legs and trunk were insufficient for the preservation of his equilibrium. We have in this test, therefore, a valuable method of ascertaining whether the function of the posterior columns is being carried out normally.

To judge from the descriptions given in some text-books, the typical gait of tabes is one in which the legs are thrown into the air and the feet brought to the ground with a more or less noisy stamp. As a matter of fact, this type of gait is seen only in a small proportion of cases, and is rarely observed except when the patient is depending for support either on a couple of sticks or on one or two attendants. In other words, he has become so ataxic that he cannot walk unsupported, and, being supported, he no longer attempts to control the exuberance of his leg movements by means of his sight.

Tabetic ataxia in its moderate and extreme degrees can be demonstrated when the patient is at rest in bed, by asking him to carry out accurate movements with his hands and feet, with and without the aid of his vision. In slighter degrees the fact that the ataxia is dependent on interference with his sense of position and movement may be proved by asking him to describe the position of a finger or toe which the observer moves in different directions. Sometimes it is as well in testing this sense in one limb to ask the patient to place the corresponding limb in the same position, when the error will be made more obvious.

The diagnosis of tabes cannot be made from the character of the ataxy alone, since in other diseases, such as Friedreich's ataxy, disseminated sclerosis, or combined degeneration of the cord, there is or may be sclerosis of the posterior columns resulting in similar inco-ordination. It is important, therefore, to remember that in tabes the posterior roots are affected also, and that there is very often some interference with other afferent impulses, especially those which convey sensations of pain from the muscles and skin, and those which are concerned with the deep reflexes and the maintenance of muscular tone. Thus, in this disease one of the earliest symptoms is relative analgesia to pin-pricks and to deep pressure on the muscles in the lower extremities ; at the same time it must not be forgotten that the tabetic phenomena may be limited to the upper extremities (cervical tabes).

In ***Friedreich's ataxy, disseminated sclerosis***, and other spinal disease, as well as in some cases of tabes, the ataxy due to the lesion of the posterior columns may be complicated and intensified by the fact that there is also interference in the path of the non-sensory afferent impulses, which pass from the extremities to the cerebellum *viâ* the ascending cerebellar tracts in the spinal cord. If this form of ataxy is present, the help which the patient derives from vision for the purpose of controlling his inco-ordinate movements is largely discounted, and he may be as ataxic with open as with closed eyes.

In some lesions, such as those resulting from *syringomyelia* or *new growths*, only one side of the cord may be affected, and a Brown-Séquard form of paralysis be exhibited (p. 437). If the paralysis is not complete, some ataxy may be observed in the paretic limb.

3. **The Brain-stem.**—Lesions of the medulla, pons, or crura may produce ataxy if they interfere with the passage of either sensory afferent impulses to the cerebrum or non-sensory afferent impulses to the cerebellum. The cerebellar impulses can be interfered with only at the medullary level; that is to say, before they have passed into the cerebellum *viâ* the inferior peduncle. A good example of hemiataxia of this origin is afforded by any case of *thrombosis of one posterior inferior cerebellar artery.* This uncommon condition affects the structures on one side of the medulla, and is characterized by hemiataxia of the homolateral limbs, together with loss of sensibility to pain, heat, and cold, on the contralateral side. The ataxy is of the cerebellar type; that is to say, it is not associated with loss of sense of position and movement in the affected limbs, and is little influenced by closure of the eyes. Above the medulla, lesions which are capable of producing ataxy by interfering with the sensory impulses from the muscles, joints, and tendons, usually cause paralysis of the same parts, so that the co-ordination is more latent than real, and therefore of little diagnostic importance.

4. **The Cerebrum.**—From the basal ganglia to the cortex, the path of the afferent impulses necessary for co-ordinate movements lies near to that of the efferent impulses from the motor area, and it is only rarely that lesions affect the sensory fibres and leave the motor intact. Every now and then, however, a patient complaining of loss of use of the limbs on one side, is found on examination to be suffering from impaired sense of position and movement in those limbs rather than from paralysis. His co-ordination may be fairly good so long as he can utilize his vision, but with closed eyes he has no notion of the position of his arm or leg, and no knowledge of the nature of objects placed in his hand (*astereognosis*). This may even be the case when other sensory stimuli, such as those of touch, pain, and heat, are appreciated perfectly. A similar condition may be observed during recovery from a slight hemiplegic 'stroke,' the patient displaying a degree of clumsiness and awkwardness with his fingers quite out of proportion to his loss of voluntary power. A process of re-education for finer movements, similar to the education of early life, is necessary before he is able to overcome this form of ataxy.

Ataxic movements are not uncommon in the subjects of *infantile hemiplegia.* The hand on the affected side may be permanently clumsy and incapable of carrying out the delicate manipulations necessary for writing, sewing, etc. In other cases all voluntary efforts are interfered with by the constant presence of involuntary movements of an athetotic, choreiform, or tremulous character, sufficient to prevent their attaining any dexterity.

Whatever the nature of the lesion, cerebral ataxy is generally characterized by its hemiplegic distribution, and by its increase when the eyes are closed; generally the loss of impulses subserving the sense of position and movement, and often of other sensory impulses, can be demonstrated by suitable tests.

5. **The Cerebellum.**—Cerebellar ataxy may be unilateral, as in some cases of *tumour* of one lateral lobe, or bilateral, as in the *acute cerebellar ataxia* of children due to encephalitis. In unilateral cases the ataxy is most marked on the same side as the lesion, and is associated with hypotonia and some paresis of the affected limbs. On the other hand, it is important to note that the reflexes on the affected side are normal, that the ataxy is not accompanied by any loss of sense of position and movement, and that closure of the eyes does not materially increase the patient's disability. The ataxy often differs from that due to disease of the posterior spinal column in that it is complicated by vertigo. This may take the form of a sensation of rotation on the part of the patient, or of rotation of surrounding objects, sometimes of both. The vertigo and the ataxy are generally much less noticeable in the recumbent position. The cerebellar gait resembles that of a drunken man; the patient reels from side to side, with a general tendency to deviate or fall to the side of the

lesion if only one lobe is affected. He is unable to balance himself properly on the homolateral foot, and his manual dexterity is impaired, so that he may be unable to feed or clothe himself. The ataxia is not always limited to the trunk and limbs, but may affect the tongue, lips, palate, and vocal cords, so that their movements may be controlled imperfectly, and a characteristic 'cerebellar articulation' attracts attention. Finally, a lesion of the cerebellum sufficient to cause ataxy nearly always causes nystagmus also, which, in disease of one lobe, is more marked during deviation of the eyes to that side.

6. **Hysterical Ataxy.**—Ataxy is sometimes hysterical, and may then be the only disorder of function exhibited by the patient, or may be associated with hysterical hemiplegia, paraplegia, hemianæsthesia, etc. The diagnosis depends partly upon the absence of signs of organic disease, partly on the presence of other hysterical stigmata, and partly on its character. For example, we may cite the case of a boy who, when lying in bed, was able to feed himself and to carry out all movements of his upper and lower limbs with perfect accuracy, but who, when placed on his feet and told to walk, displayed the wildest inco-ordination and loss of equilibrium. It was noticeable, however, that he always reached some chair or bed on which to collapse finally, even when placed in the middle of the room at some distance from any support. It would, of course, be unjustifiable to apply this last test before the observer was satisfied from careful examination that there were no signs of organic disease.

E. Farquhar Buzzard.

ATHETOSIS.—(See CONTRACTIONS, p. 131.)

ATROPHY, MUSCULAR.—Muscular atrophy is often merely part of a *general wasting of the whole body*, due either to chronic lesions such as carcinoma, sarcoma, tuberculosis, syphilis, malaria, ulcerative colitis, marasmus, starvation, hepatic abscess, cirrhosis of the liver, diabetes, anorexia nervosa, or to acuter maladies, such as diarrhœa and vomiting, ptomaine poisoning, typhoid fever, dysentery, cholera, and so forth. The history, and the other symptoms in the case, will usually serve to indicate these. If any doubt remains as to whether the atrophy is neurotrophic or not, the electrical reactions will be tested; there will be no reaction of degeneration (R.D.) when the atrophy is merely part of a general wasting, whereas if —as might be the case in a diabetic patient, for instance—there is peripheral neuritis in addition, this will be indicated by a partial or complete R.D. (See REACTION OF DEGENERATION, p. 582.)

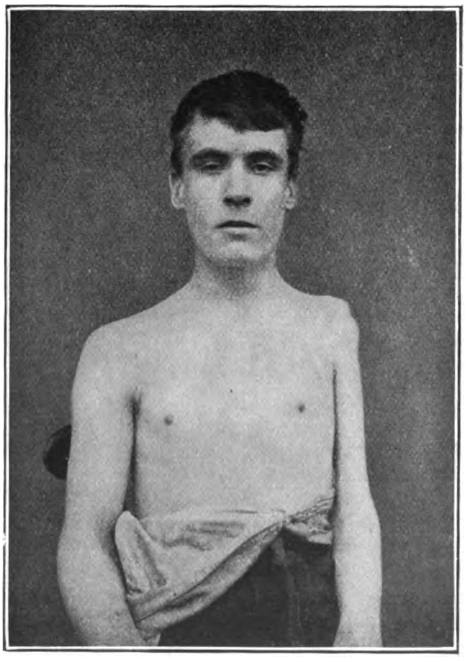


Fig. 19.—Atrophy of the muscles of the left shoulder and upper arm, the result of former acute anterior poliomyelitis—infantile paralysis.

In the next place, the atrophy may be the result of *disuse*. Organic disease of the nervous system may or may not be present at the same time; the patient may be bedridden from locomotor ataxy, for example, or from general paralysis of the insane; and the muscles of the limbs may consequently become so thin that peripheral neuritis or degeneration of the anterior cornual cells may be simulated, and a determination of the absence of R.D. may be the only means of excluding these. It is important to remember that in the *primary muscular dystrophies*, whether of the pseudo-hypertrophic, the juvenile, the infantile, the facio-scapulohumeral or Landouzy-Dejerine or other types, there is no reaction of degeneration, the electrical responses and the superficial and deep reflexes remaining normal in type, though they diminish in degree as the amount of muscle grows less and less, until finally there is no muscle to respond at all. The primary muscular dystrophies (p. 518) are comparatively easy to diagnose, however, on account of their insidious onset in children, their slow but progressive downhill course, their occurrence in different members of the same family, the absence of sensory disorder, and the absence of R.D. They are distinguished from the *infantile paralysis* which results from acute anterior poliomyelitis (*Fig.* 19) by the latter having a sudden onset, R.D. at its height, whilst the resultant wasting does not advance progressively, but after recovering to a certain degree, tends to remain stationary.

Peripheral neuritis is distinguished from primary muscular dystrophy by the history and course, and by the presence of R.D. at some period of the malady. Two other affections that may be confused with a primary muscular dystrophy—particularly as they also are hereditary, begin insidiously at an early age, and slowly advance—are *Friedreich's ataxy*, and *Tooth's peroneal type of progressive muscular atrophy*. Each of these may cause talipes, moreover, and therefore simulate infantile paralysis, except that in the latter the talipes is generally one-sided, whereas in the other two it is bilateral. In Friedreich's ataxy (see p. 512) there is no real wasting, but rather a lack of development. Tooth's peroneal type of progressive muscular atrophy is apt to come on after some febrile malady such as measles or whooping-cough, the first thing noted being inability to dorsiflex the big toe, which hangs down in a way that is the exact converse of its erect position in Friedreich's ataxy (*Fig.* 20); the paresis takes months or years to spread to the rest of the legs, and finally to the hands (*Fig.* 21), the slowness of the progress and the absence of sensory symptoms showing that it is not peripheral neuritis, whilst the R.D. in the affected muscles excludes

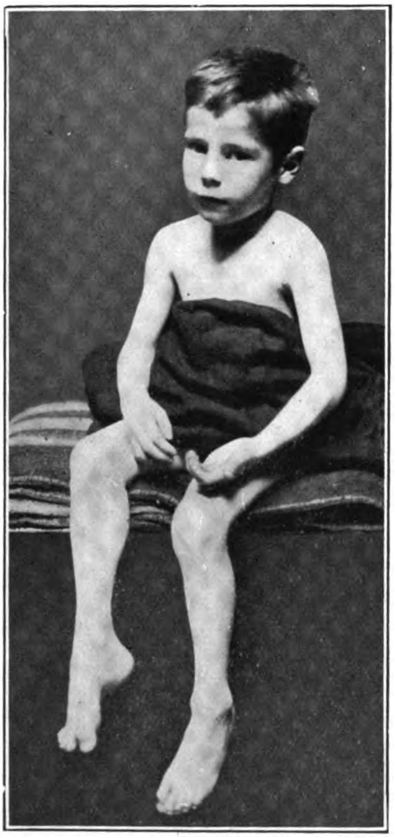


Fig. 20.—Tooth's peroneal type of neuro-muscular dystrophy—early; the patient is the younger brother of the girl in *Fig.* 21. Note the plantar-flexion of the big toes and the dropping of the feet; the calves are not yet wasted.

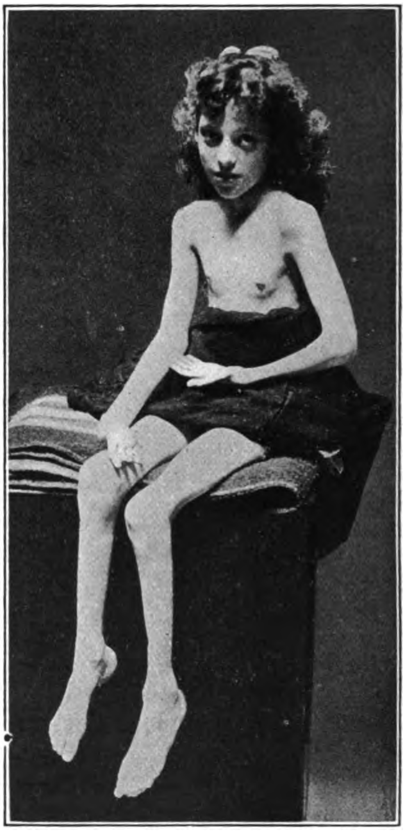


Fig. 21.—Tooth's peroneal type of neuro-muscular dystrophy: the patient is sister to the boy in *Fig.* 20; her malady is in a much more advanced stage than is his, wasting having extended to the calves, thighs, hands, and forearms.

a primary muscular dystrophy. The lesion is in the anterior cornual cells and starts in the lumbar enlargement. The knee-jerks are retained until the quadriceps of the thigh is involved.

Local muscular atrophy may be due to *disease of the parts beneath*, as in the case of the pectoralis major, the supraspinatus, the deltoid, the infraspinatus, and other shoulder muscles when the underlying lung is the site of active phthisis. Similar local atrophy results very quickly from acute and subacute affections of joints, especially in the muscles whose origin is above the affected joint. The gluteal atrophy associated with tuberculous hip-joint is well known; similarly, knee-joint disease leads to thigh atrophy, elbow disease to atrophy of the muscles of the upper arm, and so on. The same applies to the effects of fractures, new growths, sprains, and splints; the atrophy is sometimes so rapid that some think it cannot be due simply to disuse, but must have a neuropathic factor also. The affected muscles present no R.D., however. One particular form of paralysis associated with the use of splints merits special mention, namely, Volkmann's paralysis of the forearm. (See PARALYSIS OF THE UPPER EXTREMITY, p. 503.)

Hemiatrophy of the face or trunk is generally congenital, and the diagnosis is not difficult (see p. 494).

If it can be decided definitely that there is some nervous cause for muscular atrophy, the best proof of which is the detection of partial or complete R.D., the diagnosis lies between one or other of the following conditions:—

1. Causes in the Spinal Cord.—

Progressive muscular atrophy
Amyotrophic lateral sclerosis
A few cases of transverse myelitis
Syringomyelia
Tooth's peroneal type of progressive muscular atrophy
Acute anterior poliomyelitis

2. Causes in the Peripheral Nerves.

Tumours of the cauda equina
Pelvic tumours involving the lumbo-sacral plexus
Sciatica
Aneurysm
New growth
Accessory cervical rib, etc., pressing on the brachial plexus
Gummata, etc., involving the cranial or other nerves
Injury to peripheral nerves, including the effects of callus after fractures

Peripheral neuritis, of which the following are some of the causes:—

- Certain inorganic chemical substances, notably
  - Lead
  - Arsenic
  - Mercury
- Certain organic chemical compounds, notably
  - Alcohol
  - Ether
  - Carbon bisulphide
  - Naphtha
- Certain severe anæmias:
  - Pernicious anæmia
  - Spleno-medullary leukæmia
  - Lymphatic leukæmia
  - Hodgkin's disease
  - Splenic anæmia
- Certain microbial or allied toxins:
  - Diphtheria
  - Leprosy
  - Malaria
  - Chronic pyæmia
  - Infective endocarditis
  - Beri-beri
  - Syphilis
  - Typhoid fever
  - Influenza
  - Oral sepsis
- Certain constitutional diseases sometimes attributed to endogenous poisons:
  - Gout
  - Diabetes mellitus
- Pregnancy
- Other causes as yet undetermined.

In arriving at a diagnosis in a particular case, it is important not to use the term 'neuritis' until all the other possible lesions have been excluded. Tooth's peroneal type of progressive muscular atrophy and acute anterior poliomyelitis have already been discussed. The latter is sometimes regarded as essentially a disease of early life, but it is important to remember that it is by no means impossible for it to affect an adult, in whom the symptoms and results may be precisely similar to what they would be in a child.

Progressive muscular atrophy is a disease of adults. It shows no particular tendency to occur in several members of the same family. It begins insidiously, and advances slowly for months and years, affecting first the small muscles of the hands, causing atrophy with R.D. in the interossei and in the muscles of the thenar and hypothenar eminences; the peculiar deformity described as 'main-en-griffe' results (p. 109). In the course of months the paresis spreads from the hands to the forearm, and later to the upper arm. Disease of the peripheral nerves, such as the ulnar, is excluded by the fact that the paralyzed muscles are not all supplied from the same nerve trunk—the thenar muscles supplied by the median being affected equally with the hypothenar supplied by the ulnar. All the muscles below the wrist are involved more or less together, then all the muscles below the elbow, and so on; this paralysis of associated groups of muscles as distinct from muscles supplied by the same nerve, at once suggests a progressive degeneration of the anterior cornual cells of the cervical enlargement of the cord. Disease of the brachial plexus would be excluded first by the fact that the lesion is bilateral and symmetrical, and secondly by the absence of pain or other sensory disturbance. The pathology of the disease is analogous to the nuclear cell-degeneration in the medulla oblongata that leads to bulbar (labio-glosso-pharyngo-laryngeal) paralysis; and indeed, progressive muscular atrophy may either follow or be followed by bulbar paralysis.

If, at the same time that there are the signs of progressive muscular atrophy in the hands, there is also spastic paresis of the legs, with no wasting, but increased knee-jerks, ankle clonus, and extensor plantar reflexes, the onset having been quite gradual, without sensory disorder, and without bladder or rectal trouble unless the disease has reached quite a late stage, the condition is *amyotrophic lateral sclerosis.*

It is important that the character of the onset and the absence of sensory symptoms be insisted on, in order to exclude syringomyelia and anomalous cases of transverse myelitis. *Syringomyelia* is rare, but it has one very characteristic feature, namely, the preservation of ordinary cutaneous sensibility with the loss of power of distinguishing heat from cold, or pain from touch, in some part of the limbs or trunk. There need be no other symptom than this dissociation of sensations, or skin lesions in the paræsthetic parts may be a prominent feature—*Morvan's disease*; if the enlargement in and around the central canal of the cord displaces and destroys the anterior cornual cells in the lower part of the cervical enlargement, progressive muscular atrophy is simulated; if at the same time the bulging of the central canal and the changes around it cause compression of the crossed pyramidal tracts, there will be all the motor symptoms and signs of amyotrophic lateral sclerosis, the diagnosis being only possible when the sensory symptoms are typical.

It is generally stated that *transverse myelitis* causes spastic paraplegia without muscular wasting or R.D. This is in the main true, because the few anterior cornual cells destroyed by the transverse softening of the cord in the commonest site, namely, the dorsal region, correspond to an intercostal or abdominal segment, the wasting of which is difficult to detect. If, however, the transverse myelitis occurs so high up as to involve the lower part of the cervical enlargement—to involve the cord yet higher up is incompatible with life, because both the intercostals and the phrenic nerves would be paralyzed—a certain number of the anterior cornual cells sending motor nerves to the hands and arms would be destroyed, the result being a main-en-griffe like that of progressive muscular atrophy; and the simultaneous interference with the crossed pyramidal tracts would produce a picture identical at first sight with amyotrophic lateral sclerosis. Not only, however, would there very likely be impairment of all forms of sensation as well as paresis, in a case of transverse myelitis, but instead of the onset being gradual and the progress a steady advance downhill, as in progressive muscular atrophy or amyotrophic lateral sclerosis, the onset would have been comparatively rapid, followed by a cessation or even by an improvement if the patient lived. Similarly, if transverse myelitis occurs so low down as to involve the lumbar enlargement of the cord, it would cause, not spastic paraplegia with increased knee-jerk, ankle clonus, extensor plantar reflex, no wasting and no R.D.; but absence of knee-jerk, no ankle clonus, no extensor plantar reflex, marked muscular atrophy of the legs, with R.D., paræsthesia, bladder and rectal trouble. The involvement of the sphincters in such a case would be of considerable aid in excluding peripheral neuritis; whilst Tooth's peroneal type of progressive muscular atrophy and acute anterior poliomyelitis would be excluded not only by the paræsthesia, but also by the history of the mode of onset and the course of the malady.

A *tumour involving the cauda equina* is rare, but it is not altogether difficult to diagnose. It may be more difficult to determine the nature of the mass—gumma, glioma, primary sarcoma, secondary sarcoma or carcinoma—than its site. The onset of symptoms is generally gradual, and one leg is affected either earlier than, or more than, the other. Weakness in the leg, together with severe pains both in it and in the lower part of the lumbar region of the spinal column, will be followed by muscular atrophy and R.D. Sciatica may at first suggest itself, until it is found that neither the pains nor the paresis correspond to one single nerve; and when the disease progresses and the other leg is affected, anæsthesia supervenes upon the paralysis. The site of the pain over the region of the cauda equina is an important point in the diagnosis, whilst rectal and possibly vaginal examinations are essential for the exclusion of a pelvic mass—such as carcinoma of the rectum, uterus, or ovary, a fibromyoma, a cyst, a sarcomatous, gummatous, tuberculous, or inflammatory mass, or even a displacement of the womb—which, by interfering with the nerves at the back of the pelvis might produce very similar symptoms. Sacro-iliac joint disease can generally be excluded by the fact that the pains are not definitely referred to the joint, whilst any wasting that might be associated with disease of that joint would not be accompanied by R.D.

Sciatica (p. 438) does not always give rise to wasting of the corresponding muscles; but sometimes it does, and occasionally it may do so bilaterally, with R.D. The localization of the pain, tenderness, and atrophy to the parts supplied by the great sciatic nerve, without affection of other nerves and muscles in the leg or calf, would point to sciatica, especially if the lesion were unilateral, and if the patient, though unable to flex his thigh to a right angle with his abdomen at the same time that he keeps his knee extended, can extend his leg backwards at the hip-joint in a way that would be impossible if he had a psoas abscess; and if he is able to bear firm backward pressure on the knee when the leg of the affected side is flexed and outwardly rotated in such a way that the foot lies across the opposite knee—a test which will exclude hip-joint disease.

When the lesion is a *thoracic aneurysm* or *neoplasm*, or *an accessory cervical rib pressing on or involving the brachial plexus*, the wasting is almost certain to affect one arm only, or one arm much more than the other, and the diagnosis will be made by physical examination of the thorax, assisted by the x-rays.

The only *cranial nerve paralyses* that are likely to be associated with marked atrophy of muscles, are those of the seventh with facial atrophy (p. 493), and of the twelfth with atrophy of the tongue.

Injuries to peripheral nerves, or inclusion of the latter in callus, will generally be diagnosed by the history, and by the fact that in distribution the muscular atrophy and R.D. correspond accurately with one or more of the peripheral nerves that may have been divided or otherwise injured.

If all the conditions described above can be excluded, it is probable that the cause of the muscular atrophy is some variety of *peripheral neuritis*. To merit this diagnosis, the affected muscles should be multiple and symmetrical; partial or complete R.D. should be obtained; there may or may not be sensory changes; the reflexes, both superficial and deep, are for a short time exaggerated, and then become deficient or disappear altogether for the time being. Wasting may be extreme, but the tendency is for slow recovery to ensue, improvement beginning to set in some three or four months after the neuritis ceases. Sometimes the nature of the case is obvious, but it is often easier to diagnose peripheral neuritis than to discover its exact cause. The different conditions that may produce it are enumerated above. In diagnosing between them the history is very important. For instance, if the patient has never been abroad *leprosy* and *beri-beri* are unlikely, whereas if he has been abroad amongst lepers, and if he has areas of anæsthesia without much paresis, with or without the characteristic nodules and bosses of subcutaneous infiltration (*Fig.* 173, p. 401), followed by ulceration and necrosis, the diagnosis of leprosy will at once suggest itself. The chief difficulties will perhaps be to exclude syringomyelia on the one hand and tertiary syphilis on the other. The good effects of treatment by potassium iodide and mercury may assist in detecting syphilis, and Wassermann's reaction may be positive; in syringomyelia there is little or no loss of cutaneous sensibility like there is in leprosy, though there is loss of power to distinguish heat from cold, and pain from touch. The ultimate test of leprosy would be to excise a small portion of the affected tissue and to examine it for the acid-fast leprosy bacilli.

Beri-beri is sometimes seen in this country, generally in patients who have come into port in a ship from the East; several of the crew have generally been affected at the same time, some may have died; the peripheral neuritis and muscular wasting will often be associated with œdema, and there is often a history that the dietary has consisted of decorticated rice.

The presence or absence of glycosuria will serve to diagnose or exclude *diabetic neuritis*. Loss of knee-jerk in diabetes mellitus is comparatively common, but extensive peripheral neuritis is much rarer. It is associated with pain and paræsthesia as well as paresis and muscular atrophy, and it affects the limbs, especially the legs, rather than the trunk.

Gout as a cause of peripheral neuritis is always open to doubt, for often the neuritis of a gouty subject is really due to the indulgences that brought on the gout. Difficulty may also arise in attributing a neuritis to *pregnancy* even when the patient is, or has been recently, pregnant.

In the case of *blood diseases* it is important to bear in mind that these are usually treated with arsenic, so that the peripheral neuritis may be due to the treatment rather than the disease. This will be rendered the more probable if there are or have been other symptoms of subacute or chronic arsenical poisoning, such as coryza, nausea, vomiting, abdominal colic, diarrhœa, headache, pigmentation of the skin not unlike that of Addison's disease, hyperkeratosis of the palms and soles, or herpetiform eruptions. With arsenical neuritis the limbs are involved most, particularly the legs, and there are pains and paræsthesia as well as paresis. The blood diseases may themselves cause peripheral neuritis, however, just as severe anæmias, such as pernicious anæmia, may cause degeneration in other parts of the nervous system also, notably in the long tracts in the spinal cord, with consequent sensory, ataxic, or paretic symptoms, varying with the parts involved. If the peripheral neuritis occurs early in the blood disease, the latter may not come to mind as a possibility. A blood-count is essential (p. 24). Oligocythæmia with high colour index, no leucocytosis, a relative lymphocytosis, and the presence in blood films of a preponderance of megalocytes, are changes characteristic of *pernicious anœmia,* in addition to which the primrose-yellow skin may be typical. Great increase in the total number of leucocytes up to anything from 50,000 to 1,000,000 per c.mm. would suggest *leucocythœmia* : if this were the spleno-medullary form, myelocytes would probably be 30 per cent or more of all the white cells seen in films, whilst in the lymphatic form the lymphocytes would similarly amount to 90 per cent ; in both forms, particularly the spleno-medullary, the spleen and liver would be big, whilst in the lymphatic type there would probably be general enlargement of the lymphatic glands.

Hodgkin's disease or *lymphadenoma* suggests itself when the spleen and many of the lymphatic glands are enlarged, without any characteristic blood changes—at most a simple anæmia without leucocytosis, with relative lymphocytosis, and an occasional myelocyte, basophile corpuscle, and nucleated red cell in films. *Splenic anœmia* is a doubtful entity, the name being applied when there is simple anæmia with apparently idiopathic enlargement of the spleen. Many such patients ultimately turn out to have cirrhosis of the liver —Banti's disease. Peripheral neuritis in such a case may well be alcoholic.

Malaria will be diagnosed by the history, and by the discovery of the hæmatozoa in the blood (p. 29). The difficulty may be to exclude alcohol as a cause for the neuritis in a patient who has also suffered from severe malaria.

Infective endocarditis is sometimes so chronic and insidious that it escapes detection. Points to lay stress on are summarized on p. 34.

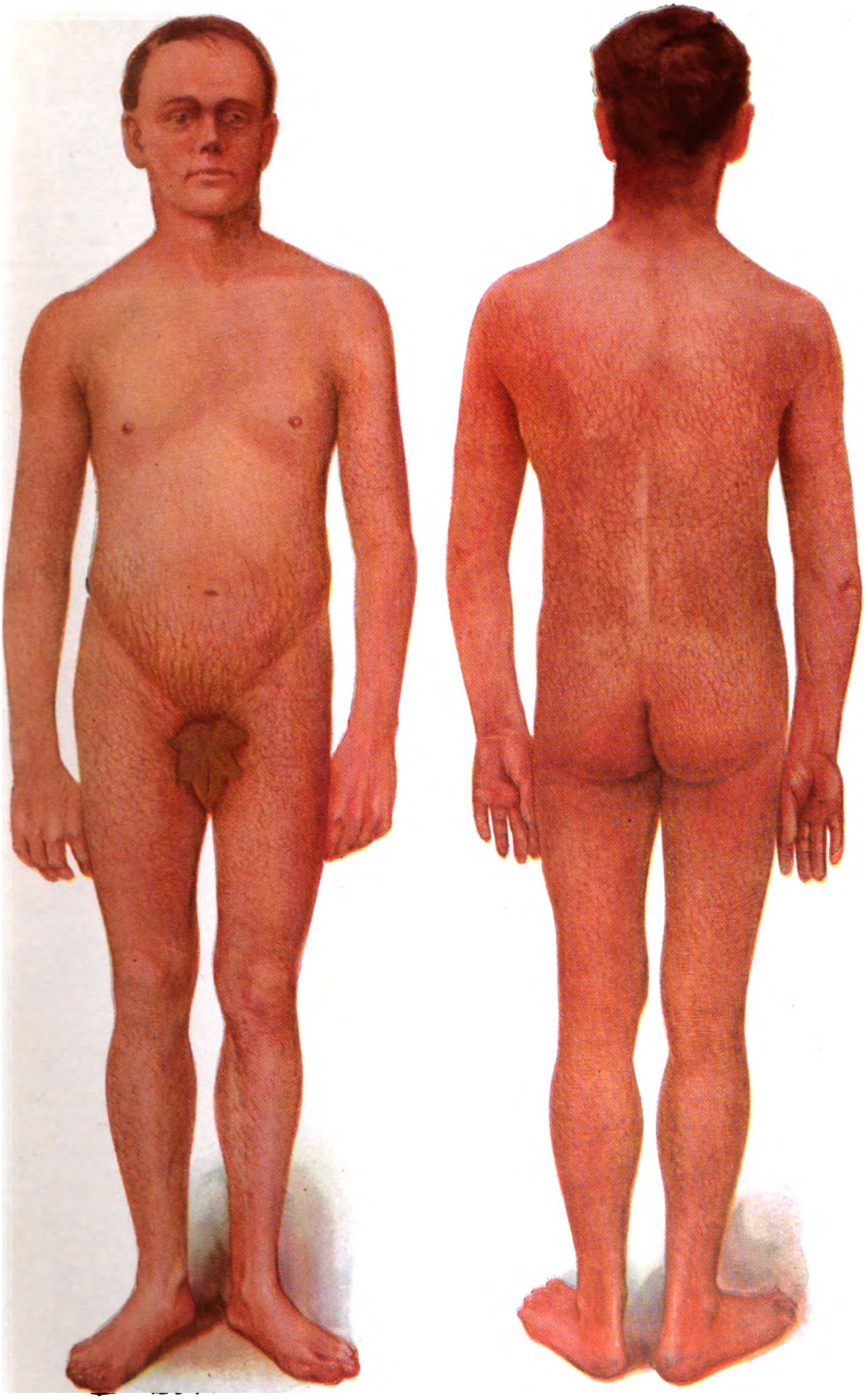
It may not be easy to convince oneself that some other cause of *chronic pyœmia,* whether uterine, pelvic, pulmonary, oral, or otherwise, is the cause of peripheral neuritis in a given case. The same applies to *syphilis,* especially if the patient is also addicted to alcohol.

Influenza is not to be diagnosed as the cause until every other possible explanation has been exhausted ; it is too easy to attribute things to influenza. Peripheral neuritis from *typhoid fever* generally arises as a direct sequel of a typical attack confirmed by Widal's test, so that the diagnosis is not difficult as a rule. It has the same type, sensory and motor, as arsenical neuritis.

Diphtheria is one of the most important of all the causes, and if the diphtheria itself has been slight, it may have been overlooked entirely, especially as the neuritis develops two or three weeks or longer after the sore throat. It is important, therefore, to lose no time in taking cultivations from the throat in all doubtful cases of peripheral neuritis ; it may still be possible to find the causal organisms in swabbings. The nature of the case may be suggested at once, however, if there has been a nasal alteration in the voice (p. 589), or if there is an inability to swallow liquids owing to their regurgitation through the nose—evidence of paralysis of the palate that is almost characteristic of diphtheria ; the pupil reflexes are also apt to be affected, and the patient may be thought to have an error of refraction because paresis of the ciliary muscle renders accommodation difficult or

PLATE VII

PIGMENTATION DUE TO ARSENIC



From a case of Hodgkin's disease in which marked pigmentation of the skin, both general and of the net-work type, developed under arsenical treatment.

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impossible for the time being. The symptoms may stop at the palate and eye; but in bad cases—perhaps as the result of a toxin different from that which directly affects the palate—paralysis and extreme atrophy of the limbs, without much sensory disorder, follow. The vagus nerves may be involved, causing tachycardia, and perhaps death; equally serious may be the involvement of the phrenic nerves, with weakness or paralysis of the diaphragm.

In regard to the *various chemical substances* that may produce peripheral neuritis, inquiries into the patient's occupation may assist the diagnosis. Workers amongst india-rubber come in contact with *carbon bisulphide* fumes, this compound being used to dissolve the rubber. *Naphtha* is used extensively in some trades. The use of a chemical may not always be obvious until careful inquiries are made—for instance, one may not at first see what a person who prepares rabbit skins for conversion into hats has to do with mercury, until it is learned that mercurials are used to preserve the pelts. *Mercurial neuritis* is characterized by a remarkable tremor of the hands and arms, in addition to the muscular atrophy in the arms and legs; there are not many sensory symptoms as a rule. *Lead neuritis* is easily diagnosed when it causes the characteristic wrist-drop, though a similar paralysis of the hand may be due to other forms of peripheral neuritis such as diphtheritic (*Fig.* 22), or to the result of compression of the musculospiral nerve by callus or crutch-head, or by sleeping with both arms across the arms of a chair—'Saturday night palsy'; in plumbic wrist-drop all the muscles supplied by the musculospiral nerve beyond the triceps become paralyzed, except the supinator longus and the extensor ossis metacarpi pollicis, and there is no sensory disorder; the escape of the supinator longus distinguishes wrist-drop due to plumbism from that due to compression of the musculospiral nerve; the diagnosis is confirmed by finding a blue line upon the gums and the other signs of lead poisoning described on p. 34. The difficulty arises in less typical cases in which the lead causes generalized peripheral neuritis in both legs and arms, perhaps without any other symptoms, without even a blue line upon the gums if the teeth are kept clean. The source of the lead may be very far from obvious—it may be some obscure thing, such as a hair-wash, or the result of water contamination due to electrolysis in water-pipes, the result of leakage in an electric main. In case of doubt it may even be worth while to analyze the fæces or evaporate down a large bulk of urine and apply the ammonium sulphide test for lead to the residue: a drop or two of the latter, allowed to fall into a tall glass full of ammonium sulphide, will cause a white trail to develop in the fluid as the drop descends.

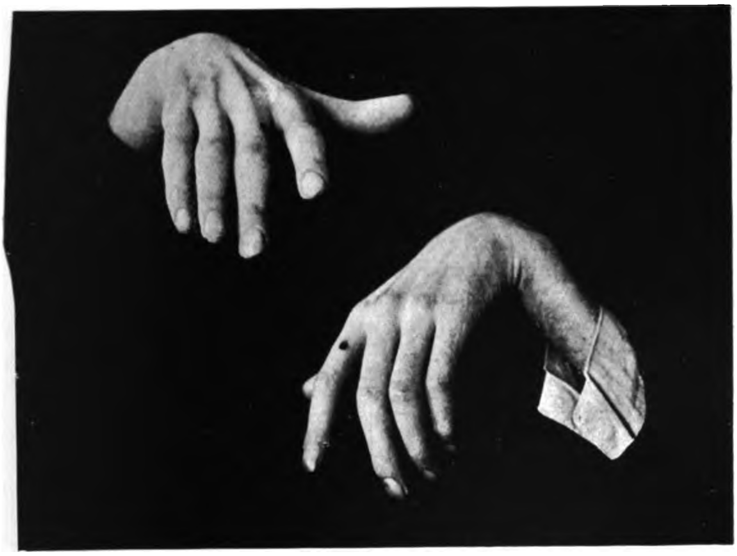


Fig. 22.—Wrist-drop after diphtheria, similar to that of plumbism.

Arsenical neuritis has been mentioned above (p. 64); it may arise in patients who are taking arsenic in medicinal doses, for instance for chorea or pernicious anæmia, or the poison may be taken unawares, as in the Manchester epidemic, in which fatal results followed contamination of beer with arsenic. It has even been held that alcohol itself is no cause of peripheral neuritis, and that those patients who have developed it as the result of long-continued drinking to excess—possibly without a single actual intoxication in the popular sense—owe the nerve trouble and generalized muscular atrophy, not to the chemical substance C_2H_6O, but to other bodies associated with it. Clinically, however, it is sufficient if the diagnosis of the cause of peripheral neuritis can be narrowed down to *alcohol* in some form or other, and for this to be possible an accurate history is essential. The greatest difficulty arises in the case of secret drinkers, especially women who may appear to be above suspicion. The neuritis is ushered in with pains and cramps in the limbs, followed by wasting which may reach an extreme degree; the trunk and limbs sometimes look like those of a person who has been starved to death; if arsenic is suspected, a portion of hair should be sent for chemical analysis; the hair of a person taking arsenic stores the latter in proportions sufficient to allow of its detection.

It only remains to add that there will always be some cases in which the cause of the peripheral neuritis fails to be found.

Herbert French.

ATROPHY, OPTIC.—(See OPHTHALMOSCOPIC APPEARANCES, NOTES ON, p. 416.)

ATROPHY, TESTICULAR.—When one testis is smaller than the other, it is first necessary to determine which is the abnormal one; for when one is slightly enlarged, it may be regarded erroneously as normal and the other as too small. Some inequality may be physiological, as is the case with paired organs generally. Physiological atrophy of the testes is apt to occur in advanced life; it may begin as early as fifty, though many old men have testicles of normal size.

A testis in an abnormal position, in the inguinal canal or elsewhere, is subject not only to such causes of atrophy as may affect one normally situated, but may also be inhibited in growth from compression by surrounding parts.

The causes of atrophy of a normally situated testis may be grouped under three main headings, as follows:—

1. **Interference with the Blood Supply**:—

Compression of the spermatic cord, as by an inguinal hernia, a spermatocele, or an ill-fitting truss.
Compression of the testicle by affections of the tunica vaginalis, such as hydrocele or hæmatocele.
Venous stasis, the result of varicocele.
As a sequel of operation in the region of the spermatic cord, such as those for the cure of varicocele, spermatocele, or hernia.
Elephantiasis.

2. **Atrophy, after Orchitis or Epididymitis**, due to such causes as—

Gonorrhœa
Tubercle
Injury
Mumps
X-rays
Typhoid fever
Gout
Syphilis
Influenza (?)

3. **Neurotrophic Causes**, especially after injury to the brain or spine.

It has been stated that the atrophy may result from iodide of potassium; this is difficult to prove, for it seldom happens that this drug is given unless there is already some other possible cause, particularly syphilis or orchitis.

In the differential diagnosis between the above causes the history is in most instances very important.

The cause in any of the cases in Group 1 will generally be obvious. It is only necessary to bear in mind that an operation for varicocele, for instance, may have been performed successfully, and the patient may thereafter contract an orchitis followed by testicular atrophy for which the operation may be blamed unjustly.

As regards Group 2, it is very doubtful whether influenza ever really produced either orchitis or testicular atrophy. There may be a definite history of gonorrhœa, followed by orchitis, which preceded the atrophy, and then diagnosis is easy. It is to be remembered, however, that by no means every orchitis is gonococcal. If mumps, typhoid fever, gout, and injury are borne in mind, these causes of orchitis and testicular atrophy will be recognized more often than they are. Mumps is particularly apt to be overlooked; orchitis may be the sole evidence of this complaint. If the patient is seen when the orchitis is active, bacteriological examination of any urethral discharge is essential to the diagnosis, which depends on whether gonococci are detected or not. If gonorrhœa can be excluded, then the diagnosis of the nature of the orchitis is arrived at by considering the evidence as to gout, mumps, and so on.

It is sometimes stated that orchitis may result from strain, atrophy resulting in due course. There are a few cases in which, apparently as the result of great bodily exertion, especially the lifting of heavy loads, inflammation of the testicle follows; but it is difficult to say that in these cases the strain alone produced the symptoms; there is the possibility that there may have been residual gonorrhœa in the prostate or posterior urethra, the action of the strain being merely to light up the latent inflammation. It is possible that sometimes the latent infection is not gonococcal, but due to other organisms, such as staphylococci or streptococci, whilst recent observers record the bacillus coli communis as the causal organism in some cases of 'spontaneous' orchitis.

There remain a number of cases, however, in which there is no clear history of orchitis, the latter having been relatively slight. Testicular atrophy will then seem to have arisen idiopathically, and it is important to remember how often it is the result of former injury, such as a kick at football, a blow from a cricket ball, contusion from falling astraddle on a fence or bicycle, and so on. The injury may date back to boyhood, many years before testicular atrophy is noticed, and it will often be difficult to prove that the latter was really due to the former.

Apart from obvious tuberculous epididymo-orchitis, transient enlargement of a testis is to be observed, if looked for, in tuberculous subjects; whether this can be regarded as a definite tuberculous orchitis or not, it sometimes results in atrophy.

The *x*-rays are a possible cause of testicular atrophy, and all users of *x*-rays should be careful to have a suitable lead shield. That sterility can result from repeated applications of these rays is well known.

As regards Group 3, the history as a rule gives the diagnosis. Remarkable instances have been recorded in which, within a few months of injury to the brain or spinal cord, particularly after injury to the lumbar vertebræ, or the occipital region of the skull, the glandular elements of the testicle have disappeared. A case of Kocher's exemplifies this: A man, age 41, the father of four children, fell on his head from a considerable height. At first he did not appear to be greatly damaged, but presently twitchings occurred, and the patient became unable to work. From this time on his sexual powers diminished greatly, and his beard and pubic hair fell out. Eighteen months later this hair was gone completely, and about five years after the accident the left testicle was the size of a hazel nut, the right the size of a bean. *Herbert French.*

AURA is the term applied to the immediate prelude of an epileptic seizure. It is recognized in some form or another in about 30 or 40 per cent of epileptics, and with rare exceptions always takes the same shape with every attack in each individual. An aura may be motor, sensory, psychical, visceral, or related to some special sense. A *motor* aura may be represented by an involuntary movement of a limb or a part of a limb; in other cases it takes the form of a general movement such as running. A *sensory* aura is common, and is described as a pain, a numbness, or a tingling in some part of the patient's body. A *psychical* aura is often expressed as a vague apprehension, or an indescribable feeling, or a sense of unreality. A *visceral* aura is frequent, usually as an 'epigastric sensation' or queer feeling starting in the region of the stomach and rising to the throat, or less often as a peremptory desire to go to stool. An aura of special sense may be *olfactory*, *visual*, *auditory*, or *gustatory*; a pleasant or unpleasant odour or flavour may be perceived by the patient, or some alteration in vision may warn him of the onset of a seizure, or he may hear voices or some particular kind of sound.

The aura of epilepsy is, in relation to diagnosis, important from at least two points of view. In the first place, it often affords a clue to the particular locality in the brain from which the 'fit' or 'storm' originates and spreads. This may not be of much value in the case of idiopathic epilepsy, because there is no method at present known to us by which the seat of the disease can be treated successfully. In the case of Jacksonian epilepsy, on the other hand, the knowledge of the locality in which a fit is generated sometimes, although unfortunately not often, allows of benefit being obtained from surgical assistance. For instance, an aura may be the first symptom of the presence of an *intracranial growth.* A tumour of the uncinate region of the temporo-sphenoidal lobe may be revealed by the presence of signs of increased intracranial pressure and the repeated occurrence of an olfactory aura, followed by a vague, dreamy state of consciousness. A lesion of one occipital lobe may be suspected from the occurrence of epileptiform fits immediately preceded by an aura in which there is loss of sight in the opposite visual field. An aura of pain starting in the left foot, spreading up the left side of the body, and terminating in a generalized convulsion, suggests a lesion in the post-Rolandic region of the right parietal lobe. Such instances of the importance of an aura as a localizing sign in diagnosis might easily be multiplied, but a general knowledge of the functional anatomy of the brain will suffice to supply other examples of a similar kind to the reader's mind.

In the second place, the importance of recognizing a subjective sensation as an aura, and so recognizing the existence of epilepsy in its simplest and sometimes earliest form, can hardly be over-estimated from the point of view of treatment. When a patient describes himself as being liable to subjective sensations occurring at intervals, and for which he cannot account, careful inquiry should be made as to their nature. The chief characteristics of an aura are: (1) Its spontaneous development without cause, generally during good health; (2) The suddenness of its onset; and (3) The identity of each sensation with the last. It should be understood clearly that an aura may occur alone, or may be followed by momentary loss of consciousness (petit mal), or by loss of consciousness with convulsions (grand mal). In some cases an aura may be repeated with frequency for many months before a typical epileptic seizure supervenes, and if recognized as such during this stage, it is reasonable to expect that treatment will have more chance of success than at a later period, when the 'habit' of convulsions has been established firmly.

Finally, it should be emphasized that in cases of epilepsy the recurrence of an aura, even without further manifestations of the disease, is evidence that the morbid tendency is not controlled completely, and that discontinuance of treatment will lead to the reappearance of more serious attacks.

E. Farquhar Buzzard.

BABINSKI'S SIGN consists in a modification of the plantar reflex. In testing the latter the patient should be lying upon his back, with his legs very slightly flexed and each foot everted so that its outer border lies comfortably in contact with the bed or couch; the sole should be warm and dry; the ankle should be gently but firmly grasped by one of the observer's hands, to prevent the undue dorsiflexion of the whole foot which often makes it difficult to decide which way the toes themselves move, whilst the outer side of the sole is firmly and steadily stroked from the heel forwards with some such instrument as the butt end of a pencil. In healthy adults the big toe and the other toes will become plantar-flexed; when the great toe becomes dorsiflexed instead, it presents the extensor plantar reflex, or Babinski's sign. Whichever way the other toes move, it is with the direction of movement of the big toe alone that Babinski's sign is concerned. It is noteworthy that if Babinski's sign is present, the fact is usually ascertained with ease; when there is any doubt as to which way the great toe moves, the plantar reflex is seldom really extensor.

The great value of the sign is in distinguishing between functional and organic affections of the nervous system. If the patient is a fully conscious adult with paresis of one or both legs, the existence of an extensor plantar reflex is proof that the lesion is organic. The converse is not true; for with locomotor ataxy, and with lower neuron affections such as infantile paralysis, Tooth's peroneal type of progressive muscular atrophy, peripheral neuritis, Landry's acute ascending paralysis, and primary muscular dystrophies, the plantar reflex is flexor if it is obtainable at all.

Babinski's sign is seen best when there is a lesion in the crossed pyramidal tract. Thus it is present in cases in which tumour, abscess, hæmorrhage, thrombosis, or embolism has caused hemiparesis or hemiplegia by affecting either the pyramidal cells themselves in the motor cortex or the pyramidal fibres in the internal capsule; in cases of cerebellar tumour, owing to the fact that this, by compressing the medulla, nearly always causes lateral sclerosis of the cord as well; and in cases of disseminated sclerosis, transverse myelitis, either primary or due to compression, ataxic paraplegia, Friedreich's ataxy, amyotrophic lateral sclerosis, primary lateral sclerosis, some cases of syringomyelia, and in those cases of irregular sclerosis of the cord that may be associated with severe oligocythæmias such as pernicious anæmia. The differential diagnosis of these conditions will be found under HEMIPLEGIA (p. 302) and PARAPLEGIA (p. 510) and elsewhere. Babinski's sign is not found in those cases of hysteria that sometimes simulate one or other of the above conditions; provided always that the patient is a conscious adult. This proviso is important, because the plantar reflex may be extensor without there being any decided changes in the cord or brain in infants and quite young children; also in a considerable proportion of older children suffering from *chorea*; and also sometimes in adults during deep sleep, or under conditions of unnatural unconsciousness such as that due to a general anæsthetic, or acute alcoholic intoxication, or such affections as epilepsy, uræmia, concussion, saturnine encephalopathy, and in some other forms of coma. These exceptions, however, scarcely detract from the great value the sign has as a means of distinguishing between organic and functional paralysis of the legs of the upper neuron type. *Herbert French.*

BACILLURIA.—(See BACTERIURIA, *infra.*)

BACTERIURIA (see *Plate XXVIII*, p. 614) is a comprehensive term employed to indicate that the urine when freshly voided contains micro-organisms. *Bacilluria* is a term of similar import, but is restricted to those cases in which rod-shaped bacteria are present. The vaginal segment of the female urethra and the anterior portion of the male urethra are normally inhabited by certain non-pathogenic bacteria (chiefly cocci, such as *Streptococcus brevis*, *Staphylococcus albus*, also varieties of *Bacillus xerosis*, etc.), which are, of course, present in urine obtained under ordinary conditions, and so constitute what may be termed physiological bacteriuria. Bacteriuria as a pathological condition due to some lesion of the urinary system posterior to the urethra can only be recognized with certainty by the examination in the laboratory of a catheter specimen of the urine collected with the most scrupulous attention to asepsis; for, on the one hand, a perfectly clear acid urine may be heavily loaded with bacteria, and, on the other, a urine may owe its turbidity either to purely physico-chemical causes, or to the growth in it of bacteria which have gained access after its exit from the urethra. Moreover, although the identity of the infecting organism may be suspected from general clinical considerations, cultivation experiments are essential in order to settle the matter beyond doubt.

Bacteriuria may be persistent and may indicate either general or local infection. It is a rare symptom of general infection, save one of such intensity that an acute nephritis, associated with a definite hæmaturia, has supervened. Usually its appearance indicates a local infection of the urinary tract; it then occurs with greatest frequency in young children and pregnant women, when the micro-organism concerned is usually *B. coli*, and the site of the infection the pelvis of the right kidney. It is, however, met with at all ages and in both sexes, and many different bacteria have been recorded as the causative factors, and whilst the infection is commonly due to some particular micro-organism, the possibility of multiple infection must not be forgotten—the most usual being a double infection due to *B. coli communis* and *Streptococcus pyogenes longus*.

When intermittent, bacteriuria may indicate a general infection, or a local infection of some area distant from the urinary tract, as, for example, a tonsillitis or a dental abscess, and often in an obscure case of pyrexia a bacteriological examination of the urine will well repay the trouble involved. Intermittent bacteriuria, particularly of the staphylococcic type, is often associated with kidney calculus, and it is also not uncommon in cases of rheumatoid arthritis.

Bacteriuria may be a symptom in:—

A. **General Infections,** with or without associated nephritis, due to:—

Streptococcus pyogenes longus
Pneumococcus
Gonococcus
Staphylococcus pyogenes aureus
B. typhosus
B. coli communis
B. paratyphosus
Micrococcus melitensis

B **Local Infections:**—

Nephritis, pyelonephritis, or ureteritis due to:—

B. coli
B. tuberculosis
B. pyocyaneus
B. pneumoniæ (Friedländer's bacillus)
Streptococcus pyogenes longus
Staphylococcus pyogenes aureus
Pneumococcus

Cystitis due to :—

B. coli
B. tuberculosis
B. typhosus
Streptococcus pyogenes longus
Staphylococcus pyogenes aureus

Prostatitis due to :—

B. coli
Gonococcus
Staphylococcus pyogenes aureus
Streptococcus pyogenes longus

Urethritis due to :—

Gonococcus
Staphylococcus aureus or albus
Pneumococcus
Streptococcus pyogenes longus
Micrococcus catarrhalis

In the above table the various micro-organisms are, speaking generally, arranged in the order of their frequency.

Finally, a slight and transitory bacteriuria due to *B. coli communis*, and one usually passing off without any treatment, can frequently be observed following operative measures upon the rectum or anus, or the organs of generation.

In general infections the urine is either normal in appearance, or by reason of its admixture with blood may present any tint from 'smoky' to bright red. The reaction is usually acid; often a degree of acidity is recorded which if present in an artificial culture medium would inhibit the growth of the infecting micro-organism. Albumin is present, varying in amount from a trace to 7, 8, or more parts per thousand, and microscopical examination of the centrifugalized deposit shows blood-cells, renal tube-casts, and renal epithelium, in addition to the infecting bacterium. The clinical symptoms presented by the patient are those of the general systemic infection.

In local infections of the genito-urinary tract where infection is due to one species of micro-organism only, the urine presents a somewhat similar appearance; blood, however, may be entirely absent, while pus when measured by the centrifuge may vary in volume from a trace to 10 or 20 per cent of the total bulk of urine. In the early stages of a local infection, however, microscopical examination of the deposit may merely show the presence of leucocytes slightly in excess of normal, so that without the use of the microscope the fact of pyuria may easily be missed altogether.

Occasionally, and particularly in adult cases, it may be noted that the urine passed during the day is neutral or faintly alkaline—the change in reaction then being due to physiological causes. In those cases where the urine is strongly alkaline the alkalinity is due to ammonia resulting from the decomposition of urea, not by the pathogenic infecting organism but by non-pathogenic saprophytes which have gained access to the urine, either after it has been voided or whilst still *intra vesicam*. In the latter instance the contamination may have taken place as a result of careless instrumentation, or (as in the female) by continuity of surface, but it also frequently occurs owing to the passage of micro-organisms through the inflamed bladder wall from the lumen of the adjacent large intestine.

The clinical symptoms associated with bacteriuria due to local infection vary enormously with different patients. Frequency of micturition, scalding, dull aching pains in one or both loins, with tenderness on deep pressure over the kidney or ureters, pains in the perineum and hypogastrium (according to the situation of the primary infection), severe rigors, pyrexia (*Fig.* 193, p. 456), anorexia, nausea, and vomiting are amongst those commonly observed. It is important to remember its relatively common occurrence in children, in whom there may be hardly any symptoms at all, or perhaps general delicacy or ill-health, or gastro-intestinal disturbance, without any special urinary symptoms attracting notice. The urine generally contains only a trace of albumin, and no obvious pus; the diagnosis then depends upon bacteriological investigation of a catheter specimen, the need for which will be suggested by the discovery of a decided excess of leucocytes in the centrifugalized deposit from the specimen first collected during the routine examination of the patient.

Jno. Eyre.

BALDNESS.—Alopecia, or baldness, may vary in degree from slight thinning to complete loss of the hair. There are three main varieties of simple baldness or alopecia, namely: (1) *Congenital*, (2) *Senile*, and (3) *Premature*.

Congenital Alopecia is seldom complete, and the hair may be lanugo-like. In the latter case the diagnosis is certain, as it also is when the baldness is accompanied by developmental defects in the skin or its appendages. When there is complete absence of the hair, not only of the head but also of the eyelids, face, trunk, armpits, and pubic regions, the diagnosis is obvious.

Senile Alopecia needs no description.

Premature Alopecia may be (*a*) idiopathic or (*b*) symptomatic. The former, much less frequent than the latter, and due to no recognizable cause except heredity, usually begins between the ages of twenty and thirty-five ; in many cases at the vertex, like senile baldness, but often at the temple, when it extends backwards elliptically. Symptomatic premature baldness may be either temporary or permanent, gradual or rapid, and is dependent upon a great variety of local or constitutional causes, including *seborrhœa* of the scalp, *psoriasis*, *chronic eczema*, *erysipelas*, *ringworm*, *favus*. *lupus*, *erythematosus*, *syphilis*; it is also a sequela of *fevers* or other acute systemic diseases, and sometimes of a severe shock to the nervous system such as may result from a sudden and unexpected bereavement or the like. When it occurs as a sequel to fevers, in syphilis, ringworm (except after severe kerion), erysipelas, and eczema, the loss of hair is usually but temporary ; in seborrhœa, favus, lupus erythematosus, morphœa, and *folliculitis decalvans*, it is generally permanent ; it is always so when the hair-follicles have been destroyed.

The most important form of symptomatic baldness is that which is associated with *seborrhœa*, whether of the oily or of the dry kind. Seborrhœic alopecia has the same distribution as idiopathic baldness. Another form of symptomatic baldness is the condition known as *alopecia areata*, in which the hair falls out in more or less circular smooth white patches, generally of irregular distribution. Usually the patches continue to spread for a time, and may run into others, denuded areas of irregular outline thus being formed, with a surface white and smooth as a billiard ball. The hairs at the edges of the patches are looser than the others, and among them may be seen short stumps that have atrophied close to the root, so that they resemble a note of exclamation (!). In rare cases the hair falls out not in patches but more generally and very rapidly ; and soon the whole scalp may be bared, and even the hair of the whole body may be lost, and with it the nails of the fingers and toes. The affection with which alopecia areata is most easily confounded is *ringworm* of the trichophytic variety : the differential diagnosis between the two affections will be found under FUNGOUS AFFECTIONS OF THE SKIN (p. 246). Alopecia areata may also be confused with another form of symptomatic baldness, namely, *alopecia cicatrisata*, the *pseudo-pelade* of Brocq, in which depressed islands of baldness, round or of irregular shape, occur on the scalp, the patches usually spreading and coalescing into large, smooth, shiny areas ; these are cicatricial ; there is destruction of the follicles so that the hair is never restored ; there are normal-looking hairs on the bald areas, and the note-of-exclamation stumps of alopecia areata are absent. The bald patches sometimes met with in *secondary syphilis* may be distinguished from those of alopecia areata by the co-existence of other syphilitic symptoms, by the positive Wassermann's serum reaction, and by the effects of specific treatment. The bald areas of *lupus erythematosus* are in greater or less degree cicatricial, there is destruction of the follicles, and a border which is slightly or distinctly inflamed. *Folliculitis decalvans* is cicatricial also, and at the edge of the bare patches a small red papule or patch of erythema can be seen surrounding each follicle.

Malcolm Morris.

BEARING-DOWN PAIN.—(See PAIN, BEARING-DOWN, p. 426.)

BLACK SPECKS BEFORE THE EYES are of two types : (1) *Moving*, (2) *Fixed*.

Moving Black Specks are practically always due to *muscæ volitantes*. The aqueous and vitreous humours are not absolutely homogeneous ; in both there are minute particles in most persons, and these throw shadows upon the retina which are referred by the patient to points in the visual field outside him. They seem to be in front of his eyes, interfering with what he wishes to look at ; yet when he tries to locate them definitely by looking directly at them, they immediately float away, as it were, from his direct field of vision to a peripheral part. He can never focus them, and yet he may be conscious of seeing them all the time. Only few persons in perfect health are troubled in this way, for although the muscæ volitantes may be present all the time, the mind neglects them and fails to notice them. When the eye is tired by close work, however, or the patient is suffering from brain-fag, worry, insomnia, biliousness or other similar condition, they may attract his notice very much and make him fear that he is developing some serious lesion such as a cataract. Microscopists often find them a great nuisance. After a rest or a holiday they will cease to obtrude themselves upon the patient's notice, but he will notice them again when he gets overworked or run down. In a similar way muscæ volitantes may be troublesome in those who are suffering ill health due to almost any organic cause, especially if it is associated with Anæmia (p. 20). The way in which the specks float away when an attempt is made to focus them is characteristic.

Fixed Black Specks.—When, on the other hand, the patient notices a black spot or spots in his field of vision, always present and always in exactly the same relationship to the point upon which he is focussing his eye—not floating away into different parts of the field of vision like muscæ volitantes—a careful examination of the eye with the ophthalmoscope, assisted perhaps by the perimeter to map out the abnormal blind spot with accuracy, will generally reveal some organic lesion in the eye to account for them. An opacity in the cornea from old *keratitis*, or synechiæ from adhesions due to old *iritis*, or a *cataract*, may be seen, or tiny white patches at the macula indicative of incipient *albuminuric retinitis* of grave omen ; or a small *detachment of the retina* ; or a *melanotic sarcoma* of the eyeball ; or *early optic neuritis* ; or a *thrombosed retinal vein* ; or an *embolized* branch of a *retinal artery* ; or a *hæmorrhage* into the vitreous ; or a scotoma from localized *optic atrophy*, such as is met with sometimes in cases of *disseminated sclerosis*. Special ophthalmic experience will be needed to diagnose between these different conditions, although the ophthalmoscopic appearances (p. 415) of some of them are pathognomonic.

Herbert French.

BLEEDING GUMS.—A spongy, bleeding condition of the gums, attaining such a degree that the teeth become covered by the exuberant blood-oozing tissues, was a prominent feature of *scurvy*, a serious and often fatal disease which used to be common on sailing ships when fresh food was necessarily absent from the diet for weeks or even months at a time. It is now rare in its full development, but is still found in a mild form amongst children—infantile scurvy, or Barlow's disease—as the result of long-continued feeding with tinned milk without fresh food. Its chief features are anæmia and tenderness of the long bones due to hæmorrhages under the periosteum ; in severer cases, besides sponginess and bleeding of the gum with more or less general stomatitis, there may be purpura and other hæmorrhages. The diagnosis is suggested by the diet history, and confirmed by the benefit that follows the addition of fresh milk and, in older children, fresh vegetables. A similar condition may arise in adults whose circumstances compel them to live on tinned foods. There are, however, many other causes of bleeding of the gums besides scurvy. The differential diagnosis is generally easy, but sometimes very difficult. The first point to determine is whether the gum condition is due to local changes only, or whether it is part of a more general condition.

(*A*). **Bleeding Gums due to General Conditions or preceded by Lesions elsewhere than in the Mouth :—**

Scurvy
Splenomedullary leukæmia
Lymphatic leukæmia
Hodgkin's disease
Pernicious anæmia
Aplastic anæmia
Splenic anæmia
Hæmophilia
Purpura (see Purpura, p. 552)
Syphilis
Mercurialism
Iodide poisoning
Phosphorus poisoning
Arsenic poisoning
Lead poisoning
Febrile or asthenic states accompanied by sordes, e.g., pneumonia, typhoid fever, the later stages of malignant cachexia, general paralysis, acute yellow atrophy of the liver, and so forth
Dyspepsia

(*B*). **Bleeding Gums due to purely Local Conditions :—**

Injury, e.g., by tooth brush
Dental caries
Tartar
Pyorrhœa alveolaris
Alveolar abscess
Papilloma
Epulis
Myeloid sarcoma
Epithelioma
Actinomycosis
Acute or chronic stomatitis not obviously due to any of the causes already mentioned, e.g. :
  Aphthous stomatitis
  Ulcerative stomatitis
Gangrenous stomatitis (cancrum oris, phagedæna oris, noma oris)
Tuberculous gingivitis
Erythema bullosum, dermatitis herpetiformis, pemphigus, affecting the mouth as well as the epidermis

A. **Bleeding Gums due to General Conditions.**—Many of the above conditions are discussed under other and more prominent symptoms, so that here we need refer to them but briefly (see Spleen, Enlargement of, p. 628 ; Anæmia, p. 20 ; Purpura, p. 552 ; etc.).

A blood-count is required to diagnose or exclude *leukæmia* or *pernicious anæmia.* The family history may suggest *hæmophilia. Splenic anæmia, Hodgkin's disease,* and *aplastic anæmia* attract attention more on account of the enlargement of the spleen (p. 628), or of the lymphatic glands (p. 376), or of the anæmia (p. 20), than because of spongy gums. *Purpura* (p. 552) is itself a symptom and not a disease.

Syphilis, particularly in its secondary stage, may produce stomatitis, pharyngitis, laryngitis, and gingivitis, with bleeding, even when no mercurial treatment has been adopted; the secondary roseola may still be present, or the history may be obvious. Difficulty arises mainly in women and children, and when the chancre has been extragenital (*Fig.* 23). Wassermann's serum test may be tried, or the *Spirochæta pallida* (*Plate XXVIII, Fig. J,* p. 614) looked for in scrapings from the mucous lesions.

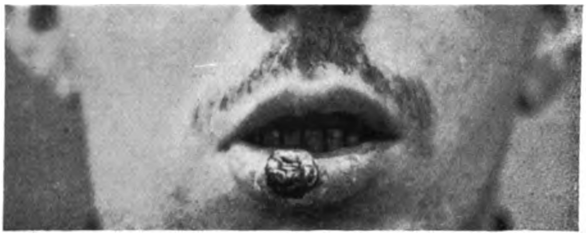


Fig. 23.—Primary syphilitic sore on the lower lip.

Mercury is very liable to cause profuse salivation and acute stomatitis, with distressing and painful swelling of lips, gums, tongue, and cheeks; swallowing may become impossible, the glairy saliva hangs in strings from the protruding tongue and bulging lips, the mucosa bleeds on the slightest touch, and the patient is the picture of abject misery. Some persons are far more intolerant of mercury than others, but its worst effects have occurred when the remedy has been employed when the teeth are carious, or the mouth unclean, and when there is albuminuria (syphilitic nephritis). The diagnosis depends upon a knowledge of the drugs that are being given or, in occupation cases, of the chemicals that the patient has been working with.

Iodides may cause profuse coryza, due to conjunctival, nasal, and oral catarrh, but the amount of bleeding that accompanies it is slight. The nature of the drugs being taken will suggest the diagnosis, or if there is doubt as to the drugs, the urine may be tested for iodides.

Phosphorus used to produce very severe stomatitis, going on to necrosis of the jaw—'phossy jaw'—not infrequently ending in death as the result of fatty degeneration of the liver and heart; this is uncommon since restrictions have been laid upon the use of crude yellow phosphorus in the manufacture of matches. The occupation generally serves to suggest the diagnosis.

Arsenic and *lead* are both rare causes of bleeding gums; occupation, or medical prescription, or habits as regards drinking, may suggest the diagnosis, and there may be other signs of the poisoning, particularly pigmentation of the skin, vomiting, diarrhœa, hyperkeratosis of the soles and palms, and generalized peripheral neuritis in the case of arsenic; and the symptoms given elsewhere (p. 34) in the case of lead. Arsenic may be found in excess in the hair, or lead may be detected in the fæces or in the residue from a bulk of urine.

Febrile and *asthenic states* only cause sordes and bleeding gums when the patient has already been ill some while, or when the nursing has been remiss; the diagnosis will depend on symptoms other than those connected with the gums.

B. **Bleeding Gums due to Local Conditions.**—When care has been taken to exclude general causes of bleeding of the gums, differentiation between the various local causes is not difficult. Some patients are alarmed by the symptom, when its cause is nothing more than the use of a *new tooth-brush* whose bristles have slightly lacerated gums that are accustomed to an older and softer brush. The history will indicate other forms of local injury—an ill-fitting tooth-plate, perhaps. Hæmoptysis may be simulated.

Dental caries may be obvious, or it may be hidden away between adjacent teeth and yet be irritating the gum enough to cause it to bleed with undue readiness when the teeth are brushed. *Tartar* is obvious on inspection. *Pyorrhœa alveolaris,* also known as *suppurative gingivitis* or *Rigg's disease,* is the result of septic infection extending down into the sockets, loosening the teeth, causing the gum margins to recede by erosion, and leading to a purulent discharge from between the gums and the teeth. This condition may be present even when the external aspect of the teeth seems perfect; a very fine probe may sometimes be passed painlessly down into the tooth-socket between adjacent teeth where the suppurative process has been progressing unsuspected, and out of the reach of the tooth-brush. The gums bleed on the slightest touch in severe cases, the breath is foul, and the constant swallowing of pyogenic organisms and their products leads to dyspepsia, anæmia, chronic ill health, listlessness, functional nerve disorders, and sometimes more acute symptoms of general pyæmia, especially multiple infective synovitis and arthritis. Neurasthenia and depression ultimately ensue in many cases, and sometimes very severe and even fatal anæmia or purpura.

The diagnosis of *alveolar abscess* is generally obvious, though infection of a *benign* or *malignant new growth* may simulate it for a time. Microscope examination of the excised tumour is the only certain way of diagnosing the nature of an odontoma, papilloma, simple epulis, myeloid sarcomatous epulis, or epithelioma of the gum.

Actinomycosis is rare in man; but the jaw, gum, or cheek are parts least uncommonly affected. The chronic nature of that which partakes of the characters partly of a neoplasm and partly of an abscess, in a person who has had occasion to put straws, cotton, or other vegetable products into his mouth, may suggest the diagnosis, which will be confirmed by the finding of the ray fungi in the purulent discharge, or in sections from parts excised. Minute grey or yellowish specks in the pus are said to be characteristic, but they are not always seen, and it is by microscopical examination that the diagnosis is made with certainty (see *Plate XXVIII, Fig. S*, p. 614).

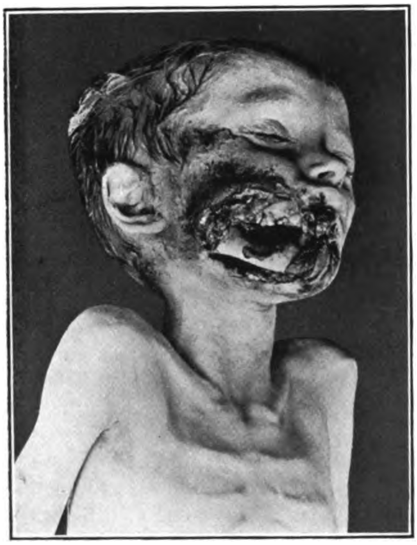


Fig. 24.—Cancrum oris.

Stomatitis in its various degrees may have a general cause, such as mercurialism (see above); or it may be due to purely local infection with micro-organisms. It might perhaps be classified bacteriologically—the variety spoken of as thrush being due to the *oidium albicans*, for instance. Clinically, however, it is more often classified by its degree—into acute catarrhal, ulcerative, and gangrenous. All these affect the mucosa of cheeks, lips, tongue, and palate, in addition to the gums, and any of the inflamed parts bleed readily. The first degree is characterized by redness, swelling, tenderness, and pain, with inability to move the tongue about in order to eat and swallow, swelling and protrusion of the lips, foulness of the breath, and very often salivation. There may or may not be localized greyish or white aphthous patches; these are commoner in children. When ulcers occur, these are generally multiple and shallow, very painful, with more or less glazing of the ulcerated surface, and acute hyperæmia of the margins. The gangrenous form is better known as *cancrum oris* (*Fig.* 24), fortunately rare, though sometimes seen in ill-cared-for children who have contracted measles or some other acute debilitating fever. The cheek is affected first, a dusky-red or black spot appearing within and without, spreading rapidly and leading to sloughing and perforation of the cheek, gangrene of the gums and jaw, falling out of the teeth, a very foul nauseating odour of the breath, and death from utter exhaustion. The diagnosis is generally obvious.

Tuberculous gingivitis is rare, but when it does occur it is very severe. The nature of the bleeding gums will be suggested by the co-existence of phthisis, and tubercle bacilli may abound in smears from the gum.

Erythema bullosum, dermatitis herpetiformis, and *pemphigus*—particularly the first—may affect mucous membranes as well as the skin, especially the mouth, colon, and vagina. The result as regards the mouth is very distressing; the crusts and resultant inflammation of lips, gums, tongue, cheeks, palate, fauces, and pharynx, may make it impossible for food to be taken orally, and the patient loses weight rapidly and becomes very ill. The mucous membrane everywhere bleeds on the slightest touch, and the condition is pitiable. There is generally pyrexia. The diagnosis is, as a rule, easy, for the mucous membranes are seldom attacked unless the skin is affected also (see BULLÆ, p. 86, and EOSINOPHILIA, p. 218).

Herbert French.

BLEEDING NOSE.—(See EPISTAXIS, p. 220.)

BLEEDING, UTERINE.—(See MENORRHAGIA, p. 385; METRORRHAGIA, p. 390; and METROSTAXIS, p. 392.)

BLINDNESS.—(See VISION, DEFECTS OF, p. 757.)

BLISTERS.—(See BULLÆ, p. 96.)

BLOOD, COUGHING UP OF.—(See HÆMOPTYSIS, p. 285.)

BLOOD IN THE URINE.—(See HÆMATURIA, p. 275.)

BLOOD PER ANUM.—Blood may be passed per anum whenever bleeding takes place from any part of the alimentary canal. If it comes from a point high up, as from the stomach or duodenum, it is usually altered in appearance, so that black, tarry stools are passed (melæna); if it comes from the colon or from the lower end of the ileum, it is passed as red blood, easily recognizable as such. If the quantity is very large it may be bright red even in the case of lesions high up; the colour depends on the rapidity of passage through the bowel and the consequent extent to which the digestive juices have acted upon it.

Recognition of the actual presence of blood, pure or mixed with the motions, is not often difficult, except when the quantity is small. The typical tarry stools of hæmorrhage high up in the alimentary tract are unlike anything else. The black colour is much more pronounced than the pigmentation of the stools caused by iron or bismuth sulphide, which produce rather a slaty or dirty greyish-black tint; while the viscid consistency of the hæmorrhagic stool is also characteristic. Administration of charcoal by the mouth may produce deep black stools, and eating bilberries is also said to do so. In case of doubt, the chemical and spectroscopical tests for blood may be applied; for which purpose it is best to acidulate the fæces strongly with acetic acid and to extract the acid mixture with ether; a clear solution of blood-pigment is thus obtained, suitable for the spectroscope or for the guaiacum test. In some cases blood corpuscles may be recognizable under the microscope if a portion of the fæces is rubbed up with physiological saline solution. Grains of charcoal will be distinguishable under the microscope if this substance has been taken.

The conditions associated with the passage of blood per anum may be divided conveniently for diagnostic purpose into: (1) *Those in which large quantities of altered blood are passed* (true melæna); (2) *Those in which large quantities of red or unaltered blood are voided;* (3) *Those in which small amounts of such blood are seen;* and (4) *Cases of so-called occult hæmorrhage*, only recognizable by chemical or other special tests. The conditions classed under headings (2) and (3) necessarily overlap, inasmuch as the exact quantity of blood discharged is very variable; the former comprise, roughly speaking, affections of the bowel; the latter, lesions about the rectum and anus.

Large quantities of altered blood may escape in cases of ulceration of the stomach or duodenum. It is usually mixed with acid gastric juice, and thus blackened. Such cases are generally associated with pain after meals, vomiting, hæmatemesis, and increased acidity of the gastric juice. Tenderness will be elicited on pressure over the epigastrium, most often at a point rather to the right of the middle line and about four inches below the xiphisternal junction in the pyloric region. Distinction between lesions of the stomach and of the duodenum is difficult; but in gastric ulceration the pain usually arises within an hour after meals, and is relieved by vomiting; in duodenal ulcer, it often reaches its acme about three or four hours after a meal, and it may at first be relieved by taking food ('hunger pain'). In gastric ulcer, the greater part of the blood which escapes is likely to be vomited; in duodenal, most of it to be passed per anum. Duodenal ulceration is most common in men. The symptoms of gastric ulcer are much more common in women; but it has been shown that in many such instances no actual ulcer can be found, the blood escaping apparently by a process of oozing through the mucous membrane—a condition referred to as gastrostaxis. Evidence obtained from post-mortem findings shows the two sexes to be about equally liable to this affection.

Large quantities of unaltered or but slightly altered blood may be passed in cases of ulceration of the small intestine, as in enteric fever, tuberculosis, or the peculiar lesions associated with chronic interstitial nephritis. The phenomena of *enteric fever* need not be detailed at length—initial headache, epistaxis, and fever ; fullness of the abdomen and possibly diarrhœa, rose spots, enlargement of the spleen, mental dullness or delirium ; leucopenia, and Widal's agglutinative reaction in the blood. *Tuberculous ulceration* of the intestine seldom, if ever, occurs apart from tuberculosis of the lungs, and it is a rare cause of profuse intestinal hæmorrhage. It is associated with pain and tenderness in the abdomen, and with emaciation and signs of pulmonary disease. Tubercle bacilli may be found on examination of the fæces.

Chronic Bright's disease may be associated with hæmorrhage from the bowel as from other parts of the body. The absence of other causes, such as ulceration ; the existence of high blood-pressure and enlargement of the left ventricle, the cardiac impulse being displaced outwards and downwards ; and the constant or occasional appearance of albumin and renal tube casts in the urine, with weakness, anæmia, and perhaps epistaxis, will point to this cause.

Bleeding into the pancreas and *embolism* or *thrombosis of one of the mesenteric vessels* may both lead to moderate hæmorrhage from the bowel. In both alike there will be symptoms of sudden abdominal pain and constipation with collapse, closely resembling the phenomena of intestinal obstruction. A certain diagnosis can hardly be made without laparotomy. Patients who suffer from pancreatic apoplexy are usually fat. Blocking of a mesenteric vessel by embolism is most likely to occur in sufferers from some form of cardiac disease, especially malignant or ulcerative endocarditis (p. 34).

In the peculiar condition known as *Henoch's purpura* (p. 556) there occur attacks of colic, constipation and vomiting, with passage of blood per anum. The symptoms may closely simulate intestinal obstruction or intussusception, and may be indistinguishable from mesenteric embolism. A diagnosis may sometimes be made when other phenomena of bleeding are present, such as hæmatemesis, hæmaturia, petechiæ in the skin, or epistaxis, or by concomitant affections of joints (hæmorrhagic arthritis): the patient is generally young ; a history of previous attacks may also be obtained.

A good deal of blood may be passed per anum in some cases of *general hæmorrhagic conditions,* such as profound anæmia, leukæmia, and purpura hæmorrhagica. The general appearance of the patient, and examination of the blood (p. 24) will suffice to distinguish the two former ; and in the last there will probably be visible hæmorrhages in the skin and bleeding from other mucous surfaces.

The possibility of the *rupture of an aneurysm* into the stomach or bowel may be mentioned for the sake of completeness ; a diagnosis can only be made by recognition of the pulsating aneurysmal swelling, and the condition will probably be rapidly fatal.

In infants, considerable quantities of blood may be passed per anum owing to *septic infection of the umbilical cord,* the hæmorrhage arising either from an actual ulcer of the stomach or duodenum, or from a purpuric condition caused by bacterial toxæmia ; in a few such cases running a rapidly fatal course the passage of dark or bright blood per rectum in increasing quantities is almost the only symptom, and the cause of the bleeding is not clear even when searched for at autopsy ; the fatal symptom may develop within a day or two of birth (Melæna neonatorum).

Hæmorrhage of moderate degree is usually associated with disease of the large intestine, though occasionally profuse bleeding may occur in such affections. The blood is bright in colour and generally mixed with mucus. In *tropical dysentery* there is severe tenesmus and great frequency of defæcation, only blood-stained mucus in small quantities being passed when the disease is well established. In *ulcerative colitis,* which appears to be a bacillary dysentery of temperate climates, there are the same diarrhœa, frequency of defæcation, and wasting as characterize the tropical malady, but tenesmus is less marked and the stools are usually more fæcal. Some cases of ulcerative colitis closely simulate enteric fever ; they may be distinguished by the absence of Widal's reaction, and by recognition of the ulcers in the lower part of the large bowel by means of the sigmoidoscope.

Examination of the stools in cases of tropical dysentery may reveal the presence of the *Amœba histolytica* (*Fig.* 25). This large organism measures some 30 to 40 μ in diameter, and is distinguished from the harmless *Amœba coli* by its well-developed clear outer layer of ectoplasm, by its small and eccentrically placed nucleus, and by the presence of ingested blood-corpuscles within its substance.

In the search for amœbæ a flake of mucus should be spread out as thinly as possible on a slide, and if the organisms are very scanty, the addition of a drop of 1 per cent watery methylene blue is of assistance, as it stains the pus and epithelial cells at once, whilst for a time the amœbæ resist taking up the stain and also retain their activity : they thus stand out clearly amid their blue surroundings as light refractile motile bodies. In such a preparation examined directly after it is made, it is possible to detect them with a very low power, such as a Zeiss A or a half-inch lens, a higher power being turned on to verify the find. With some practice they may also be seen in unstained mucus with the low power as small glistening particles, the condenser being fully lowered for this method of examination, and any likely object being scrutinized further by a $\frac{1}{6}$ inch lens. Full doses of ipecacuanha or emetine should not be given before the stools are examined microscopically, or a negative result is likely to be obtained in amœbic cases, just as in malaria after quinine has been taken. The stools should always be examined as fresh as possible, preferably within an hour of being passed. In cool climates the specimen may be kept at blood-heat for a short time and the slide warmed. The organisms should always be seen in active motion before a positive diagnosis is made, for there are often large mucoid cells present, especially in bacillary dysentery, which may easily be mistaken by the inexperienced for inactive amœbæ.

In bacillary dysentery the pathogenic organisms belong to a group of closely allied bacteria classed under the title *B. dysenteriæ.* They are short, rod-shaped bacteria, with rounded ends, somewhat resembling *B. typhosus,* but non-motile. These bacilli grow on ordinary laboratory media, do not coagulate milk, and do not form indol. They are not stained by Gram's method. The exact bacteriology of ulcerative colitis is undetermined, but organisms resembling *B. dysenteriæ* have been isolated by some observers.

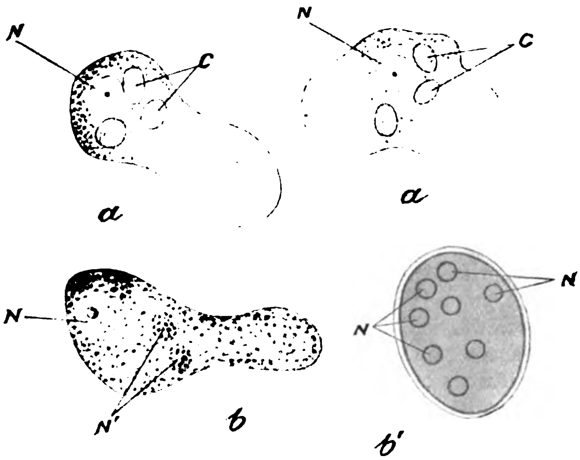


Fig. 25.—(*aa*) *Amœba histolytica* (after Jürgens) ; (*b*) *Amœba coli*, motile form ; (*b′*) *Amœba coli*, encysted (after Casagrandi and Barbagallo). (N) Nucleus ; (N′) Nuclei after division ; (C) Blood corpuscles. (*High power.*)

Malignant disease of the intestine may give rise to some degree of hæmorrhage. In a typical case of cancer of the large bowel, an elderly person has suffered from gradually increasing weakness, wasting, and constipation. Attacks of colicky pain may supervene, and some enlargement of the abdomen may be noticed. Blood may be present in the motions from time to time, but is not often a marked feature. Examination of the abdomen may reveal vermicular movements of the hypertrophied bowel, which tend to pass in a definite direction along the course of the colon, and to cease at a particular point. Here a definite tumour may be palpable ; but as the flexures of the colon are favourite seats for neoplasms, it often happens that the growth is situated deeply in the pelvis or beneath the lower ribs, and cannot be felt. There is little or no fever unless there are extensive secondary deposits, especially in the liver, which may become greatly enlarged. Acute intestinal obstruction may finally occur. The diagnosis is often assisted either by the sigmoidoscope or by the use of x-rays after a bismuth meal (p. 125).

As contrasted with the above, *non-malignant ulceration of the colon* is likely to have a more marked onset, with pain, frequency of defæcation, and loose motions. The stools often contain considerable quantities of blood mixed with mucus. The body temperature is raised, often to a high degree (103° F.), pain is more constant, and tenderness may be elicited all along the course of the large intestine. Often the ulceration extends into the sigmoid flexure of the colon, and may be visible on examination with the sigmoidoscope.

In *intussusception*, blood and mucus are passed without fæcal matter accompanying them. The condition is commonest in infants and young children. There are usually symptoms of severe illness, with screaming, drawing up of the legs, frequent pulse, and some collapse ; rarely the condition may be encountered with but few grave signs. A rectal examination is essential, as in many cases the intussusceptum may be felt with the finger ; a careful palpation of the abdomen will usually reveal an elongated tumour, which may sometimes be felt to harden and relax again with the peristalsis of the gut.

In infants, *simple colitis* may give rise to the appearance of blood and mucus in the motions, but there is generally some fæcal material passed at the same time, which is not the case in intussusception after the contents of the colon below the intussusception have been evacuated. In simple colitis the motions are frequent and loose, and they may contain mucus. In milder cases they may be green and slimy, but in the more severe they are brownish and very offensive, and in the worst cases consist of little more than a dirty serous discharge. The child's temperature will probably be raised ; the pulse is frequent, and there may be vomiting. A collapsed condition may occur at a late period of this malady—rarely, in acute choleraic cases, it may ensue within the first twenty-four hours. In intussusception, on the other hand, collapse usually occurs quickly ; and there is absolute constipation, with passage only of a small amount of blood-stained mucus. The only cases which can give rise to a difficulty of diagnosis are the rare instances in which intussusception is present without severe symptoms ; and here rectal and abdominal examination will probably reveal the true condition of affairs. By means of rectal examination in an infant a considerable area of the abdomen can be investigated, especially if an anæsthetic be administered. In all cases of doubt in intestinal affections accompanied by bleeding this procedure is urgently demanded.

The intense diarrhœa accompanying *arsenical poisoning* may be accompanied by the passage of traces of blood and mucus. The condition will be distinguished by its rapid onset, some half-hour or so after a meal, by the epigastric pain, tenderness, and vomiting, followed by collapse, with rapid irregular pulse, and clammy skin. A chemical examination of the vomited matters should be made in suspected cases, by Reinsch's or Marsh's test.

Traces of blood smeared over the motions are suggestive of *piles*, which may be seen on inspection if external, and felt by the examining finger if internal to the sphincter. Occasionally a sharp attack of bleeding may occur from this cause if a varix be ruptured. The condition is usually accompanied by a sense of fullness, weight, and even pain in the rectum, and the patient may be conscious of " something coming down " and having to be replaced after defæcation.

Some amount of blood may arise from an *anal fistula*, which may also lead to a discharge of mucus and of pus. Inspection and digital examination will discover this affection, the external opening of the fistula being close to the margin of the anus, the internal often just above the border of the sphincter.

Cancer of the rectum does not usually give rise to much hæmorrhage, but traces of blood may be passed from time to time, and sometimes a sanious discharge occurs. The main symptoms are usually wasting and cachexia ; gradually increasing difficulty in defæcation ; and rarely, alteration in the size and shape of the fæcal masses, which may be thin or ribbon-like. Sometimes alternating periods of diarrhœa and constipation occur ; or there may be morning diarrhœa, the matter passed being thin fluid. Pain in the sacral region generally occurs at some period of the disease, and it may radiate down the thighs. The growth may be seen by means of the speculum or sigmoidoscope, and also felt by the examining finger.

Rectal polypi are common in children, and may rarely be encountered in adults. They give rise to frequent bleeding, which may occasionally be considerable in amount. The patient may be conscious of something present in the rectum giving rise to a sensation of fullness and frequent desire to defæcate. Digital examination will reveal the existence of a pedunculated tumour, or rarely of multiple tumours. Occasionally a polypus may protrude at the anus after defæcation, and must be distinguished from prolapse of mucous membrane by examination with the finger.

Another condition affecting the rectum which may be signalized by free bleeding is that of *papilloma* or *villous tumour*. The symptoms will closely resemble those of rectal polypi, but the blood is likely to appear in large quantities. Digital examination may discover a soft, velvety patch on the rectal wall, and the examining finger will be withdrawn covered with blood. The growth may be seen by means of a speculum as a soft, vascular mass, bleeding on the slightest touch. The condition is uncommon. It is likely to occur at an earlier age than cancer, but the latter is not unknown in persons under twenty.

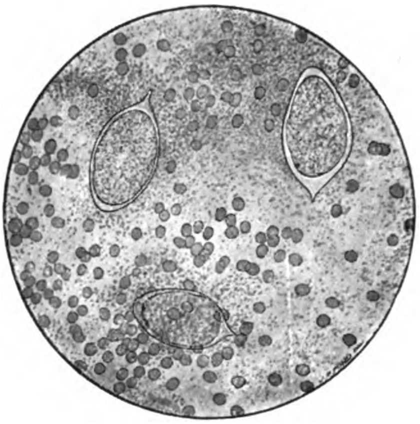


Fig. 26.—*Bilharzia hæmatobia*, with red corpuscles from centrifugalized urine. (*High power*).
(From a specimen kindly supplied by Captain Hallowes, R.A.M.C.)

Simple prolapse of the anal mucosa will lead to slight hæmorrhage. The condition is often seen in children, and may be recognized on inspection of the anus, when a red globular swelling of everted mucous membrane is visible. Adults will be conscious of having to push the part back after passing a motion. Such prolapse often accompanies piles.

Ulceration of the rectum, of venereal origin, occurs chiefly in women. Bleeding is not usually a very marked feature, but attacks of hæmorrhage may take place. The condition is recognizable by digital examination, and by inspection through a rectal speculum or the sigmoidoscope. The ulceration usually extends right down to the anus, whereas there is nearly always an interval of normal mucosa between the anus and an ulcerating cancer of the rectum.

The parasite called *Bilharzia hæmatobia* may occur in the rectum, though less frequently here than in the bladder. Its presence gives rise to the passage of mucus and blood per anum. There may be discomfort in the rectum and frequency of defæcation. Infection is contracted abroad, especially in Egypt—a fact which may lead to a suspicion of the presence of the affection in patients who have resided out of England. Diagnosis can only be made by finding the ova of the parasite in the fæces. Their well-known shape—oval with a pointed spike at one end (*Fig.* 26), or rarely at the side —renders them unmistakeable under the microscope.

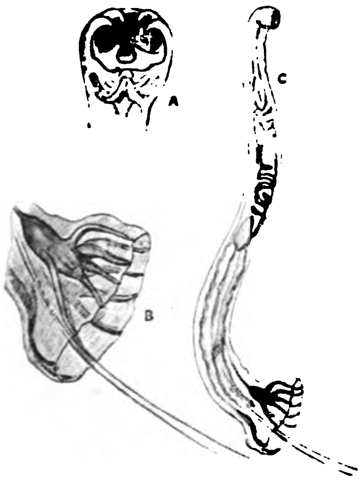


Fig. 27.—*Ankylostomum duodenale.* A. Head with hooks; B. Tail; C. Entire worm. (*Low power*).
(From *Medical Laboratory Methods.* Dr. Herbert French.)

In children the presence of thread-worms (*Oxyuris vermicularis*) in the rectum may lead to the discharge of small amounts of mucus coloured by a trace of blood. The worms will be seen readily on inspection of the child's motions. They are white, about as thick as coarse thread, and ½ to ⅝ in. long.

In some cases the actual cause of even much blood being passed per anum remains undiagnosable, and occasionally the cause seems to be 'vicarious menstruation,' notwithstanding the doubts held by many as to the possibility of the latter. The following is a very suggestive case from the practice of Dr. Reuell Atkinson : "A girl, age 13½, very tall for her age, menstruated regularly for more than a year. I was sent for because she was passing blood per anum ; that was on April 9. The blood was quite bright and in considerable quantity ; there was no other symptom except a little nausea and vomiting of frothy mucus. She complained of pain, mostly over the pubes on each evacuation, but none at other times. No abdominal tenderness or distention. No temperature. Nothing to be felt per rectum. On April 14 she passed a large dark, semi-digested clot. About the 24th she ought to have menstruated and did not. The blood continued to be passed until the 28th. Just a trace on the 29th. None since. She has remained well, menstruation has recurred regularly, and there has been no repetition of

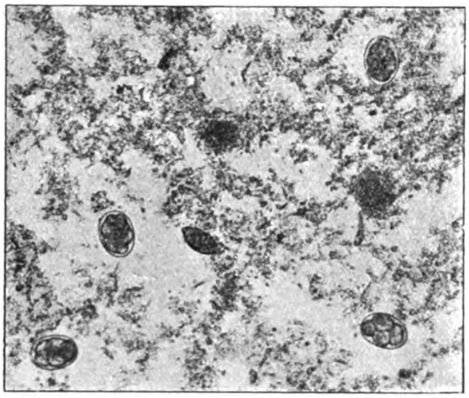


Fig. 28.—*Ankylostomum duodenale.* Ova at different stages. Near the centre is an ovum of *Trichocephalus dispar* (× 50). (By permission, from Dr. Haldane and Dr. Boycott's paper in *The Journal of Hygiene*, Vol. iii.)

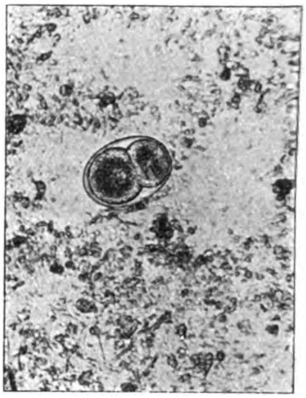


Fig. 29.—*Ankylostomum duodenale.* Two-cell stage of developing ovum (× 200). (By permission, from Dr. Haldane and Dr. Boycott's paper in *The Journal of Hygiene*, Vol. iii.)

SPECTRAL ABSORPTION BANDS.

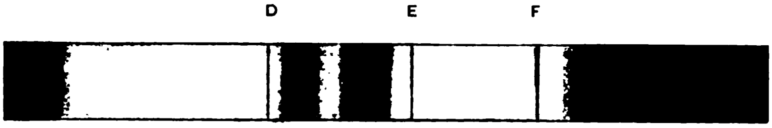


Fig. 30.—Oxyhæmoglobin.

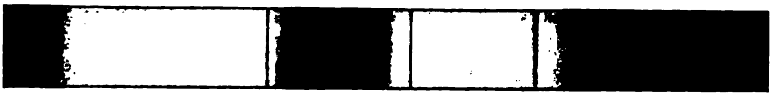


Fig. 31.—Reduced hæmoglobin.

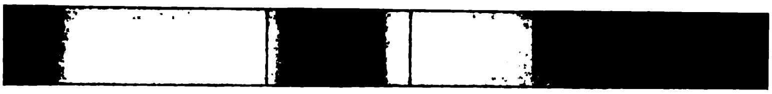


Fig. 32.—Carboxyhæmoglobin.

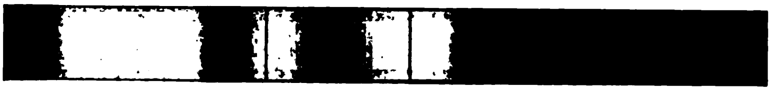


Fig. 33.—Hæmatin in alkaline solution.

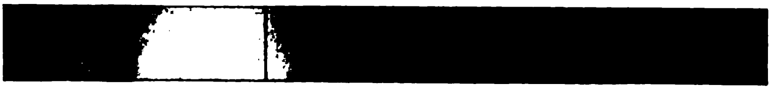


Fig. 34.—Acid hæmatin.

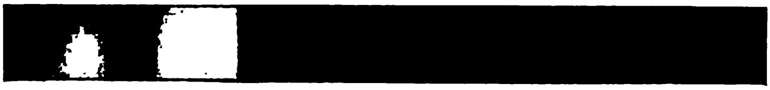


Fig. 35.—Methæmoglobin.

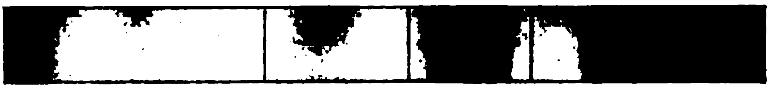


Fig. 36.—Urobilin.

the passage of blood per rectum, and she seems to be a normal girl. What was the cause of the bleeding, and where did it come from? Was it an instance of vicarious menstruation?"

Occult hæmorrhage is the term applied to the presence of minute traces of blood in the motions, revealed only by chemical or spectroscopical examination. It may occur in any lesion of the alimentary canal in which there is breach of surface, as in ulcer, cancer, or severe inflammation. Such hæmorrhage will also be present constantly in cases of infection with the parasitic worm *Ankylostomum duodenale* (*Fig.* 27). This condition, which is met with in persons who have resided in certain parts of the tropics such as India, or who have worked in mines or tunnels in which the soil has been contaminated by fellow-workers suffering from the disease, leads to profound anæmia; and the ova of the worms may be found in the fæces by microscopical examination (*Figs.* 28 and 29). The tests for occult bleeding may be applied in cases of difficulty when there is reason to suspect ulceration or cancer. No meat or meat-extracts must be administered for a day or two before the test is made, lest the hæmoglobin present in them should vitiate the results. The existence of any bleeding from the gums must also be excluded. One of the simplest methods of detecting occult hæmorrhage is to rub up some of the fæces with water, acidify with strong acetic acid, and then shake out with about $\frac{1}{5}$ volume of ether; the latter extracts the hæmatin, and the characteristic bands may be detected in the ethereal extract by means of the spectroscope (see *Figs.* 33 and 34). *W. Cecil Bosanquet.*

BLOOD, VOMITING OF.—(See HÆMATEMESIS, p. 265.)

BLOOD-PRESSURE, ABNORMAL.—Blood-pressure cannot be gauged accurately with the finger; when instruments of precision are used to verify opinions expressed as the result of merely feeling the pulse, it is astounding how erroneous digital impressions of pulse-tension and blood-pressure are. It is most important not to diagnose an abnormality of blood-pressure until the latter has been measured instrumentally. There are four main kinds of blood-pressure, namely, maximum systemic arterial; minimum arterial; mean arterial; and venous. Instruments have been devised for measuring all these, but clinically the only really important variety is the maximum systemic arterial blood-pressure. This may be either *abnormally low* or *abnormally high*, but no stress should be put upon any but considerable departures from the normal. Healthy individuals who have not been kept in bed have an average pressure in early adult life of 120 to 130 mm. Hg. Children have less than this, though at this early age it seldom happens that anything is to be learned by measuring the blood-pressure. As years advance, the blood-pressure tends normally to rise, so that at fifty or sixty a reading of 150 or 160 mm. Hg, or thereabouts, which in a younger person would indicate disease, would be normal.

Abnormally high blood-pressure may reach figures such as 320 mm. Hg, and anything from 170 mm. Hg upwards is essentially abnormal, whatever the age of the patient. It nearly always indicates rigidity of the vessels as the result of *arteriosclerosis*, and it is very often associated with *renal degeneration*, which, as time goes on, ultimately becomes red granular contracted kidney. Curiously enough, and contrary to what might be expected, the maximum systolic blood-pressure is higher than normal in cases of heart failure such as result from *mitral stenosis*, even when the pulse is so irregular and feeble that it can only be felt with certain beats, and when one would have thought that there must be a fall in the blood-pressure; the cause for the rise in such cases is probably the *partial asphyxia* acting upon the vasomotor centre; similarly, a rise of blood-pressure, even to 220 mm. Hg or more, may accompany the asphyxial attacks of Raynaud's syndrome. Cases of *melancholia* have abnormally high blood-pressures; when the melancholia improves, the pressure falls, and may return to normal when the patient recovers from the mental symptoms. The chief importance of high blood-pressure is in diagnosing arterial or renal degeneration, with consequent tendency to *apoplexy* or to *chronic heart failure*. It should be remembered that any patient who is kept in bed tends to have a diminution in the blood-pressure, and this applies to arteriosclerotic patients as well as others; a person may have a blood-pressure of 250 mm. Hg or more when up and about, and yet when he is kept in bed the pressure may fall to 150 mm. Hg, to rise again when he returns to active life. Prolongation of the first sound at the impulse or a ringing accentuation of the aortic second sound, may serve to indicate that there is a high blood-pressure when no instrument is at hand to verify the fact.

Abnormally low blood-pressure of moderate degree may be observed in many different circumstances associated with *asthenia* ; it is apt to accompany *Graves's disease* ; and *excessive smoking of cigarettes* ; but in itself a low maximum systemic blood-pressure is seldom of diagnostic significance excepting in *Addison's disease.* In a case in which the degree of pigmentation of the skin or of mucous membranes may leave doubt as to whether Addison's disease is the diagnosis or not, a blood-pressure so low as 80 mm. Hg would be confirmative of the diagnosis, although there are cases of Addison's disease in which the blood-pressure may be no lower than 110 mm. Hg. *Herbert French.*

BLUE SCLEROTICS.—(See FRACTURE, SPONTANEOUS, p. 242.)

BLUE-BRAIN.—(See DEAD FINGERS, p. 162.)

BOILS.—(See PUSTULES, p. 557.)

BONE, SWELLING ON A.—(See SWELLING ON A BONE, p. 667.)

BONES, SPONTANEOUS FRACTURE OF.—(See FRACTURE, SPONTANEOUS, p. 242.)

BORBORYGMI are gurgling noises in the abdomen produced by peristaltic movements of the bowel acting upon the mixed gaseous and fluid contents. With the stethoscope applied to the abdomen they may be heard in all normal persons, varying in intensity at different phases of digestion. When a meal has been taken after a period of fasting, the passage of the intestinal contents through the ileocæcal valve may be heard distinctly with the stethoscope placed over the right iliac fossa some six hours or less after the meal ; but it is seldom possible to decide what precise portion of the bowel is responsible for the production of borborygmi heard elsewhere.

Normally, these sounds should not be audible either to the patient or to other persons ; but occasionally even in health they may be heard quite loudly. In some individuals indeed, especially in women, the sounds become annoyingly obtrusive, and they may even acquire a pathological degree. They may be very loud when a person is beginning to get over-hungry. It may be very difficult, however, to decide exactly as to their cause ; sometimes the patient seems to be otherwise perfectly healthy. More often there is evidence of functional nerve disorder or hysteria, so that the borborygmi may be due to functional errors in the intestinal peristalsis or in the secretions within the bowel. They may be associated with FLATULENCE (p. 240), though by no means necessarily so. Observation of the patient may detect air-swallowing ; intestinal putrefaction is indicated by excess of indican in the urine, or by a high ratio of organic to inorganic urinary sulphates ; fermentation of carbohydrate is suggested when there is no evidence of air-swallowing, when urine analyses do not confirm any suspicion of proteid putrefaction, and when the borborygmi are increased by carbohydrate foods.

Borborygmi are apt to be increased in asphyxial conditions, and may be very marked in cases of heart failure with cyanosis.

The absence of borborygmi may sometimes be important, for one of the first effects of peritonitis is to inhibit peristalsis ; without peristalsis borborygmi cannot be produced, and therefore, if peritonitis is suspected, the presence of well-marked borborygmi on auscultation of the abdomen is an argument against there being general peritonitis, whilst complete silence of the abdomen is in favour of this diagnosis. *Herbert French.*

BRADYCARDIA, or undue slowness of the pulse-rate, is compatible with health, some individuals having a normal pulse-rate of 50, whilst in a few it does not exceed 40 or even 30 per minute. Occasionally bradycardia of this kind is found in more than one member of the family. It is important to auscultate the heart to exclude the possibility of the rate of the pulse as felt at the wrist not being the same as the rate of the heart-beat ; often, particularly with mitral stenosis, by no means every pulse wave becomes palpable at the wrist, and the rate may then seem to be slow when perhaps in reality it is twice the apparent rate.

Absolute slowness of the pulse-beat, as distinct from its relative slowness in proportion to the pyrexia, is best seen in the symptom-complex termed *Stokes-Adams' disease*, the phenomena of which are syncopal attacks associated with epileptiform convulsions, coma, stertor, and cyanosis, the rate of the heart-beat being found to have dropped to a half or even to less than half of that which is natural. These symptoms are due to difficulty in the transmission of the contraction-stimulus from the auricle to the ventricle along the auriculo-ventricular bundle of His. The inhibitory factor is not the same in all cases, but is often associated with arteriosclerosis and degenerative changes in the bundle of His, together with myocardial degeneration and atheroma of the coronary arteries ; or to syphilis of the bundle of His or to destruction of that bundle by a gumma, sarcoma, or carcinoma. The diagnosis is apt to be that of epilepsy until the fact has been established that the pulse-rate falls during an attack to about half the normal ; but when this observation has been made, the difference between Stokes-Adams' disease and ordinary epilepsy is clear. The phenomena are those of 'heart-block,' the diagnosis of which in its lesser degrees cannot be made without careful instrumental records of the venous and arterial pulses and of the cardiac movements, made either by means of the polygraph, or better still the electrocardiograph. Considerable slowing of the pulse-rate has also been noted in some cases of *uræmia*, even without heart-block ; both in the chronic type of the affection and during uræmic coma. Bradycardia is by no means constant in uræmia however.

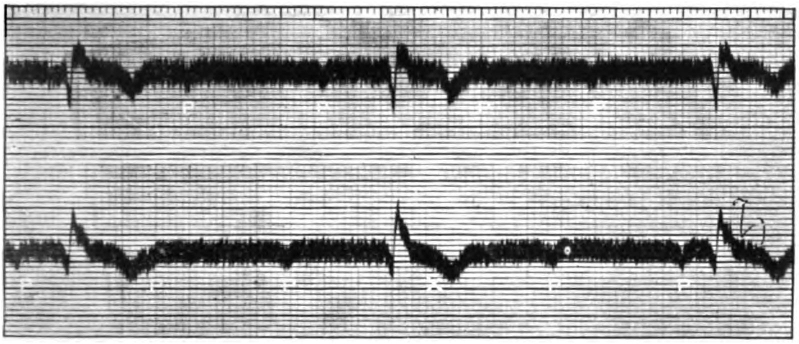


Fig. 37.—Electro-cardiogram showing complete heart-block. The auricular waves (P) recur at equal intervals, but bear no relationship to the ventricular waves. At X the auricular and the ventricular waves are simultaneous.

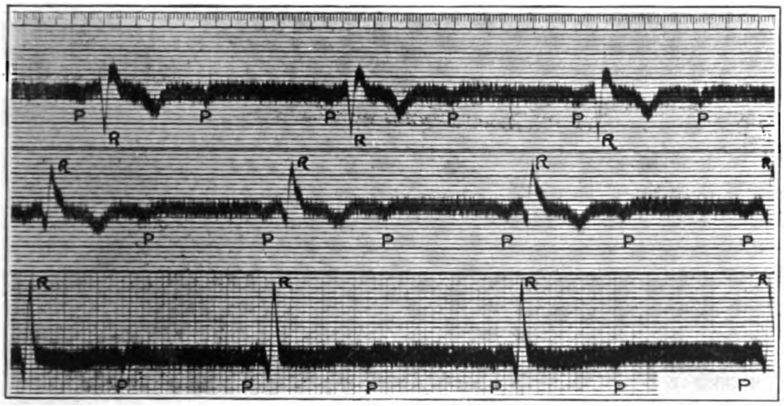

Lead 1. Right arm to left arm.
Lead 2. Right arm to left leg.
Lead 3. Left arm to left leg.
P
R
P
P
R
P
P
R
P
R
R
R
R
P
P
P
P
P
P
R
R
R
R
P
P
P
P
P
P

Fig. 38.—Electro-cardiogram to illustrate one-in-two heart-block. It will be noticed that the auricles and ventricles are both beating perfectly regularly, but the auricle notches (P) are twice as numerous as the ventricle notches (R). Only every second auricular beat was followed by a ventricular beat. The illustration is from the same patient as *Fig.* 37, but upon a different occasion. He sometimes exhibited partial heart-block as above, and sometimes complete, as in *Fig.* 37.

Increased intracranial pressure sometimes causes bradycardia in certain cases of *cerebral hæmorrhage, tumour* or *abscess*, and in the early stages of *tuberculous meningitis*; in other forms of meningitis, and in the later stages of tuberculous meningitis, the initial bradycardia changes to tachycardia. If in a given case there is otitis media or some other local infective focus which might produce a cerebral abscess, pyrexia with a pulse-rate of 50, 55, or 60 is an argument in favour of intracranial abscess; the other complications of otitis media, especially lateral sinus thrombosis, mastoid abscess, or suppurative meningitis, produce a rapid pulse-rate instead of a slow one; the reverse is not true, for it is not possible to exclude cerebral abscess merely on the ground that there is no bradycardia. Cerebral tumour can generally be distinguished from cerebral abscess by the greater length of the history, the more pronounced optic neuritis, or the absence of predisposing cause to cerebral abscess, such as otitis media or bronchiectasis; whilst cerebral hæmorrhage is more rapid in its onset, is less likely to have marked optic neuritis, and if there is pyrexia it is apt to be extreme, reaching the level of hyperpyrexia; generally the patient is an elderly man who has either high blood-pressure, albuminuria, or other evidence of degenerated arteries or granular kidneys.

In *myxœdema* the pulse-rate is seldom fast, and it may be abnormally slow.

Certain drugs are apt to slow the heart markedly when they have been administered in full doses over a long period, the three most important being *digitalis, strophanthus*, and *sodium salicylate*: the diagnosis depends on knowledge of the medicine the patient is taking.

Jaundice is generally stated to cause marked slowing of the pulse-rate: it is true that artificial introduction of bile salts and pigments into the circulation in animals slows the heart, but clinically in man it is rare to find jaundice and absolute bradycardia associated.

Herbert French.

BRADYPNŒA, or undue slowness of breathing, is not a very common symptom, but it may be met with in marked degree under various conditions, of which the following are the chief:—

1. **As an Effect of certain Drugs or Poisons**:—

Chloroform
Opium
Morphia
Alcohol
Chloral
Chloral hydrate
Butyl chloral hydrate
Veronal
Sulphonal
Trional
Tetronal
Medinal
Aconite
Antimony.

2. **Cerebral Compression resulting from**:—

Depressed fracture of the skull
Meningeal hæmorrhage
Cerebral hæmorrhage
Pontine hæmorrhage
Cerebral tumour
Cerebral abscess
Cerebellar tumour
Cerebellar abscess
Osteoma of the cranium
Gumma of the meninges.

3. **Shock or Collapse from**:—

Severe injury
Sudden onset of acute illness
Operations
Excessive loss of fluid from choleraic diarrhœa.

4. **Caseous Bronchial Glands.**

5. **Functional Conditions**:—

Hysteria | Epilepsy | Catalepsy | Trance.

6. **Uræmia.**

7. **Diabetes Mellitus with Impending Coma ('Air-hunger').**

Although bradypnœa may result from any of the above causes, it is not constant in most of them, and in the majority it is an incident which, even if present, is not of diagnostic importance. This applies particularly to the conditions mentioned in Groups (1) and (2), in many of which the patient is likely to be at least stuporous, and perhaps completely comatose (see COMA, p. 117). The *cerebral lesions* will be indicated by associated headache, vertigo and vomiting, and confirmed by the discovery of optic neuritis (*Plate XIX, Figs. K, L*, p. 416).

Now and then, in the case of a child suffering from tuberculous meningitis, one comes across a curious type of slow breathing, in which two or perhaps three short respirations occur in quick succession, followed by so long a pause that the patient may appear to be dead. This type, known as Biot's breathing, does not resemble Cheyne Stokes' breathing (p. 107) clinically at all, but it is probably related to it pathologically. It occurs in those who are approaching death, but may be present for a day or more before death actually occurs.

If the bradypnœa is due to a *poison*, the circumstances of the case may suggest this, and it may be confirmed by chemical analysis of the gastric contents or of the contents of adjacent bottles; though there may be the same difficulties of deciding whether the patient is 'drunk or dying,' as are discussed on p. 118. One important point is not to conclude forthwith that the presence of sugar in the urine indicates diabetic bradypnœa and coma, for numbers of patients suffering from deep alcoholism have sugar in their urine for the time being; generally, however, without acetone (p. 8) and in a low specific gravity urine.

In cases of *shock* or *collapse* the existence of bradypnœa will be overshadowed by the other symptoms in the case, and it is not in itself important.

The slow breathing that results sometimes from *caseous bronchial glands* differs from most of the above in that it affects patients, generally children, who are not acutely ill; though delicate, they may even be going to school, and yet their respiration rate may be as slow as 12 or even 10 to the minute for weeks or months. There is generally tachycardia at the same time. Many such children shake off their delicacy in the course of a year or two, for the majority of cases with caseous bronchial glands get well without being diagnosed; but the relationship between this bradypnœa and affection of the glands has been established repeatedly in patients dying from accident or other causes. During life the diagnosis may be established by finding the shadow of the caseous glands in the thorax with the x-rays (*Fig.* 61, p. 149).

Little need be said about the functional conditions in which bradypnœa may occur. *Old people* tend to breathe much more slowly than young unless there is shortness of breath from emphysema, bronchitis, or myocardial affection. *Epileptics* breathe normally between their attacks; but during a seizure they cease breathing altogether for the first twenty seconds or so—the tonic stage—and then their respirations start slowly and stertorously; the bradypnœa may then cease suddenly, or it may persist in minor degree during the period of post-epileptic stupor. *Hysteria* may produce almost any symptom; bradypnœa is possible, though tachypnœa is more common; the diagnosis depends upon other features of the case (p. 465). *Catalepsy* and *trance* are both mental conditions, diagnosed by watching the case or by the history; in catalepsy the movements of respiration may be very slow, but they are obvious; in trance, on the other hand, the breathing movements may be apparently absent altogether for days or weeks, the patient lying motionless like one dead. The chief difficulty is to exclude actual death; the thermometer helps much—the body does not become cold; the heart sounds may be just audible even though the pulse cannot be felt; and the fact that some respiration is taking place may be recognized by holding a bright mirror close to the nostrils and mouth, when a slight dimming from condensation of expired air will be seen. In very exceptional cases, however, death is simulated so closely that the patient has been upon the point of being buried before the mistake has been discovered.

Uræmia may be associated with breathing that is either rapid, or normal, or slow; the latter is exceptional; but in some cases of uræmic coma bradypnœa is pronounced. Cerebral compression by a hæmorrhage, abscess, or tumour may be simulated, and a knowledge that the urine contains albumin and tube casts, and that the blood-pressure is high, will not always decide between them. Recurrent convulsive seizures would point to uræmia to some extent, but they may also occur from gross brain lesions, and optic neuritis may also be common to both. To clinch the diagnosis of uræmia it may be necessary to test blood or cerebrospinal fluid to see if it contains excess of urea.

Diabetes mellitus is liable to cause the most characteristic bradypnœa of all—the 'air-hunger' of diabetic coma. This is not a dyspnœa, as the name might suggest, but a condition of extremely deep slow breathing with a maximum respiratory excursion both in the intake and in the output of air. The 'hunger' for air is one of getting the maximum of air in and out with each deep slow breath, rather than one of getting in as many breaths as possible in a given time. The patient becomes increasingly drowsy, and generally complains of pains in the upper half of the abdomen. The breathing rate begins to fall from 18 to 16, to 14, and progressively down to perhaps only 6 to the minute. There is a long pause between each breath, and then inspiration starts and, without any hurry, the stuperose patient goes on drawing air deeper and deeper into his chest until he cannot expand it to take in any more; the head is often thrown slowly back during the process, the mouth slowly opens wider and wider as the head goes back; then there is a pause at the height of inspiration before an equally deep, slow, solemn expiration follows, and the head comes forward and the mouth closes partially until the next slow deep inspiration is in progress. The patient seldom lives much longer than forty-eight hours after this onset of air-hunger and diabetic coma, but the air-hunger is sometimes seen in cases not yet comatose. It may then pass off for a time, but it is always a sign of grave danger, and it is the most characteristic of all the forms of bradypnœa.

Herbert French.

BREAST, DISCHARGE FROM.—(See DISCHARGE FROM THE NIPPLE, p. 181.)

BREAST, PAIN IN.—(See PAIN IN THE BREAST, p. 429.)

BREAST, SWELLING OF THE.—(See SWELLING, MAMMARY, p. 685.)

BREATH, FOULNESS OF THE.—This is due to one or other of four main groups of conditions, namely, septic and putrefactive changes within the mouth or nose; septic or putrefactive changes within the lungs; smoking or the ingestion of substances, such as garlic or onions, whose products are excreted by the lungs or saliva; and severe toxic conditions, especially those affecting the alimentary canal or peritoneum.

When the foulness of the breath is not habitual, but occurs as the result of recent illness, there will be symptoms of the latter which point to the diagnosis quite apart from the condition of the breath, and one need merely indicate as possible causes such things as typhoid fever, general peritonitis, post-puerperal sepsis, intestinal obstruction, and a host of other conditions of this kind in which, even though the mouth be clean, there may be foulness of the breath, such tendency being greatly exaggerated if sordes have been allowed to collect.

Foulness of the breath due to the ingestion of foodstuffs such as *onions* or *garlic* is familiar enough; there are certain drugs, for instance *guaiacol* or *paraldehyde*, which may produce a similar symptom without the patient's friends realizing why the breath should be so tainted.

Foulness of the breath due to lung conditions will nearly always be indicated either by the abundant and putrid sputum, or by the abnormal physical signs in the thorax. The condition may be due to *phthisis* with secondary infection of the cavities by pyogenic organisms, *fœtid bronchitis*, *bronchiectasis*, *gangrene of the lungs*, *empyema* or other *abscess* which has ruptured into the lung. The cases which give rise to most difficulty in differential diagnosis are those in which an empyema has been situated deeply, for instance between the lower lobe and the diaphragm, or between two lobes, without reaching the surface: there may be absolutely no abnormal physical signs, and the diagnosis has to be made from the symptoms and history. The patient has generally had some obscure febrile illness, possibly with cough, but without much expectoration, until one day, after a particularly severe bout of coughing, a large quantity of pus—perhaps a teacupful or more—has been brought up suddenly, since when, at intervals of hours and days, there has been similar expectoration of quantities of putrid pus. Deep-seated empyema without abnormal physical signs most resembles bronchiectasis or bronchiolectasis, but is distinguished by the sudden way in which the first large quantity of purulent expectoration came on. In both cases there may be clubbing of the fingers, the sputum contains pus corpuscles and pyogenic and non-pyogenic micro-organisms other than tubercle bacilli, but no elastic fibres indicative of lung destruction.

Gangrene of the lung produces an unmistakeable stench of the worst kind; the detection of elastic fibres in the sputum, after boiling with caustic soda to destroy other tissue elements, clinches the diagnosis.

Phthisis with cavitation may produce foulness of the sputum, but hardly ever the stench of gangrene, unless gangrene has supervened. It is distinguished from bronchiectasis and from hidden empyema by discovering tubercle bacilli in the sputum. The chief difficulty arises when the tuberculous part of the malady has ceased, the cavities formerly excavated by the tuberculous process having been usurped by secondary pyogenic organisms.

Foul breath is due in the great majority of cases to local decomposition in the mouth, often diagnosable on simple inspection in the form of *tartar*, *septic gums*, *carious teeth* with decomposing food particles in them, *pyorrhœa alveolaris*, or *stomatitis* (p. 542); or it may be that the nose or throat are at fault rather than the mouth, as the result of *necrosis of the nasal bones*, *purulent hypertrophic* or *atrophic rhinitis*, *ozœna*, *septic tonsillitis* or other varieties of Sore Throat (p. 613); very vile foulness of the breath occurs with *Vincent's angina* (p. 614), and with *squamous-celled carcinoma* of the mouth or tongue; in children the possibility of some *foreign body* having got impacted in the throat, nose, or nasopharynx should not be forgotten.

It is only when all such local conditions have been excluded, and when there is no acute illness nor any lesion of the lungs, that one can attribute foulness of the breath to *constipation* or to *dyspepsia*. It is sometimes very difficult to find out why the patient's breath is not sweet, and indeed there are some persons in whom all the functions of the body seem to be normal, and the mouth clean, and yet the breath is foul. If there are any symptoms of gastro-intestinal disorder, especially flatulence or constipation, one is inclined to attribute the condition of the breath to the stomach or the bowels; but when there are no symptoms of error in these, it is more than likely that the trouble is due to some local condition not discovered on ordinary inspection, particularly putrefaction of food particles which may become impacted between the teeth even in persons who use both tooth-brush and mouth-wash daily. *Herbert French.*

BREATH, SHORTNESS OF.—This is a very common complaint which should be differentiated carefully from difficulty of breathing, the latter term being reserved entirely for cases of obstruction in the main air-passage, the larynx, and trachea—diphtheria, growths, and, very much more rarely, pressure from without being the main causes. Shortness of breath is, in the patient's mind, a *conscious* quickening of the respiratory movements to supply a conscious need of air. The following are chief causes:—

Increased Need for Oxygen.—Fevers and other septic processes inducing excessive oxygen requirements. Exercise in health—temporary shortness of breath.

Diminished Supply of Oxygen.—(1) Blood conditions in which the red corpuscles cannot carry a sufficient charge, or do not yield up their supply with sufficient ease; (2) Cardiac conditions of inefficiency of circulation; (3) Pulmonary conditions of diminished surface of contact, or ease in contact, of air and blood in alveoli; (4) Atmospheric conditions of diminished partial oxygen pressure in the alveoli of the lung; (5) Deformities of the chest mechanically preventing the expansion of the lung. The diagnosis of these conditions is not difficult when once attention is drawn to the possibility of their occurrence, but we must advert briefly to each of them to indicate the guides to the cause in a case not at once obvious.

Fevers and Septic Conditions.—The thermometer and the obvious illness of the patient will generally indicate these; nor indeed is shortness of breath a common *complaint* in such patients, their minds being filled with other ideas.

Exercise in Health.—Here it is necessary to be sure of the health; it may or may not be that the person is merely out of condition, and undertakes exercise which only a trained athlete can perform properly. The only way to avoid mistakes is to ask, Does the shortness of breath soon disappear? and then to make a careful examination of the patient to see if any of the undermentioned causes are at work:—

1. **Blood Conditions.**—These include: (i) Simple loss of blood; (ii) Anæmia simplex; (iii) Anæmia, severe, pernicious or leukæmic; (iv) Polycythæmia; (v) Some pathological constituent, as in diabetes, uræmia, Graves's disease, etc.

The actual laboratory diagnosis of the blood condition is simple enough if we decide to have it examined. The points that may lead us to have this done would naturally come in the following order. A history of loss of blood is pretty sure to be volunteered—piles, excessive menstruation, obvious trauma, loss at parturition, etc.; suspicion is very likely to be aroused by the colour of the patient's face, especially when coupled with a primary complaint of shortness of breath. *Diabetes* and *uræmia* are likely to show other signs, and the urine will give the clue to the diagnosis. Never omit to have the blood examined if the cause of a shortness of breath is not apparent on simple physical examination; indeed, one must go farther, and say if some easily diagnosable condition is not present; for it must be remembered that blood conditions are the very ones to be the exciting cause of cardiac inefficiency, which by itself is often hard to diagnose if there be no obvious bruit or irregularity in rhythm.

2. **Cardiac Conditions.**—Inefficiency in circulation. These include: (i) Valvular disease (acute and chronic); (ii) Muscular weakness (fatty, fibrosis, etc.); (iii) Nerve conditions (arrhythmia ?); (iv) Pericarditis and pericardial effusion.

i. *Valvular Disease.*—If a bruit be present, it may fairly be assumed that the heart is a factor in causing shortness of breath, but unless some other tell-tale sign be present it must not be assumed that it is the *only* factor, for it is very common to find patients with bruits who will not confess to shortness of breath.

ii. *Muscular Weakness.*—We cannot under the present heading give all the points in connection with 'morbus cordis sine murmure'; it must, however, be stated that a diminution in the muscular energy of the heart is a most important contributory factor in producing shortness of breath in all pathological conditions of the blood, including renal affections and diabetes, in convalescence from acute disease, and in acute pericardial affections; it is, perhaps, the commonest cause of all of shortness of breath. Want of tone in the sounds, likeness of the first to the second sound, and irregularities in rhythm, are the principal points to look for. The urine should be examined with care, both for albumin and tube-casts; the ophthalmoscope should be used in the detection of albuminuric retinitis; and it is often wise to measure the systemic blood-pressure to find out whether it is greatly above the normal or not. In this connection fat wants special mention; fatty degeneration of heart muscle, and overloading of the heart with interstitial fat will both cause shortness of breath, and it is practically impossible to differentiate the two with certainty during life. In very stout individuals the latter is of course to be suspected, but the former cannot be excluded; in fact, when such a patient complains of shortness of breath, his case requires the greatest acumen to decide the cause and then the treatment. The previous general health affords the strongest clue, coupled with the history of the onset of the shortness of breath. In a stout subject, as indeed in all other patients in whom I am trying to judge the question, "Am I dealing with a case of cardiac insufficiency without a bruit ?" I adopt the following simple plan. I listen with the stethoscope to the heart, counting the pulse frequency and noting the sounds while the patient is sitting in a chair in the course of conversation, and again do the same while he is standing. I then make the patient hurry in his movements, run upstairs, or several times across my consulting room, and again repeat my observations on frequency, rhythm, and sounds; I then get him to lie down on a sofa whilst I make another examination on the same points. It is thus possible in three or four minutes to get most valuable information as to the response of the heart to increased work, as well as to relief from work, and to draw pretty accurate conclusions as to its muscular efficiency, which after all is the chief point to be considered.

I roughly assume that there is an average difference in health of about five beats per minute between sitting and standing, that effort should increase this difference to somewhere about fifteen to twenty beats a minute, and then in about three minutes a reasonably healthy heart should resume its resting frequency from such mild exertion as mentioned above. If exertion removes a 'resting' irregularity in rhythm, I assume the heart is muscularly in a reasonable state of health.

iii. *Nerve Conditions.*—Local pressure on the nerves may cause cardiac arrhythmia and breathlessness, but these will have other signs and symptoms easily discoverable; general nervousness and neurasthenia are often characterized by shortness of breath on exertion or excitement, there being frequency of the beat without any arrhythmia.

iv. *Pericardial Diseases.*—A differential diagnosis between these and a hypertrophy or dilatation of the ventricles may be demanded for other reasons, but *qua* shortness of breath, there is no difficulty in determining that either cardiac or pericardial trouble is the cause.

3. **Pulmonary Conditions.**—These, again, will be fairly obvious on proper examination, including, as they do, every disease of the lung; but we would specially draw attention to the possible presence of a quiet pleural effusion, which not very infrequently is so insidious as to give rise to no complaint but that of shortness of breath. Again, in the early days of phthisis, it may be that a cough and shortness of breath are nearly all that is complained of. Bronchitis, advanced tubercle, bronchopneumonia, lobar pneumonia, and acute

pleurisy, are all easily recognizable causes of shortness of breath. The only intrinsic affection of the lungs not at once easily discoverable is emphysema without its usually accompanying bronchitis; the shape of the chest, the deficiency of vesicular sounds, the increased resonance to percussion will generally give a clue.

4. **Atmospheric Conditions** need no diagnosis; partial asphyxia by bad air, high mountains, and caisson work, are the three chief alterations in gaseous surroundings. All are obvious.

5. **Deformities of Chest** are again obvious; Pott's curvature is the chief one. They derive their importance from the fact that commonly one lung is *hors de combat* almost to start with, and hence a very slight affection of the other may cause great difficulty in breathing.

Fred J. Smith.

BREATHING, CHEYNE-STOKES.—(See CHEYNE-STOKES RESPIRATION, p. 107.)

BREATHING, SLOW.—(See BRADYPNŒA, p. 84.)

BRITTLE BONES.—(See FRACTURE, SPONTANEOUS, p. 242.)

BRUITS, CARDIAC.

I.—SYSTOLIC BRUITS.

(*A*). **Systolic Bruits in the Mitral Area.**—When a definite systolic bruit is audible over the mitral area which corresponds to that portion of the chest wall lying immediately over the cardiac apex, its cause is sometimes obvious. If, for example, a person who has previously had an attack of rheumatic fever presents a bruit with its point of maximum intensity over the cardiac apex, conducted outwards into the left axilla, there being lost, and heard again near the inferior angle of the left scapula, then such a bruit is almost certainly due to organic disease of the mitral valve causing regurgitation through it. This is confirmed by finding that the heart is enlarged, the area of cardiac dullness increased, and the apex beat displaced downwards and to the left. Such enlargement points to the cardiac condition not being of recent origin; bulging of the præcordia, often seen in children, is additional evidence in the same direction.

In some cases, however, the diagnosis is not so obvious, and for a definite conclusion to be arrived at it is necessary to consider all the following conditions which may produce a systolic bruit in the mitral area:—

(1). Mitral regurgitation, due to chronic organic disease of the mitral valve.

(2). Acute endocarditis: (*a*) Simple; (*b*) Ulcerative or malignant.

(3). Mitral regurgitation where there is no disease of the mitral valve, but dilatation of the left ventricle as the result of (*a*) Disease of the aortic valve; (*b*) Disease of the myocardium, such as myocarditis, parenchymatous degeneration, fatty heart, fibroid heart; (*c*) Disease external to the heart, causing hypertrophy and dilatation of the left ventricle such as arterial sclerosis and interstitial nephritis; (*d*) Adherent pericardium, which is frequently associated with organic disease of the valves.

(4). Functional bruits.

(5). Cardio-respiratory bruits.

(6). Congenital malformation of the heart.

(7). Aneurysm of the heart.

(8). Acute pericarditis.

1. The following points are in favour of the bruit being due to *organic disease of the mitral valve* of long standing: (*a*) Enlargement of the heart, shown by displacement of the apex beat and increase in the area of cardiac dullness. In mitral regurgitation the enlargement is due to hypertrophy and dilatation of the left ventricle, the differential diagnosis of the other causes of which will be found on p. 206; (*b*) A history of past rheumatic fever or of chorea; (*c*) The age of the patient; in children and young adults mitral regurgitation is far more likely to be the result of a previous endocarditis than of dilatation of the mitral orifice without valvular disease; (*d*) The absence of pyrexia helps in excluding a recent endocarditis, though in children suffering from rheumatic endocarditis the temperature is often normal while they are being treated with salicylates. In cases of recent endocarditis there may be no physical signs of any great enlargement of the left ventricle, and usually the apex beat is found close to its normal position.

2. *Acute endocarditis* is nearly always associated with some other affection; for example, there may be, or have been, acute rheumatism or chorea, or pneumonia or some other infectious process, such as scarlet fever, erysipelas, septicæmia, or puerperal fever. The heart is not found to be enlarged, or only to a slight extent, provided that the condition is not one of an acute endocarditis affecting old sclerotic valves; the bruit is soft and blowing—never musical in simple cases—and it is localized to the impulse instead of being transmitted into the axilla. In *malignant endocarditis* the constitutional disturbances may be severe; the points in the diagnosis will be found on p. 34.

3. The points in favour of *mitral regurgitation due to dilatation of the left ventricle* are: (*a*) The age of the patient; myocardial degenerations, except those occurring in infectious processes, are not likely to be present before middle life; (*b*) The presence of arterial sclerosis and chronic interstitial nephritis, as determined by increased blood-pressure, accentuation of the aortic second sound, thickening of the radial arteries, retinitis, and polyuria with a trace of albumin; (*c*) The existence of non-rheumatic aortic obstruction or regurgitation with hypertrophy and dilatation of the left ventricle; (*d*) Shortness of breath and cardiac distress upon exertion, without any obvious cardiac lesion; if these be associated with œdema of the legs, engorgement of the lungs, and enlargement of the liver without a very high blood-pressure and without obvious primary lung trouble such as fibrosis or emphysema, dilatation of the mitral orifice as the result of *myocardial degeneration* is probable. If this is the result of fatty infiltration, the cardiac condition is often part of general obesity.

Regurgitation through the mitral valve may be caused by a dilatation of the left ventricle dependent upon an *adherent pericardium.* The following signs of adherent pericardium must be looked for: (*a*) *Systolic retraction*, which is best determined by inspection of the chest wall from the side, and is due to an indrawing of the intercostal spaces during the ventricular systole. When this is situated near the apex beat it is due to an adherent pericardium: it may also be noticed over the lower sternal region, or at the ensiform cartilage, or over a lower left rib behind the posterior axillary line. Systolic retraction is not always due to an adherent pericardium, for in thin persons and in children a systolic indrawing of the third and fourth left intercostal spaces close to the sternum is often seen, and is produced by the normal recession of the base of the heart during each ventricular systole. Systolic retraction due to adherent pericardium is often followed by (*b*) The *diastolic shock*, palpable and due to the sudden relaxation of the ventricular wall; (*c*) *Diastolic collapse of the veins of the neck*, or Friedreich's sign, which is produced during the ventricular diastole; it is found chiefly in this condition, but does not always occur, and is sometimes seen without pericardial adhesion being present; (*d*) The *pulsus paradoxus*, the cardiac beats becoming more feeble at the end of inspiration, so that during each inspiration the pulse-beat becomes very weak, or is lost.

4. A systolic bruit at the cardiac apex may be *functional* in origin, in which case it is localized to the mitral area, being conducted only for a short distance into the axilla, and not heard posteriorly. The condition is associated with anæmia and other debilitating conditions. Other functional bruits are nearly always associated with it, especially one in the pulmonary area, and also a *bruit de diable* in the neck.

5. A *cardio-respiratory* bruit is frequently heard at the cardiac apex, and is sometimes mistaken for one caused by mitral regurgitation. The bruit varies with the movements of respiration, the more usual sounds heard being bruits corresponding with the ventricular systoles of two or three heart-beats during inspiration. Such murmurs should not be ignored, as they may be due frequently to pleuritic friction. To distinguish them from cardiac bruits is usually easy, and the chief diagnostic points are: (*a*) Cardio-respiratory bruits vary in intensity with the movements of respiration, being louder during inspiration. (*b*) When present at the apex they are abolished when the breath is held in deep inspiration. (*c*) They vary in intensity and character with alterations in the posture of the patient. (*d*) The bruits sound nearer to the ear than cardiac bruits. (*e*) Each bruit commences suddenly and ends abruptly. (*f*) They are not conducted in the recognized direction of valvular murmurs.

6. A *congenital* systolic bruit, when heard in the mitral area, is always part of a loud bruit with its point of maximum intensity nearer the base of the heart. When such a murmur is heard in children, with little or no displacement of the apex beat, and the area

of cardiac dullness is increased to the right of the sternum, the condition is always congenital. The lesion will generally be either patent septum ventriculorum, pulmonary stenosis, or patent ductus arteriosus (p. 156). Mitral regurgitation due to a congenital defect practically never occurs.

7. An *aneurysm at the cardiac apex* is rare, and is scarcely possible to diagnose, so that it need not be taken into account when considering the differential diagnosis of apical bruit.

8. When *acute pericarditis* is present, a systolic bruit which is part of a 'to-and-fro' friction murmur may be heard in the mitral area. Such a murmur changes its character with the pressure of the stethoscope and with the different phases of respiration; and it is not conducted into the axilla. Other signs of pericarditis are usually present (p. 213).

(*B*). **Systolic Bruits over the Pulmonary Area,** i.e., over the second left intercostal space close to the sternum, may be caused by the following conditions:—

(1). *Congenital cardiac malformations,* especially pulmonary stenosis and patent ductus arteriosus.

(2). *Functional bruit.*

(3). *Cardio-respiratory bruit.*

(4). *Acquired pulmonary stenosis,* which is a very rare lesion.

To distinguish between an organic and congenital defect and a functional condition is usually quite easy. *Pulmonary stenosis* is nearly always congenital, and is therefore found for the most part in children; and its presence is confirmed by other signs of congenital heart disease, such as little or no displacement of the apex beat with considerable enlargement of the right side of the heart, together with cyanosis of varying degree, and *clubbing of the fingers* (p. 111) and toes. With a *patent ductus arteriosus* the bruit is often similar, although cyanosis and clubbing of the fingers and toes are usually absent; instead of the murmur being definitely either systolic or diastolic in time, a long rumbling bruit, commencing during systole and passing on into the diastole of the ventricles, is heard. Such a bruit is considered to be pathognomonic of this congenital defect, as it is impossible for a bruit extending from systole into diastole to be produced within the heart. When a patent ductus arteriosus is present, *x*-ray examination of the heart sometimes shows a shadow bulging to the left between the arch of the aorta and the left ventricle. It appears like a 'cap' above the ventricle, due to dilatation of the pulmonary artery. *X*-ray examination will help in the diagnosis of most forms of congenital heart disease, because it shows definitely the enlargement of the right heart with little alteration in the position of the left border.

Other congenital malformations, such as a *patent interventricular septum,* may produce a systolic bruit in the pulmonary area, though the maximum intensity of the abnormal sound is lower down on the left of the sternum; in many cases, however, the differential diagnosis of the ventricular congenital malformation is impossible.

2. The *functional pulmonary bruit* is common in chlorosis and other anæmic and debilitated conditions, and in exophthalmic goitre; it is also frequent in school-children æt. 5–15. The bruit alters with the position of the patient, being louder in the recumbent than in the erect posture, whereas in congenital defects the position of the patient has very little influence upon the loudness. The presence of a *bruit de diable* in the neck confirms the diagnosis of the functional origin of the bruit, and there is generally no such increase of cardiac dullness to the right of the sternum as occurs in congenital malformation and acquired pulmonary stenosis. A systolic thrill may be present in the pulmonary area both in organic and functional conditions, but is more common in the former and therefore in favour of pulmonary stenosis.

A systolic bruit is frequently heard over the upper portion of the manubrium in young children in the sitting posture, when the head is so raised that the eyes are looking directly up at the ceiling. It disappears when the chin is lowered. It is usually of no importance, although it may be a sign of enlarged lymphatic glands at the bifurcation of the trachea.

3. *Cardio-respiratory bruits* are sometimes heard at the base of the heart, and more often on the left side of the sternum. They vary with the movements of respiration and also with changes in the posture of the patient, but not in so definite manner as do the cardio-respiratory bruits which are heard in the mitral area.

4. *Pulmonary stenosis* may be an acquired lesion, although very rarely; if in a young adult such a bruit as has just been described is present, and if there is a past history of

rheumatic fever, together with lesions of the other valves, especially the mitral, then it may be fairly presumed that the bruit is due to an acquired pulmonary stenosis. The history helps greatly in the diagnosis, for if the lesion were congenital there would be symptoms of its presence dating back to infancy.

Systolic bruits due to other valvular lesions may also be heard over the pulmonary area, but they have their point of maximum intensity over other portions of the præcordia, and are only heard over the pulmonary area on account of their loudness and extent. These bruits are not likely to be mistaken for those that have just been described.

(*C*). **Systolic Bruits over the Aortic Area.**—When a systolic bruit is heard with its point of maximum intensity in the aortic area, which corresponds to that portion of the chest wall overlying the second right costal cartilage, and is conducted upwards into the vessels of the neck, it arises either at the aortic valve or in the ascending portion of the aorta. The chief point in the diagnosis between these two conditions is the character of the aortic second sound. If the bruit be due to changes in the valves causing obstruction, then the second sound will be altered in character, being muffled and sometimes inaudible, as the rigidity of the aortic cusps prevents them closing suddenly in the normal manner. The presence of an aortic diastolic bruit would make quite clear the valvular origin of the systolic bruit. When the bruit is due to changes in the aorta, in consequence of atheroma, dilatation, or aneurysm, and not to aortic obstruction, then the second sound is usually clear. The presence of a pulsating tumour, pulsation in the second right intercostal space without a tumour, or dullness in this region, would suggest an aneurysm and so confirm the diagnosis of the bruit arising in the aorta. A systolic bruit over the aortic area is of frequent occurrence; but for the purpose of diagnosis it must be remembered that such a bruit is rarely due to stenosis, and more frequently results from a progressive sclerosis of the aortic valve without real stenosis, or from changes in the aorta. Before aortic stenosis is diagnosed there should be a loud systolic bruit in the second right intercostal space, together with a systolic thrill, and evidence of hypertrophy of the left ventricle. If the bruit is due to *acute endocarditis*, with vegetations on the semilunar valves, then the left ventricle is not enlarged to such an extent as in aortic obstruction, or in atheroma of the aorta. A *functional bruit* confined to the aortic area is very rare, but may be distinguished by there being no enlargement of the left ventricle, and by the presence of other functional bruits, especially a *bruit de diable*. If marked anæmia exists, either from some primary blood-disease or secondary to a cachectic condition, due to malignant disease, tuberculosis, malaria, a large hæmorrhage, etc., then the diagnosis of a functional bruit is confirmed.

In rare cases a very loud systolic bruit in the aortic area is due to a saccular aortic aneurysm opening into the pulmonary artery or into the superior vena cava; in either case there will generally be a history of acute dyspnœa developing suddenly, together with cyanosis; and when the superior vena cava is opened into in this way there is generally acute œdema of the face, neck, and arms also (*Fig.* 99, p. 208). *X*-ray examination may assist the diagnosis materially in either case.

(*D*). **Systolic Bruits over the Tricuspid Area.**—A bruit heard best over the tricuspid area, which corresponds to that part of the chest wall overlying the lower portion of the sternum, is of diagnostic importance in that it indicates tricuspid regurgitation, which is nearly always due to dilatation of the right ventricle. That the bruit is due to tricuspid regurgitation is confirmed by finding the cardiac dullness extending to the right of the sternum, fullness and pulsation in the veins of the neck, and evidence of failing cardiac compensation, as shown by œdema of the legs, and enlargement and pulsation of the liver. Many bruits, systolic in rhythm and produced at the tricuspid valves, are best audible in the neighbourhood of the cardiac impulse, but they are not conducted outwards into the left axilla like bruits produced at the mitral valve. On the other hand, when a mitral systolic bruit is loud enough, it may be audible in the tricuspid area, but there would not be the signs of passive congestion unless there was general failure of compensation. It should be borne in mind, of course, that tricuspid regurgitation often occurs without producing any bruit at all, so that absence of systolic bruit does not exclude tricuspid leakage.

II.—DIASTOLIC BRUITS.

A diastolic bruit heard over the precordia is always due to organic disease of the heart. If it be present over the aortic area, that is, over the second right costal cartilage close to the sternum, and conducted downwards along the left border of the sternum, and sometimes outwards towards the cardiac impulse, then the bruit is due to *aortic regurgitation*. Sometimes its point of maximum intensity is in the aortic area, sometimes to the left of the sternum in the third intercostal space. Examination of the pulse confirms the diagnosis, for the 'water-hammer' pulse is found only with aortic regurgitation. Capillary pulsation is also present; it is demonstrated by placing a glass slide on the everted lower lip, or by pressing the finger nail so that the proximal half of it remains pink and the other is blanched, or by stroking the forehead firmly with the finger and watching the alternate blanching and reddening of the resultant streak. Capillary pulsation may also be found in cases of marked anæmia, and in the normal person in a Turkish bath. A double murmur is frequently heard over the larger arteries in aortic regurgitation, particularly over the femoral artery, where it is spoken of as *Duroziez's sign*. The first murmur is produced when the vessel is distended with blood, and the second when the blood-pressure suddenly falls on account of the regurgitation.

As the diastolic bruit of aortic regurgitation is frequently associated with a systolic one, the result of aortic obstruction, a 'to-and-fro' murmur is produced which may sometimes be mistaken for pericardial friction sound. In pericardial friction the systolic and diastolic sounds do not commence accurately with the first and second sounds of the heart, are not conducted in the recognized direction of an endocardial bruit, and are altered in intensity by the pressure of the stethoscope. Having decided that the bruit is due to aortic regurgitation, it must be remembered that such a lesion may be the result of:—

1. *A progressive sclerosis* of the aortic valves, being part of a general arterial degeneration, or due to a localized syphilitic lesion.
2. *Endocarditis*, either rheumatic or malignant.
3. *Rupture of a segment*, due to either excessive strain on an already diseased valve, or to malignant endocarditis.
4. *Dilatation of the aortic ring*, secondary to dilatation or aneurysm of the ascending portion of the arch of the aorta.
5. *Congenital malformation*.

The age of the patient helps greatly in the differential diagnosis; if the lesion be found in a child or young adult, the condition is almost invariably the result of endocarditis; if, on the other hand, aortic regurgitation occurs in middle life, it is nearly always due to sclerosis of the aortic valve, especially syphilitic, and the diagnosis is confirmed by finding a positive Wassermann reaction, or degenerative changes in other arteries, chronic renal disease, and considerable hypertrophy of the left ventricle. If the regurgitation be due to dilatation of the aortic ring, it can only be diagnosed when the existence of dilatation or aneurysm of the ascending portion of the arch of the aorta is indicated by dullness in the second right intercostal space close to the sternum, by pulsation or a pulsating tumour in this area, or by an x-ray examination. Sometimes the aneurysm may be situated just above the sinuses of Valsalva, and, while producing aortic regurgitation by causing dilatation of the aortic ring, may give no other physical sign of its presence. It may be very small, and yet may cause sudden death by rupture into the pericardial sac.

A diastolic bruit heard only down the left border of the sternum is practically always due to aortic regurgitation, but on some occasions it may be produced by *pulmonary regurgitation* as the result of endocarditis, dilatation of the pulmonary ring, or a congenital defect. Pulmonary regurgitation is most often secondary to mitral stenosis, due to dilatation of the pulmonary orifice as the result of increased pressure in the pulmonary circulation. The other two forms are very rare, and difficult to distinguish from aortic regurgitation unless there is evidence of enlargement of the right ventricle and not of the left, and there is no 'water-hammer' pulse as in aortic regurgitation.

Diastolic bruits audible at the cardiac impulse are due either to endocarditis of the mitral valve, to mitral stenosis, or to aortic regurgitation. An aortic diastolic bruit is often conducted as far as the cardiac apex and replaces the second sound here; sometimes the diastolic bruit which is heard in the aortic area is lost on being traced down the left border of the sternum, to reappear at the apex. The diastolic bruits of *mitral stenosis* can be distinguished by their appearance later in the diastolic period, and the most common is presystolic—a crescendo murmur ending in a loud slapping first sound. An aortic regurgitant bruit is generally blowing in character, whereas the bruit of mitral stenosis is often rumbling. Early diastolic, mid-diastolic, and late diastolic bruits, occur also in mitral stenosis, but none of these should be mistaken for the bruit of aortic regurgitation, as the latter condition would be associated with hypertrophy and dilatation of the left ventricle, the apex beat being displaced outwards and downwards, even to the sixth intercostal space, and would be confirmed by the characteristic 'water-hammer' pulse. In mitral stenosis without mitral regurgitation there is very little displacement of the apex beat, because the left ventricle is not enlarged. The bruit of mitral stenosis is often associated with a presystolic thrill, whereas that of aortic regurgitation is not.

In order to understand the various bruits which occur in mitral stenosis, the manner in which they are produced must be discussed. They are caused by the blood being forced through the stenosed mitral valves. The two forces which produce this are the contraction of the walls of the left auricle and of the right ventricle. The suction action of the left ventricle during its diastole is probably not sufficient in itself to cause the bruit, but simply helps in the work of the left auricle and right ventricle. The presystolic bruit of mitral stenosis occurs during the end of the ventricular diastole, and corresponds to the systole of the left auricle. A mid-diastolic bruit sometimes occurs in mitral stenosis. This may be the only bruit present, but there may be a presystolic bruit as well, resulting in two distinct bruits during the ventricular diastole. These two bruits may be fused into one when the contractions of the right ventricle and left auricle are vigorous. The mid-diastolic bruit is probably due to the previous contraction of the right ventricle increasing the blood pressure in the lungs and left auricle, and so forcing the blood through the stenosed mitral valve. It varies slightly in its situation in the ventricular diastole in different cases, but whether early or late it is always separate from the previous second sound instead of replacing the latter in the way an aortic regurgitant murmur does. When the force of the contractions of the left auricle begins to fail, or when there is *auricular fibrillation*, as shown by total irregularity of the heart or by an electro-cardiogram (*Fig.* 196, p. 486), the presystolic bruit often disappears. In mitral stenosis there may therefore be a presystolic bruit, or a mid-diastolic bruit, or mid-diastolic and presystolic bruits, or a bruit which occupies almost

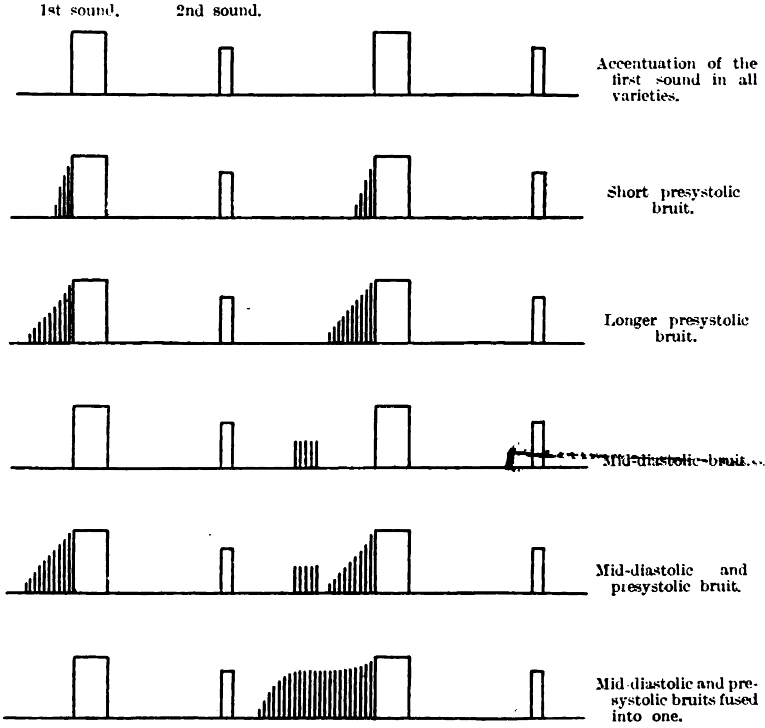
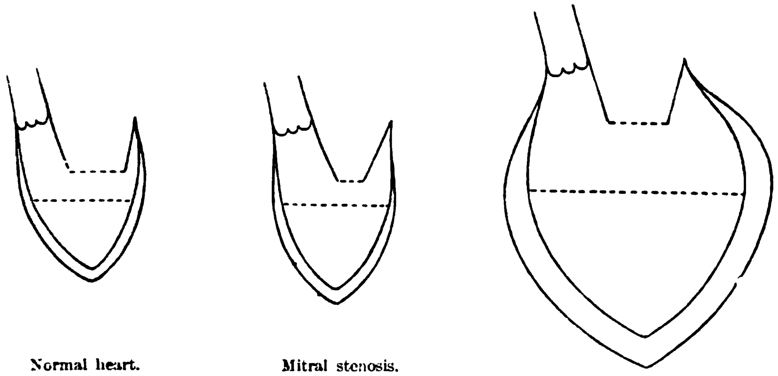

1st sound.
2nd sound.
Accentuation of the first sound in all varieties.
Short presystolic bruit.
Longer presystolic bruit.
Mid-diastolic bruit.
Mid-diastolic and presystolic bruit.
Mid-diastolic and presystolic bruits fused into one.

Fig. 39.—Diagram of the bruits of mitral stenosis (Sawyer's *Physical Signs*).

the whole of the ventricular diastole. With all these bruits the first sound at the apex is usually slapping or thumping in character. This alteration in the first sound may be present without any of the above-mentioned bruits, and is in itself very characteristic of mitral stenosis. In some cases the second sound is reduplicated at the cardiac apex, while in others—and this in the majority of the cases—it is inaudible. The pulmonary second sound is accentuated or reduplicated. The bruit may be accompanied by a mitral systolic bruit, as regurgitation often occurs through the stenosed orifice.

A presystolic bruit in the mitral area is usually due to mitral stenosis, but it also occurs without any mitral stenosis in some cases of *aortic regurgitation* or of *dilatation of the left ventricle*, when the bruit is spoken of as *Flint's murmur*. To distinguish between the latter and the similar bruit of actual mitral stenosis may be difficult; in an uncomplicated case of mitral stenosis the apex beat is normal in position, but when Flint's murmur is present the apex beat is displaced to the left on account of the enlargement of the left ventricle. The presence of aortic disease also points in the direction of the bruit being Flint's murmur. This bruit is often considered to be caused by the vibration of the anterior curtain of the mitral valve as it lies between the regurgitating blood-stream through the aortic orifice and that flowing into the ventricle from the left auricle. If this were the true explanation, Flint's murmur should occur early in diastole instead of being presystolic. Another view is that the blood regurgitating through the aortic orifice lifts the anterior curtain of the mitral valve, and so obstructs the mitral orifice at the end of the ventricular diastole. Neither of these explanations seems to be sufficient to account for the murmur. In a normal heart the ratio of the diameter of the mitral opening to that of the left ventricle is about 1 to 2; in mitral stenosis, on account of the contracted orifice, the ratio may be, say, ½ to 2, the size of the left ventricle remaining the same, and a presystolic bruit occurs. In aortic regurgitation, although the diameter of the mitral orifice remains the same, yet the diameter of the left ventricle is greatly increased on account of its dilatation. The ratio between the diameter of the mitral opening to that of the left ventricle might be, say, 1 to 4, or exactly the same ratio as occurs in mitral stenosis—a relative mitral stenosis when the size of the mitral opening is compared with that of the left ventricle. The one condition is merely on a larger scale than the other (*Fig.* 40); and as the altered ratio of these two diameters produces in mitral stenosis a presystolic bruit, it is probable that the same ratio, although the factors are on a larger scale, produces in aortic regurgitation a Flint's murmur—which is also presystolic in time. A presystolic bruit is sometimes present without any aortic regurgitation, and without mitral stenosis, but always with an enlarged left ventricle; and this seems to point to the regurgitation of the blood through the aortic valves not taking any direct part in the production of the bruit. The following diagrammatic drawings of the heart are constructed to show the probable mode of production of Flint's murmur:—



Normal heart.
Ratio of diameter of mitral valves and diameter of left ventricle, about 1 to 2.

Mitral stenosis.
Ratio about ½ to 2.
Presystolic bruit.

Dilated left ventricle.
Ratio about 1 to 4; i.e. the same proportion as in mitral stenosis.
Presystolic bruit (Flint's).

Fig. 40.—Diagram to explain the origin of Flint's murmur (Sawyer's *Physical Signs*).

Diastolic bruits are heard only very occasionally over other areas of the precordia, but it is possible for a presystolic bruit to occur in the tricuspid region as the result of tricuspid stenosis; such a bruit is rarely present without valvular disease of the left side of the heart also.

A functional bruit is never diastolic in rhythm; but it is important to distinguish the mid-diastolic bruit of acute endocarditis from the similar bruit of fibrotic stenosis. During endocarditis there is some thickening of the valve-flaps from inflammatory œdema, and this leads to bruits not unlike those of fibrous stenosis. The diagnosis depends upon (1) The development of the bruit under observation: if in a case of acute rheumatism a mid-diastolic bruit is noticed to develop rapidly, it cannot be due to fibrosis, and must result from acute inflammation of the valve; (2) The course of the bruit: if it is due to fibrosis it will persist, if to endocarditis it will change with time, becoming less definite if the endocarditis resolves, more definite if the inflammation goes on to scarring and stenosis; (3) The age of the patient: mitral stenosis does not occur commonly before puberty, so that it is most risky to interpret a diastolic apical bruit in a child as being due to mitral stenosis.

J. E. H. Sawyer.

BULLÆ.—A bulla is literally a water-bubble; it is synonymous with bleb or blister; it differs from a vesicle only in its size, which may be from half an inch in diameter to that of a tangerine orange or more. Almost any vesicular skin disease may be of bullous degree occasionally; there are certain diseases in which bullæ are characteristic; and there are yet other affections in which, although bullæ are not always present, they may occur sometimes in a marked degree. The following are the chief conditions under which bullæ are, or may be, a prominent feature of the case:—

A. CONDITIONS IN WHICH BULLÆ ARE USUAL:—

Pemphigus
Erythema bullosum
Dermatitis herpetiformis
Herpes gestationis
Erythema iris
Epidermolysis bullosa
Pemphigus neonatorum
Cheiropompholyx

Local application of vesicants, such as cantharides, arnica, rhus toxicodendron, croton oil, nitric acid, scalding water, hot solids, or extreme cold, for instance after freezing with carbon-dioxide snow.

Local friction by splints after fractures; or by boots, oars, tools, etc.

B. CONDITIONS IN WHICH TYPICAL BULLÆ MAY OCCUR, THOUGH THEY ARE NOT USUAL:

Erysipelas
Impetigo contagiosa
Iodism
Bromidism
Glanders
Syphilis
Syringomyelia
Gangrene
Frostbite
Raynaud's disease
Scurvy

Extreme œdema from Bright's disease or heart failure

Workers amongst turpentine, chrysarobin, varnish, aniline dyes, and other chemicals; tar products, resin, volatile oils; satin-wood, primula obconica, and some other plant products.

Poisoning by large doses of certain hypnotic drugs, notably veronal and acetanilide, especially towards the end of a fatal case.

The diagnosis is sometimes obvious; for instance, *herpes gestationis*—also known as hydroa gestationis, erythema gestationis, and dermatitis pruriginosa polymorpha recurrens graviditatis—is probable when a bullous eruption develops in a pregnant woman; and the diagnosis is certain if there is a history of former pregnancies each associated with a similar eruption, with complete freedom from the complaint between the pregnancies. The eruption itself is precisely similar to that of dermatitis herpetiformis, described below, and there is generally eosinophilia (p. 99). In most cases the trouble begins in the later months of pregnancy, but tends to develop earlier in each successive pregnancy; and whereas in most cases it subsides rapidly when the child is born, in a few instances it may last into the puerperium, or even develop only during that period. The most troublesome part of the complaint is the itching and irritation, that often amount to actual pain. A person subject to pemphigus or erythema bullosum might develop an attack during pregnancy; but herpes gestationis is excluded if recurrence takes place apart from pregnancy, whilst the occurrence of the bullous eruption solely in association with pregnancy makes the diagnosis obvious.

Bullæ in an infant generally receive the term *pemphigus neonatorum*, but the eruption is not related to ordinary pemphigus, so it is a pity the word pemphigus is employed at all. There are two distinct varieties, namely: (1) That in which the bullæ are chiefly on the hands and feet, one of the manifestations of a severe and generally fatal type of congenital syphilis, in which the eruption appears almost immediately after birth instead of after an interval of days or weeks, as in other cases; and (2) That in which there is an infection of the skin of the nature of impetigo—generally staphylococcal, but in some cases due to less usual organisms such as the *Bacillus pyocyaneus*—producing bullæ instead of the more usual pustules; the latter is an affection of poverty-stricken districts, occurring in more or less epidemic form, sometimes closely related to the practice of a particular midwife, and fortunately rare now-a-days.

Cheiropompholyx may generally be recognized at once. It is a dysidrosis, and the sweat-glands of the palms and soles are most affected, though those of the forehead, chest, and back may sometimes be affected too. As a rule the sweat retained in the glands produces subcutaneous vesicles that are barely larger than sago grains; as the superficial epidermis becomes worn off, the little sweat-cysts reach the surface, a process assisted by the scratching that usually results from the accompanying irritation. After each cyst bursts there is desquamation which may simulate that of scarlatina. The malady occurs in summer weather, or in tropical climates, especially in those who perspire freely.

Blisters produced by vesicants are diagnosed readily when it is known that any application is being used. Difficulty arises mainly in two classes of persons, namely, (1) In those who live in houses upon which the *Rhus toxicodendron* is grown as a Virginia creeper, the nature of the case being discovered usually from the fact that the patient is always affected when at home, and never when away; and (2) In hysterical patients, or in malingerers, who produce the skin eruption surreptitiously. If the latter is suspected, it is generally possible to place the patient under conditions which preclude self-application, when the disappearance of lesions confirms the diagnosis; or the actual vesicant employed may be discovered, liquor epispasticus for instance, or some other preparation of cantharides; croton oil; capsicum; carbolic acid; mylabris; iodine; or one of the strong mineral acids, especially nitric acid.

The relationship of occupation to a bullous dermatosis may become obvious from the way the skin trouble recurs whenever any particular work is resumed; the list above indicates the kind of occupations that are liable to produce it; nearly all these produce bullæ far more seldom than they do a vesicular dermatitis.

Extremely œdematous tissues are easily blistered, and on this account one must be chary of diagnosing anything but simple blisters when bullæ develop upon œdematous legs or other parts in association, for instance, with *Bright's disease*, or in *chronic heart cases* with failing compensation. The same applies to the blebs arising on the skin of *fractured limbs*, and also in the region of a local *gangrene*; or necrosis of the soft parts due to such causes as *frost-bite*, or *Raynaud's disease*, or *scurvy*. The diagnosis in these cases will nearly always be clear enough, and so will it be in cases of simple blisters due to friction.

Having thus excluded the more obvious cases, there remain: pemphigus, erythema bullosum, dermatitis herpetiformis, erythema iris, epidermolysis bullosa, erysipelas, impetigo contagiosa, iodism, bromism, glanders, syphilis, and syringomyelia. Of these, acquired *syphilis* is so seldom bullous that it would not be diagnosed unless there was strong collateral evidence of the nature of the complaint. *Syringomyelia* is rare also, and bullæ occur in but a small proportion of the cases; should they do so they would attract attention from being confined to a local area, the fingers and hands for instance, leaving the rest of the person free. The diagnosis would be confirmed by finding cutaneous sensibility natural, though the patient cannot distinguish pain from touch, or heat from cold, in the affected parts. The cutaneous affections of syringomyelia are known as Morvan's disease. The lesions arise because the skin is insensitive to things that are painful or hot enough to produce sores and blisters.

The patient's occupation may suggest the malady in a case of bullous *glanders*; a horse with which the patient had to do might be known to be affected with the complaint. The skin eruption is sometimes quite a late manifestation of a prolonged and obscure febrile illness when the glanders infection has started internally, for instance in the lungs. The *Bacillus mallei* may be found in direct smears from the contents of the bullæ, or in cultures from them. Bacteriological methods afford the final criterion of glanders.

Both *bromides* and *iodides* may produce various types of skin eruptions. The commonest is simple acne; but there may be a patchy erythema with cutaneous infiltration or nodular swelling studded with yellow points from which thick puriform fluid can be expressed; or a confluent furuncular lesion; or a true bullous eruption or hydroa. The latter is decidedly rare, but its occurrence should be borne in mind, and inquiry made as to any drugs that the patient may be taking; in the case of iodides the urine gives a bluish-green colour with the guaiacum test, though no blood is present, and if there is still doubt a quantity of urine may be evaporated down, and either bromine or iodine detected by ordinary chemical tests. Bromide and iodide eruptions have been recorded in infants at the breast when the mother has been taking the drug without herself presenting any cutaneous symptoms.

Bullous *impetigo contagiosa* is a variety of impetigo. Fluid accumulates in the infected spots so quickly that at first it does not appear to be purulent, but rather to take the form of big vesicles or bullæ. These often become pustular, and as they dry up the crusts over them have a characteristic yellow honey-like appearance. The condition can be diagnosed, as a rule, from the fact that other parts of the body present the typical lesions of ordinary impetigo; there may be other patients affected in the same house or school, and the condition is as readily curable by antiseptic measures as is impetigo. There is a very rare and extremely grave disease described as *impetigo herpetiformis* in pregnant women; but this seems to be an aggravated form of dermatitis herpetiformis or herpes gestationis become purulent and contagious. It is found in Austria, but not, apparently, in England.

Erysipelas is a familiar cause of bullæ, and when blebs are present upon the typical tender, slightly raised, and well demarcated red skin at the height of the affection, in association with the constitutional symptoms and pyrexia, there can seldom be difficulty in the diagnosis. It is when the erysipelas is subsiding or has subsided, whilst the bullæ, or the remains of them, are still obvious, that difficulty might arise. Streptococci may be detected bacteriologically.

If all the above conditions can be excluded, and the patient is suffering from a disease of which bullæ with more or less erythema are the chief manifestation, then the diagnosis has been narrowed down to one or other of the following: pemphigus, erythema bullosum, dermatitis herpetiformis, erythema iris, and epidermolysis bullosa; there is evidence to show that these are closely related in some respects, the different names applying to affections that differ more in type than kind. If the patient develops bullæ on various parts of the trunk and limbs without any erythema, or at any rate without any erythema until the bullæ have been present a longer or shorter time, the condition is described as *pemphigus*. If the bullæ develop, not on normal-looking skin, but upon places where there has already been erythema, associated with more or less itching, or even pain, before the bullæ develop, and if the whole eruption consists of this combined condition of erythema and large bullæ, the name used to designate it is *erythema bullosum*. If the bullæ tend to dry up at their central parts and then to be followed by a secondary ring of vesicles or blebs around the original one, these secondary vesicles being followed in turn by others upon a yet larger ring outside them, the condition is referred to as *herpes iris* or as *erythema iris*, according as there is little or much erythema before the first vesicles or bullæ appear. When the bullæ are apt to develop on any part of the body from a degree of rubbing or scratching which in the ordinary individual would be quite unlikely to produce blisters, this undue tendency to blister formation from what ought to be inadequate causes is spoken of as *epidermolysis bullosa*, a condition which may persist throughout life without necessarily leading to any other untoward symptoms; it is probably related to factitious urticaria. *Dermatitis herpetiformis* is a polymorphous eruption, of which bullæ form but a part; the trouble begins with itching of the skin, and more or less general disturbance, part of which arises from the loss of sleep entailed by the irritation. In various parts of the body or limbs erythematous and urticarial patches supervene, some of which subside without further development, whilst upon others clusters of vesicles soon appear. Many of the clusters contain twenty or thirty vesicles upon a single inflamed base; some, fewer vesicles of larger size; others develop into typical blebs varying in area from that of a sixpence to that of a half-crown. No region of the body is exempt. The characters of the lesion are precisely similar to those found in pregnant women suffering from herpes gestationis, but there must be a difference in causation, for the latter, though it occurs with every successive pregnancy in the same woman, remains in complete abeyance between the pregnancies, whilst dermatitis herpetiformis—Dühring's disease or hydroa—may occur in either sex and at almost any age, though it is less common in children than in adults. It is probably due to the action of some poison circulating in the blood, derived perhaps from the food in some cases; it is possible for two persons to be taken ill after partaking of the same food, one with acute gastro-intestinal symptoms, such as diarrhœa and vomiting; the other with acute pemphigus; it looks, therefore, as if pemphigus and its allies may be related to the acute urticaria that is so familiar in certain cases of shell-fish poisoning.

Any one of the bullous dermatoses may be either acute, subacute, or chronic; in any of these degrees there may be practically no constitutional disturbance on the one hand, or the patient may be so ill with pyrexia and anorexia as to require to stay in bed; not a few such cases prove fatal. In all the bullous dermatoses the eruption may be restricted to the cutaneous surface; but the bullæ may also occur upon mucous membranes, especially of the mouth, palate, œsophagus, nose, colon, rectum, and vagina. Even when temporary recovery has taken place there is a tendency for subsequent attacks to occur. There is also a tendency to hæmatoporphyrinuria during the exacerbations.

Finally, it may be emphasized that although it is often stated as a general rule that many skin diseases may be associated with eosinophilia, as a matter of fact few skin diseases other than the bullous dermatoses produce any marked degree of eosinophilia, so that a differential leucocyte count may afford valuable diagnostic evidence. The absence of eosinophilia by no means excludes pemphigus or erythema bullosum or any other bullous dermatosis, but the presence of eosinophilia in a doubtful case increases the probability of the condition being one of these; it is noteworthy that whereas eosinophile cells may abound in the contents of the natural bullæ, those which occur in a blister produced artificially in the same case present no such eosinophilia. *Herbert French.*

BUZZING IN THE EARS.—(See TINNITUS, p. 722.)

CACHEXIA literally means 'a bad habit,' and is an ill-defined term used to include almost any depraved condition of the body in which nutrition everywhere is defective. It is generally applied to patients who exhibit at the same time progressive loss of weight, and change of complexion in the direction of sallowness or actual anæmia. (See WEIGHT, LOSS OF, p. 768; and ANÆMIA, p. 20.) The word is generally prefixed by a qualifying adjective, such as *cancerous*, *syphilitic*, *malarial*, *tuberculous* cachexia, the diagnosis being indicated by other symptoms or by the history. Other varieties of cachexia that may be given special mention, and which, if they are borne in mind, are not as a rule difficult of diagnosis, are *C. splenica*, including blood diseases such as leucocythemia, in which with progressive loss of weight and anæmia there is enlargement of the SPLEEN (p. 628); *C. uterina*, from chronic non-fatal lesions of the uterus or other pelvic organs, notably leucorrhœa, chronic endometritis, or fibroid tumours; and often accompanied by brown disfiguring pigmentation (chloasma uterinum), especially on the forehead and round the eyes; *C. parasitica*, due to infection by the more serious intestinal or other parasites, especially *Ankylostomum duodenale*, *Bothriocephalus latus*, *Bilharzia hœmatobia*, and *Trichina spiralis*; *C. chlorotica*, a synonym for chlorosis; *C. mercurialis*, attributed to the effects of mercury, though perhaps really due to the syphilis for which the mercury has been given; *C. exophthalmica*, sometimes associated with Graves's disease; *C. palustris*, or marsh cachexia, due either to actual malaria or to constant living in unhealthy, damp surroundings; *C. alkalina*, the bad health caused by taking large quantities of alkalies for a long period, and evidenced by pallor, breathlessness, emaciation, and anæmia; *C. aquosa*, also called *pica*, and *C. africana*, a term given to an anæmic condition leading to serous effusion, and often accompanied by perversion of appetite, seen in hot climates and especially among negroes; it has received many names, such as white tongue, stomach disease of negroes, negro cachexy, intratropical anæmia, dirt-eating disease; doubtless many different disorders have been included under this name, including the results of malaria or of intestinal worms; *C. renalis*, which results from prolonged albuminuria, especially in subacute tubal nephritis; *C. scorbutica*, a condition formerly described as associated with rickets, though more likely related to the infantile scurvy of Barlow, nutrition being impaired, the head and upper part of the body perspiring profusely during sleep, anæmia developing, and the patient being intolerant of bed-clothes owing to tenderness or actual painfulness of the bones from subperiosteal hæmorrhages; there may or may not be bleeding gums; *C. saturnina*, from chronic lead poisoning.

Herbert French.

CAMMIDGE'S PANCREATIC REACTION.—The improved pancreatic reaction depends upon the fact that when the urine of a patient suffering from pancreatic inflammation is hydrolysed by boiling with dilute HCl, a substance having the reactions of a pentose is set free, and may be recognized by conversion into its osazone crystals by treatment with phenylhydrazine, a golden yellow flocculent deposit of flexible hairlike crystals forms, arranged in microscopic sheaves, readily soluble in dilute sulphuric acid. The appearance and solubility of the crystals are very characteristic, but as glycuronic acid, which is set free to a greater or less extent in all urines during the hydrolytic process, also forms a crystalline compound with phenylhydrazine, it is removed by treating the still acid urine with tribasic lead acetate, after the excess of hydrochloric acid has been neutralized with lead carbonate. The lead that goes into solution has also to be removed by converting it into an insoluble sulphide or sulphate before the phenylhydrazine test is applied.

The results of clinical experience and many animal experiments have demonstrated that a positive 'pancreatic' reaction is strong presumptive evidence of a disturbance of function and of active degenerative changes in the pancreas. In most cases these are consequent on inflammation, either acute or chronic, but in a few instances a positive reaction seems to rise from abnormal physiological activity. The latter may, however, be neglected for all practical purposes, for it is not associated with symptoms suggestive of pancreatic disease.

It has been pointed out repeatedly that the pancreatic reaction is not pathognomonic of pancreatitis, and the writer must again insist that the results of the test must be considered in conjunction with the clinical symptoms and the evidence to be obtained by a complete analysis of the urine and fæces. By doing so one can not only obtain confirmation of the indications given by this special method of examination, but also infer the probable cause of the changes in the pancreas, which is a most important point, for pancreatitis is rarely, or never, a primary disorder, but is usually secondary to an ascending catarrh from the duodenum, gall-stones in the common bile-duct or in the ampulla of Vater, invasion of the pancreas by a duodenal or gastric ulcer, malignant disease either primary in the pancreas or secondary to some other organ, back-pressure from disease of the heart or lungs, arterio-sclerosis, alcoholism and cirrhosis of the liver, syphilis, tubercle, influenza, typhoid fever, mumps, etc., etc. In many of these the clinical signs and symptoms alone are sufficient to indicate the cause of the pancreatitis, but in others they are so indefinite or obscure that it is only by considering the results of a complete quantitative and qualitative analysis of the urine, and fæces also, that a correct diagnosis can be arrived at.

A single negative pancreatic reaction does not exclude chronic pancreatitis, or rather the results of inflammation of the pancreas, for the reaction is only given when there are active degenerative changes in the gland at the time when the urine is being excreted. Cirrhosis of the pancreas due to past inflammation does not, therefore, cause a reaction after the inflammation has subsided. Cancer of the pancreas too is associated with a positive reaction in only about 25 per cent of cases, the presence of the growth being apparently unattended by any inflammatory changes in the pancreas in the remaining 75 per cent.

As the ordinary method of carrying out the test is interfered with by the presence of sugar in the urine, a modification, in which a hydrochloric acid solution of the precipitated lead salt from the basic lead acetate solution is submitted to steam distillation, has been devised (Cammidge, *Glycosuria and Allied Conditions*, p. 274). The quantity of furfuraldehyde formed is determined by treating the distillate with sodium nitrite and then titrating with iodine solution. This method, which can also be used for sugar-free urines, gives quantitative results by which one case can be compared with another, and the course of any one be followed accurately. Numerous experiments have shown that the 'iodine coefficient' of normal urines is nil, and that even when simple digestive and hepatic disturbances are present it rarely exceeds 1·0 to 1·5 per cent. When there is inflammation of the pancreas the iodine coefficient rises to ten, twenty, or more per cent, with a total for the twenty-four hours urine of 100, 200, or over. As one would expect from the qualitative test, many cases of cancer of the pancreas give a negative iodine coefficient, but in some 25 per cent similar readings to those obtained in cases of pancreatitis are obtained.

Other points to be noticed in examining the urine from suspected cases of pancreatic disease are :—

1. The presence of calcium oxalate crystals (see OXALURIA, p. 423) in the centri-

fugalized deposit ; these are met with in 63 per cent of cases of chronic pancreatitis, or 73 per cent if jaundiced cases are excluded.

2. A pathological excess of urobilin (see *Plate XXXIV*, *Fig*. 12, p. 748) ; this is a very constant indication of cholangitis, and a particularly useful sign of gall-stones in the common bile-duct, whether accompanied by jaundice or not.

3. A well-marked indican reaction : pointing to a catarrhal condition of the intestinal mucous membrane, with abnormal putrefactive changes in the contents of the intestine, and possibly a duodenal or gastric ulcer.

4. Bile pigment in the urine : showing that there is some obstruction to the free flow of bile into the intestine, due to impacted gall-stones, gripping of the common bile-duct by the inflamed head of the pancreas which surrounds the duct in 62 per cent of cases, malignant disease of the head of the pancreas, or a growth in the common bile-duct.

For the purposes of a further differential diagnosis, the results of a qualitative and quantitative analysis of the fæces are most important. In carrying out the analysis the points to be noticed particularly are :—

1. The presence or absence of stercobilin ; in gall-stone obstruction, traces at least are nearly always met with, whereas in malignant disease of the head of the pancreas total blocking of the duct is the rule, although the soft growths occurring primarily in the common duct usually allow some bile to filter through so that traces of stercobilin are met with in the fæces.

2. The percentage of unabsorbed fat : in cancer of the pancreas this is always very high, 70 to 80 per cent ; it is usually somewhat less in growths of the common duct, averaging 60 to 70 per cent, and varies from a subnormal percentage in early catarrh of the pancreas to as much as 50 or, rarely, even 80 per cent in advanced chronic pancreatitis.

3. More important still, however, is the relation of the ‘ unsaponified ’ to the ‘ saponified fats,’ for whereas the former are in excess in diseases that interfere with the digestive functions of the pancreas, such as cancer of the gland and advanced chronic pancreatitis, the latter predominate in obstruction of the common duct by gall-stones, without pancreatitis, and in malignant growths not involving the pancreas. It must be borne in mind, however, that, owing to the abnormal activity of fat-splitting bacteria in the lower bowel, such as is met with in some cases of intestinal catarrh, an excess of saponified fat may be found in cases of chronic pancreatitis where the disease is due to an infection spreading from the duodenum along the pancreatic ducts. A similar excess is often met with in early catarrhal pancreatitis, owing probably to an increased flow of pancreatic juice analogous to the salivation met with in parotitis.

4. Microscopical examination of the fæces for fat globules, fatty acid crystals, undigested muscle fibres, and connective tissue, should not be omitted : a large excess of fat globules and free fatty acid crystals, with numerous isolated undigested muscle fibres, pointing to cancer of the pancreas or advanced cirrhosis of the gland, whereas muscle associated with connective tissue points to defective gastric digestion.

5. An acid reaction of the fresh stool is in favour of a diagnosis of pancreatic disease ; in simple gall-stone obstruction, the fæces are usually alkaline.

6. Occult blood, when constantly present in the fæces (see p. 81), is suggestive of malignant disease or, more rarely, advanced pancreatitis, in which it is now well known that there is a hæmorrhagic tendency ; while the discovery of blood intermittently points to a gastric or duodenal ulcer, which may be invading the pancreas and setting up pancreatitis.

By carefully considering all the facts thus obtained, and interpreting them in the light of the clinical signs and symptoms, it is possible, not only to diagnose correctly the existence of disease of the pancreas, but also to arrive at a satisfactory conclusion as to its probable cause. Affection of the pancreas is much commoner than is generally supposed, and many trying cases of chronic indigestion, recurring or persistent jaundice, and obscure affections of the upper abdomen would be explained, and satisfactorily treated, if investigated as above. Undiagnosed, and consequently untreated, pancreatitis is probably the most common cause of diabetes. If this were more widely recognized much might be done to stay the further increase of that disease.

P. J. Cammidge.

CARDIAC BRUITS.—(See BRUITS, CARDIAC, p. 89.)

CARDIAC IMPULSE DISPLACED.—(See HEART IMPULSE, DISPLACED, p. 297.)

CARDIAC THRILLS.—(See THRILLS, PRECORDIAL, p. 720.)

CASTS IN THE URINE.—(See ALBUMINURIA, p. 6.)

CEPHALALGIA.—(See HEADACHE, p. 293.)

CHARCOT-LEYDEN CRYSTALS were at one time supposed to consist of spermin, but now there is considerable doubt as to their exact chemical nature. Their chief importance from a clinical point of view is that they are more common in certain conditions than in others. They may be found either in the sputum, the blood, or in the stools. They need the high power for their detection. Each resembles an elongated diamond with clear-cut edges, without colour, but with a slightly yellow appearance when seen obliquely. They stain with eosin, and are soluble in hot water, in mineral acids, and in alkalies, so that for their detection a fresh specimen is required.

In the *sputum*, they are commoner in *asthma* than under any other circumstances—true spasmodic asthma, such as also gives rise to CURSCHMANN'S SPIRALS (p. 153), and eosinophile corpuscles in the sputum. In determining whether a given case is one of paroxysmal dyspnœa, cough, or bronchitis on the one hand, or true asthma complicated by bronchitis upon the other, numbers of Charcot-Leyden crystals in a fresh specimen of sputum are evidence in favour of the latter. Small numbers of the crystals may be found in *bronchitis* and in association with *bronchiectasis*, but in true asthma their numbers may be quite large.

The occurrence of Charcot-Leyden crystals in the *blood* is of little diagnostic value. They are seldom found in fresh blood ; but when the latter has stood for some time in bulk they develop, particularly in *leukæmia*. Some have tried to draw important clinical deductions from the development of these crystals in blood, but it is doubtful whether they really have any significance of value.

In the *stools*, Charcot-Leyden crystals have been found in a great variety of diseases, but whether or not clinical deductions can be drawn from their presence is doubtful. It is stated that, when they abound, the patient is probably suffering from an *animal parasite* : but it affords no indication of the nature of the parasite present. Their occurrence should lead one to examine the fæces for parasites or their ova with even greater care than usual.

Herbert French.

CHEST, BLOODY EFFUSION IN.—When, on needling a pleural cavity containing fluid, this fluid is found to be obviously blood-stained, the fact is suggestive of one of three things : either the pleurisy has been exceedingly acute ; or the chest has already been tapped not long previously, so that there has been hæmorrhage into the residual fluid : or there is malignant disease of the pleura.

The history of the case may at once indicate whether the inflammation is very acute or not ; the symptoms would have been of short duration, with much pyrexia, whilst the fluid itself would be of high specific gravity, would contain a large amount of albumin, would probably coagulate spontaneously, and microscopically would exhibit numerous polymorphonuclear cells and lymphocytes, and abundant red corpuscles, but no particles of growth in the centrifugalized deposit.

If blood is found in pleuritic fluid at a second tapping, when it was not present at the first, the fact is by itself of little value in differential diagnosis, for the bleeding has probably been caused by the act of paracentesis.

When there is a new growth, and the effusion contains obvious blood at a first tapping, it is likely that the symptoms will have been of gradual onset, without marked pyrexia ; the diagnosis is sometimes cleared up by finding fragments of new growth in the centrifugalized deposit. It is of course by no means every case of malignant disease affecting the pleuræ that produces a blood-stained effusion ; but when the effusion is blood-stained at a first tapping, in a case that has not run a very acute course, one should be very suspicious of new growth. In not a few such cases there have also been comparatively large numbers of coarsely granular eosinophile corpuscles in the effusion. It is often impossible to be sure of the diagnosis until the progress of the case has been watched, sometimes for weeks ; pleural effusion, like that of a simple case, may be the only sign for a long time, but sooner or later one will expect to find evidence of obstruction to a bronchus or to the superior vena cava as the growth spreads in the mediastinum, and occasionally the peculiarity of the shadows seen with the x-rays point to the nature of the case (*Fig.* 42, p. 105.) *Herbert French.*

CHEST, DEFORMITY OF.—(See Deformity of the Chest, p. 166.)

CHEST, PAIN IN.—(See Pain in the Chest, p. 430.)

CHEST, PUS IN.—When, on needling the chest, pus wells up into the exploring syringe, it is probable that the patient has an *empyema*. Other lesions may simulate empyema, however, and even when empyema is actually present it is important not to let the diagnosis rest at that; but rather to regard it as a symptom and try to diagnose its cause. It by no means follows, of course, that when the exploring syringe fails to detect pus, an empyema is not present, for sometimes it is situated either between the lower lobe and the diaphragm, in front of the lung or between the lobes, or in some other position in which it is difficult to hit it off with the needle. When pus is found but the amount is only quite small, there may be doubt as to whether it came from an empyema outside the lung, from a bronchus, or an *abscess cavity in the lung substance*. The nature of the case may remain undecided until a subsequent puncture, or a resection of a rib, conclusively discovers intrapleural pus. Even when pus wells up in the exploring syringe, it is possible to mistake for empyema a collection of pus which is below the diaphragm. A *subdiaphragmatic abscess* and *an abscess within the liver* are the two conditions most liable to simulate empyema in this way. If, however, the history, the symptoms, and the physical signs do not serve to distinguish between these different conditions, it will still be clearly necessary to evacuate the pus, and the surgeon's finger inserted through the wound will be able to feel whether the diaphragm is above or below the collection. Even then there is one possible source of error, namely, when there is pus both above and below the diaphragm. A subdiaphragmatic abscess, secondary perhaps to appendicitis upon the right side, or to a leaking gastric ulcer upon the left, may have infected the pleura through the diaphragm, causing first a serous and then a purulent effusion, separated from that below the diaphragm merely by the thickness of that muscle. It may be very difficult indeed to be sure of this condition, even at the time of operation, the nature of the case not being fully cleared up until, when one of the pus-containing cavities has been evacuated, the abnormal physical signs persist, and a second collection of pus, above or below the diaphragm as the case may be, is found at a subsequent exploration. The *x*-rays may be of considerable assistance sometimes in showing whether the diaphragm is above the pus or below it.

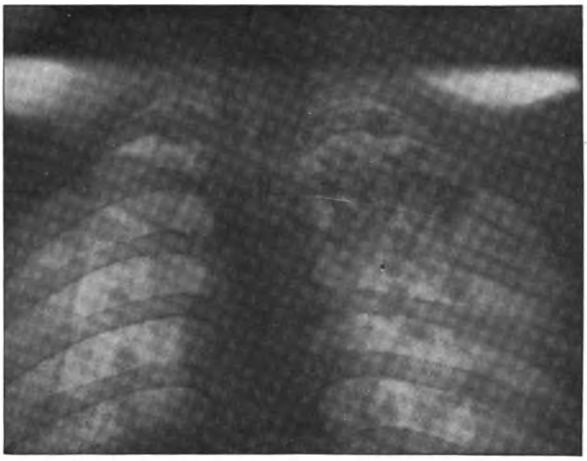


Fig. 41.—Skiagram showing the mottling of the lung apices produced by phthisis. Posterior view. (*By Dr. A. C. Jordan.*)

If, however, the physical signs, symptoms, and the result of needling, all prove conclusively that the chest contains an empyema, it is still necessary to decide as far as possible the nature of the latter. Its commonest cause is pneumococcal infection, nearly always preceded by lobar pneumonia in adults, in children sometimes by bronchopneumonia, but not infrequently arising insidiously. It is probable that many of the so-called latent empyemata of children have really been preceded by undiagnosed bronchopneumonia. Difficulty often arises from the fact that the amount of pus present is not great, so that though it compresses the lung sufficiently to render the alveoli airless, the bronchial tubes still remain patent, and there is no complete dullness at the base or wherever the empyema may be; and over the affected area there may be bronchial breathing and crackling râles, instead of the absence of breath-sounds and of voice-sounds that usually accompanies empyema in adults. If there is doubt as to the nature of the empyema as judged by the history, bacteriological examination of the pus will often indicate its origin. The commonest organisms to be found are pneumococci, streptococci, and staphylococci, though *Bacillus coli communis*, typhoid bacilli, and the *Bacillus pyocyaneus* also occur, and even other organisms may be present in some instances. The mode of infection is generally either viâ the lung, or from beneath the diaphragm; and careful inquiry into the history and symptoms will generally indicate which of these two paths has been the more likely. When infection from any peritoneal condition such as appendicitis, leaking gastric or duodenal ulcer, infected gall-bladder, or subdiaphragmatic, perinephric, or hepatic abscess. can be excluded, when there has been no injury to the chest with broken rib, or a wound communicating with the exterior, and when there is nothing to indicate whether the infection has succeeded pneumonia or is itself pneumococcal, suspicion will arise that the patient has been suffering from phthisis, which has caused a pleurisy which was at first non-purulent. but which became converted into an empyema as the result of secondary infection with pyogenic organisms, especially if there is a tuberculous family history, or if the patient has himself been weakly for some time. The sputum should be examined with particular care. and *x*-ray examination is often helpful; for even when the compression of the lung by empyema has led to marked opacity at the base, it may still be possible to make out that apical mottling which is almost pathognomonic of phthisis (*Fig.* 41).

Rarer causes of empyema than those mentioned above will generally have been accompanied by other symptoms, or by a history which suggests the nature of the individual case.

Herbert French.

CHEST, SEROUS EFFUSION IN.—When exploratory needling of the chest discovers clear serous fluid in the pleural cavity, it is important to regard the fact merely as a symptom, for there are many different causes to which it may be due, and, whenever possible, one should decide what is the actual cause in each particular case. In the first place, the effusion may be either inflammatory or merely a transudate; the *pleuritic* must be distinguished from the *pleural* effusion. Clinical points indicating that the effusion is inflammatory rather than passive would be: its being unilateral, not bilateral and symmetrical; and the non-existence of the more common causes for passive effusion, particularly chronic heart failure or nephritis with general anasarca. Physical, chemical, and microscopical analyses of the fluid may also serve to indicate whether the effusion was active or passive (see ASCITES, p. 48). There are cases, of course, in which there may be doubt, but it is generally easy to determine whether the effusion is due to pleurisy or not. Pleural effusions not due to pleurisy occur late, and the diagnosis will have been made already from the existence of prominent symptoms earlier in the disease, for instance, ALBUMINURIA (p. 4), ORTHOPNŒA (p. 418), ŒDEMA (p. 411), and so forth.

Pleuritic effusion, on the other hand, may be the first and most prominent symptom in the case, and it is not always easy to determine its cause. It should be an invariable rule to have the effusion examined microscopically, both for cells and for micro-organisms, and sometimes to have guinea-pigs injected with it in order to see whether in six weeks' time the inoculated animals have developed general tuberculosis. The commonest cause for apparently idiopathic pleuritic effusion is latent or undiagnosed *tuberculosis* of the lung; there may be no sputum; *x*-ray shadows may be indeterminate; there may be no abnormal apical physical signs; there may be too few bacilli for them to be detected on direct examination of the deposit, even when it has been most carefully centrifugalized, and yet inoculated guinea-pigs may develop typical tuberculosis and thus indicate the nature of the pleurisy.

Intrathoracic new growth, whether of the mediastinum, lung, or pleura, is fortunately uncommon; but when it occurs, the symptoms and physical signs to which it gives rise are often very difficult to interpret. The growth may obstruct a bronchus and give all the physical signs of fibroid lung, with or without bronchiectasis; it may cause a big mass, bodily displacing the lungs and heart; it may cause multiple nodules which, unless they obstruct the superior vena cava and produce obvious varicose veins on the chest wall, may give rise to no very definite signs or symptoms at all; or, what is not at all infrequent, the growth may lead to pleuritic effusion which may at first seem to be simple, or even be taken to be tuberculous, growth not being suspected until the rapid recurrence of the effusion, repeated tappings, and rapid downhill course of the disease ultimately suggest its nature.

Microscopical examination of the centrifugalized deposit of the pleuritic fluid sometimes leads to the detection of particles of new growth which clinch the diagnosis, whilst if the fluid in a case which is not absolutely acute is blood-stained at a first tapping, this by itself is highly suggestive of neoplasm (p. 102). The x-rays often assist materially in making the diagnosis (***Fig.*** 42).

Acute rheumatism is a common cause of pleurisy with effusion, particularly between the ages of five and twenty. It may occur when there have already been joint-pains, or other symptoms of acute rheumatism, such as chorea, recurrent tonsillitis, pericarditis, endocarditis followed by valvular disease, skin affections such as erythema multiforme, erythema nodosum, peliosis rheumatica, or subcutaneous nodules. In such cases the diagnosis is not difficult; it is less easy when the pleuritic effusion is itself the main symptom. The youth of the patient, the absence of anæmia or of previous ill-health, the absence of abnormal apical lung signs, of a family history of phthisis, the presence of a cardiac bruit, the occurrence of heart disease, acute rheumatism, or chorea in other members of the same family, the rapid onset of the disease, and the almost equally rapid resolution of the effusion, are points in favour of acute rheumatism rather than tuberculosis. When in doubt, the negative results of guinea-pig inoculation would point in the same direction, and von Pirquet's skin reaction would be negative. There are, however, many cases of pleuritic effusion in young people, in whom it is impossible to allocate the cause either to rheumatism or phthisis, and such cases are sometimes spoken of as 'simple'; doubtless most of these are either tuberculous or rheumatic, many ultimately proving to be the former.

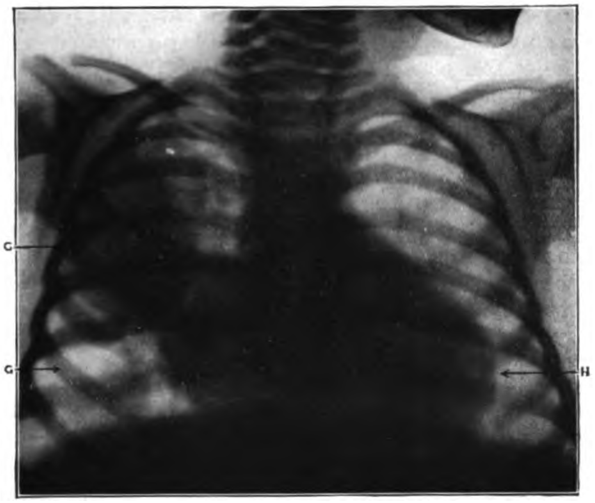


Fig. 42.—Skiagram showing sarcoma of the right lung secondary to sarcoma of a kidney. The patient was a child, aged 1½ years. GG. Masses of new growth. H. Heart. The lower mark G points in the direction of the lower mass of growth, but the line has not been prolonged so far as to the shadow of the growth.

(*Skiagram by Dr. C. Thurstan Holland.*)

Pneumococcal lesions of the lung generally produce pleurisy; *lobar pneumonia*, indeed, never occurs without it, though *bronchopneumonia*, even when it is pneumococcal, often does. It is also possible for pneumococcal pleuritic effusion to occur without definite lobar pneumonia or bronchopneumonia preceding it—primary pneumococcal pleurisy, the diagnosis being confirmed by the discovery of pneumococci in the fluid. It is difficult to say where pneumococcal serous effusion stops, however, and pneumococcal empyema begins, the two merging into one another, and the same case often exhibiting clear fluid at one exploration, cloudy fluid a few days later, and pus later still.

Bright's disease may cause either a passive effusion from heart failure in chronic cases, or simple accumulation of œdema fluid in the pleural cavities without heart failure in cases in which the general œdema of Bright's disease is extreme; or actual pleurisy with serous effusion, probably the result of intercurrent infection by some organism, corresponding with the peritonitis with ASCITES (p. 43) and the pericarditis that may also occur in these cases. The diagnosis will be indicated by the ALBUMINURIA (p. 4), associated with renal tube-casts; and if there is bilateral effusion without universal œdema, but with signs of heart failure in the form of orthopnœa, œdema of the legs, and perhaps ascites, the effusion is passive; it belongs to the second category if there is universal œdema; whilst if the effusion is inflammatory it will probably be unilateral, or else more marked in one side of the chest than in the other. In a few cases an extensive pleuritic effusion in a middle-aged or elderly person is the first indication that there is anything renal the matter, the diagnosis of red granular contracted kidney being confirmed by the urinary changes, big heart, ringing aortic second sound, high blood-pressure, or by albuminuric retinitis.

Any of the severe blood diseases, particularly *Hodgkin's disease, lymphadenoma, leukæmia, splenic anæmia, pseudo-leukæmia infantum*, and to a less extent *pernicious anæmia*, may give rise to inflammation of any of the serous membranes, and thus lead to ascites, pericarditis, or pleurisy with effusion. The latter is seldom an early symptom in such cases, however, and the diagnosis will generally be known already from the presence of pronounced ANÆMIA (p. 20), enlargement of the LYMPHATIC GLANDS (p. 376), or enlargement of the SPLEEN (p. 628), with or without pathognomonic blood-changes already discussed under these various headings.

Pleuritic effusion may sometimes be secondary to *infection of the pleuræ from inflammatory changes below the diaphragm*; thus *appendicitis* may lead to micro-organisms tracking up behind the ascending colon to reach the diaphragm, there perhaps producing a small subdiaphragmatic abscess, or a local inflammation which, stopping short of pus formation, ultimately subsides. The bacteria in contact with the lower surface of the diaphragm can pass through the latter and infect the pleura without there being any actual perforation of the diaphragm; it is noteworthy that passage of micro-organisms in the reverse direction is so rare as almost to be negligible; acute peritonitis often produces acute pleurisy, but the latter, or even empyema, seldom produces peritonitis. Any inflammatory mischief below the diaphragm may lead to dry pleurisy, pleuritic effusion, or empyema. One need not enumerate all such causes, but they should be borne in mind as possibilities. There may have been acute general peritonitis, or a more local inflammation of the peritoneum tracking in the manner already described in connection with appendicitis. This is possible when there is leaking from a gastric or duodenal ulcer; local infection from the gall-bladder; pyosalpinx; pelvic peritonitis due to whatever cause; perinephric inflammation secondary to renal calculus or injury, acute ascending nephritis, tuberculosis of the kidney; hepatic abscess or other inflammatory changes in or about the liver, such as infective cholangitis, suppurative pylephlebitis, or the softening and breaking down of new growth, gumma, or hydatid cyst. When the possibility of a pleuritic effusion being secondary to an abdominal lesion of some kind is borne in mind, the diagnosis of the case is generally indicated, at least approximately, by the preceding history and symptoms. If the fluid obtained smells as though it were infected with *Bacillus coli communis*, this would be an additional argument in favour of some subdiaphragmatic cause.

Infarction of the lung, whether thrombotic or embolic, is apt to cause dry pleurisy; but if the infarct has been extensive, or is due to embolism from some septic source such as a lateral sinus or jugular vein thrombosis in connection with otitis media, or other similar lesions causing venous clotting, the inflammation of the pleura tends to go further and produce an effusion which, at first serous, may later become purulent. The diagnosis is sometimes obvious; but when after an operation, perhaps for excision of an inflamed appendix, the patient a few days later develops pleurisy with effusion, it may not occur to one that a possible explanation of the trouble is that more than one systemic vein in the region of the right iliac fossa has become inflamed and thrombosed, and that portions of the clot have been detached and carried to the lung, where multiple infected emboli have led to pleurisy and serous effusion, without going so far as to produce either abscess in the lungs or empyema. Should hæmoptysis occur in such cases, as it sometimes does, phthisis may be feared; but it will be excluded by the absence of tubercle bacilli on repeated examination of the sputum.

Occasionally the fluid obtained on needling the chest is distinctly *chylous*, in which case the first suspicion to be aroused is that there has been some interference with the thoracic duct, either by *injury* or by an *intrathoracic new growth*. Sometimes, however, this rare symptom is due to remoter causes, such as *chronic nephritis* or *leukæmia*, just as these may occasionally produce chylous ascites (see p. 50); in a few instances a chylous effusion into the chest has cleared up after tapping, no ascertainable cause being found.

Multiple serositis or **polyorrhomenitis** is a term used to express any condition in which there is recurrent inflammation and serous effusion into more than one serous membrane. It generally affects the peritoneum, pericardium, and both pleuræ either simultaneously or successively. It is not a disease in itself, so that the differential diagnosis of the cause of the combined effusions has to be made upon the same lines as that described for each separately. There are cases in which, even when the patient dies, the precise nature of the multiple serous inflammations and effusions is obscure ; it is very possible that the original microbial cause has disappeared, leaving behind it so much fibrotic thickening of the membranes that even the normal secretions are unable to drain away as fast as they should. The result is that recurrent tapping at comparatively short intervals becomes necessary, and the patient ultimately dies of exhaustion, nothing being found post mortem except fibrous thickening of the peritoneum, pericardium, and pleuræ, with more or less extensive perihepatitis, perisplenitis, adherent pericardium, and chronic mediastinitis. The general opinion is that the primary cause in these cases has been either acute rheumatism or tuberculosis. Sometimes secondary malignant disease affects more than one of the serous membranes at the same time, and produces a clinical picture which at first simulates chronic simple polyorrhomenitis ; there are generally symptoms due to the primary growth ; but occasionally, especially in connection with diffuse carcinoma of the stomach—' indiarubber-bottle ' stomach—the primary growth causes no symptoms, and the nature of the multiple serous effusions may be obscure unless particles of new growth can be detected in the centrifugalized deposit, or secondary masses can be found in the liver or lymphatic glands. The left supraclavicular glands should be examined carefully (*Fig.* 17, p. 49). Sometimes the diagnosis is not arrived at until a post-mortem examination is made.

Besides chronic tuberculous, rheumatic, and malignant polyorrhomenitis, a similar condition may be due to Bright's disease or any of the severe anæmias ; the differential diagnosis of the serous effusions to which these may give rise has been discussed above. Careful examination of the blood and urine, together with estimation of the blood-pressure, examination of the optic discs, and routine physical examination of the various body systems, are essential before the correct diagnosis can be arrived at. *Herbert French.*

CHEST, VARICOSE VEINS ON.—(See VEINS, VARICOSE THORACIC, p. 750.)

CHEYNE-STOKES RESPIRATION, or periodic breathing, consists in the occurrence of a series of inspirations, beginning with a hardly perceptible movement increasing to a maximum, and then declining in force and length until they cease in a period of apnœa of some seconds' duration, during which the patient may appear to be dead, but at the end of which a low inspiration, followed by one more decided, and then others of increasing depth, mark the beginning of a new ascending series of inspirations, which in their turn, when the maximum has been reached, become progressively smaller again, to end in another period of apnœa ; and so on with more or less periodicity (*Fig.* 43). The duration of each period varies from half a minute to two minutes or even more. There is a peculiar variety of periodic breathing in which, instead of a waxing and waning sequence, only two or perhaps three rapid deep breaths are made at a time, with long periods of apnœa between them—a variety of periodic breathing which is sometimes spoken of as Biot's.

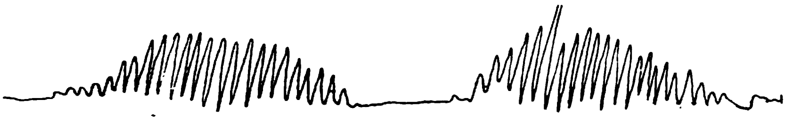


Fig. 43.—Cheyne-Stokes Breathing. A graphic record from a case of arterial degeneration and softening of the medulla oblongata. The two curves were completed in 143 seconds.

Periodic breathing may occur during sleep in the very young and in the very old without there being any actual disease. In other persons Cheyne-Stokes breathing is generally a late phenomenon, having been preceded by other symptoms, particularly uræmic or cardiac ; in a few cases of progressive softening in the medulla oblongata secondary to arterial degeneration, Cheyne-Stokes respiration may be the salient symptom in the case. Broadly speaking, one may classify the chief causes of periodic breathing as follows :—

1. **Arterial, especially with Degenerative Changes in the Medulla Oblongata.**—
   Arteriosclerosis, with or without granular kidney.
   Senile degeneration.

2. **Uræmic,** in cases of :—

- Acute nephritis
- Chronic nephritis
- Calculous disease of the kidney
- Tuberculous kidney
- Ascending nephritis, acute or chronic
- Cystic kidneys
- Carcinoma of the kidney
- Sarcoma of the kidney.

3. **Chronic Heart Failure** :—

- Secondary to valvular heart disease
- Secondary to myocardial degeneration, especially fatty or fibroid heart
- Secondary to chronic obstruction in the lungs, especially from emphysema and bronchitis, or fibroid lung
- Associated with very high systemic blood-pressure.

4. **Narcotic Poisoning, especially from**

- Morphia
- Opium
- Chloral
- Butyl chloral hydrate
- Veronal
- Sulphonal

5. **Macroscopic Lesions of the Brain or its Coverings** :—

- Meningitis, tuberculous, suppurative, posterior basal, cerebrospinal
- Hydrocephalus
- Tumour of the brain, especially of the pons or medulla
- Hæmorrhage
- Softening of the brain secondary to :
  - Chronic arterial degeneration
  - Syphilis
  - Embolism
  - Caisson disease
  - General paralysis

6. **Acute Specific Fevers,** such as :—

- Pneumonia
- Cholera
- Diphtheria
- Typhoid fever
- Malaria
- Infective endocarditis

The differential diagnosis of these various conditions will be indicated by symptoms and signs other than the Cheyne-Stokes respiration, for the latter will have occurred late in the great majority of the cases. The urine will be examined, the blood-pressure measured, the physical signs of the heart noted, the retina examined for retinitis, optic neuritis, or for choroidal tubercles, and careful inquiries will be made into the history. Where narcotic poisoning may be suspected, the gastric contents may be recovered and analyzed, bottles found under suspicious circumstances may be examined in the same way, or evidence of hypodermic injections sought for on the patient's body or limbs. When Cheyne-Stokes respiration occurs as the main symptom in the case, the great probability is that there are degenerative changes in the medulla oblongata, nearly always secondary to arterial degeneration, either senile, syphilitic, or sclerotic. When there have been obvious symptoms of some other kind before Cheyne-Stokes respiration develops, the latter is far more important from the prognostic than from the diagnostic standpoint. It is a sign of evil omen, though in a few cases it has persisted for many months before the end came, and in a few it has disappeared entirely for the time being, even after it had been well marked for days or weeks.

Herbert French.

CHILLS.—(See Rigors, p. 594.)

CHORDEE.—A condition in which, during erection, the penis, instead of remaining straight, becomes curved like a banana, either downwards or to one side. It is nearly always due to gonorrhœa, though in rare cases it has resulted from injury without gonorrhœa. The differential diagnosis will depend upon the history and the existence or otherwise of a urethral discharge containing gonococci. The condition itself is probably due to inflammatory effusion into one corpus cavernosum, or the corpus spongiosum, as the case may be ; or, in the absence of inflammation, to blood extravasation from a burst vessel. Fracture of the penis has occurred during resisted coitus, the diagnosis depending on the history and the break that is palpable in the penis during erection. *Herbert French.*

CHYLURIA.—The passage of milky-looking urine, due to the admixture with it of emulsified fat, is known as chyluria. It is not likely to be mistaken for phosphaturia, even when the latter, especially after the largest meal of the day, causes the urine to be almost like thin milk from the spontaneous deposition of the excess of phosphates whilst the urine is still in the bladder. The opacity in the latter case disappears on the addition of a drop or two of acetic acid, whilst the fat droplets of chyluria do not clear up with acids, are obvious under the microscope, and may be brought out still more clearly by the use of special fat stains, such as osmic acid, sudan III, or saffranin. As a rule the urine coagulates on standing, and subsequently liquefies again, when it throws up a fatty scum and deposits a sediment. The fat is most plentiful after meals which contain fat; the degree of chyluria consequently varies considerably in the same patient, and may sometimes be almost absent.

The commonest cause for the symptom is infection by *Filaria sanguinis hominis* in the tropics, adults being affected more often than children, and females more often than males. There may or may not be *elephantiasis* at the same time; the diagnosis may be suggested by eosinophilia and confirmed by the discovery of the embryos in the blood (*Plate XXVIII, Fig. F*, p. 614).

Chyluria may also occur, however, in those who have never been abroad, and it is sometimes associated in some way that is not yet fully understood with sub-acute nephritis; there may be chylous ascites (p. 50) at the same time. The diagnosis depends upon the history, the general œdema, the anæmia, cardiac hypertrophy, and upon the discovery of an abundance of albumin with renal epithelial cells and tube-casts in the centrifugalized urinary deposit, as well as fat droplets in the supernatant fluid.

Sometimes chyluria develops quite apart from any renal lesion, either spontaneously or as the result of abdominal injury; and it has generally been found in these rare cases that there has been either rupture of the receptaculum chyli, or else a blockage in the thoracic duct. The latter sometimes results in cases of malignant disease, especially carcinoma of some intra-abdominal organ with secondary deposits in the glands in the posterior mediastinum. The development of chyluria in such cases would be a late symptom, and the diagnosis would probably have been made already on account of other symptoms, especially the discovery of a primary tumour. It is important not to forget rectal and vaginal examination, lest the growth should be pelvic. *Herbert French.*

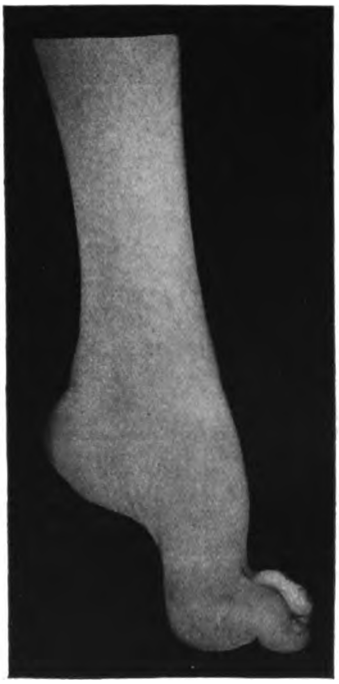


Fig. 44.—Claw-foot.

CLAW-FOOT (Pied-en-griffe) (*Fig.* 44) is much less common than CLAW-HAND, but it may arise from similar causes. The internal popliteal nerve, which supplies the interossei and lumbricals of the foot through its external plantar branch, is homologous to the ulnar nerve in the upper extremity. Its buried course in the leg does not, however, expose it to the same chances of injury as the more superficial ulnar nerve, and consequently claw-foot is not often the result of trauma. Disease or injury of the first and second sacral segments of spinal roots may produce the characteristic deformity of the toes, in which case there would probably be disturbances of sensibility in the corresponding cutaneous areas. In acute poliomyelitis affecting those segments, the diagnosis depends on the history of onset, as in the case of claw-hand of similar origin. *E. Farquhar Buzzard.*

CLAW-HAND (Main-en-griffe) is the name used to describe a hand characterized by a claw-like position of the fingers (*Fig.* 45). The fingers are extended at the metacarpo-phalangeal joints and flexed at both interphalangeal joints. This position is the result of the over-action of the extensor communis digitorum and flexores digitorum when unopposed by the normal antagonism of the interossei and lumbricals. It is not symptomatic of any particular disease, but results from any morbid condition which produces atrophic paralysis of the intrinsic hand muscles so long as the long extensors of the fingers remain intact. *Progressive muscular atrophy, ulnar paralysis, syringomyelia, cervical pachymeningitis, acute poliomyelitis, peroneal atrophy,* and *supernumerary ribs* are among the conditions which may give rise to claw-hand to a lesser or greater degree. In any particular case the diagnosis of the underlying condition depends on the result of further investigation.

In *progressive muscular atrophy*, wasting of the intrinsic hand muscles is often an early symptom (p. 61), and a claw-hand may develop before the long extensor muscles of the fingers have become involved in the disease. All four fingers are usually affected to an approximately equal extent, and there is often marked wasting of the thenar and hypothenar eminences. When the abductor pollicis is also involved, the thumb tends to come into line with the fingers and gives an appearance to the hand resembling that of the ape (ape's hand). The flexors of the wrist often become involved before the extensors, with the result that the wrist is hyperextended, and a 'preacher's hand' results. The absence of pain and of all sensory disturbance, the gradual onset, and the general exaggeration of the deep reflexes, serve to distinguish this condition from some of the other causes of claw-hand.

In *ulnar paralysis* the claw-position is more marked in the ring and little fingers than in the middle and first fingers, owing to the fact that the two outer lumbricals are supplied by the median nerve. The adductor pollicis is the only thenar muscle to suffer, but the hypothenar eminence is wasted. If the injury to the nerve is above the point where it gives off the branch to the flexor carpi ulnaris, the latter muscle will also be paralyzed, and flexion of the wrist will be carried out with a leaning towards the radial side. In ulnar paralysis the palsy is limited to the muscles supplied by the ulnar nerve, and there is usually some sensory loss in the area of skin innervated by the latter.

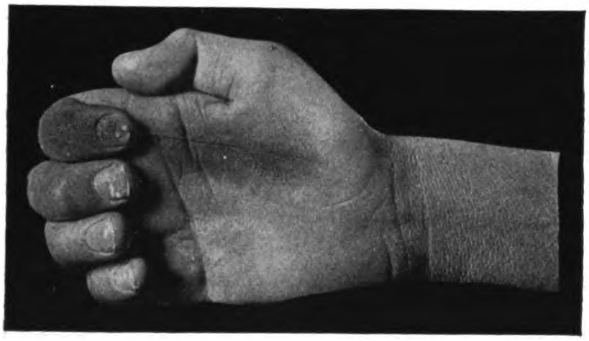


Fig. 45.—Syringomyelic claw-hand.

The claw-hand of *syringomyelia* (*Fig.* 45) resembles that of progressive muscular atrophy in general appearance, and may show the modifications to which the term 'ape's hand' and 'preacher's hand' have been applied. The muscular atrophy is not limited to the distribution of a single nerve, but involves the musculature innervated by the eighth cervical and first dorsal spinal segments,—the segments, in fact, in which the gliosis frequently begins. The diagnosis depends on the presence of dissociative anæsthesia, trophic and vasomotor disturbances such as whitlows, glossy skin (peau lisse), main succulente, and is often corroborated by the occurrence of oculo-pupillary phenomena, nystagmus, scoliosis, and evidence of spastic paralysis in the leg of the same side.

Cervical pachymeningitis only leads to a claw-hand when it interferes with the function of the eighth cervical and first dorsal anterior roots, and leaves uninjured the sixth and seventh cervical roots. The condition is generally bilateral with some asymmetry, and it is usually associated with pain and ill-defined disturbances of sensibility in the two arms.

An *acute poliomyelitis* affecting the eighth cervical and first dorsal segments, and leaving intact the sixth and seventh cervical segments, is uncommon. The history of acute onset, with constitutional symptoms such as headache, fever, vomiting, and convulsions, affords a clue to the diagnosis. The absence of sensory loss, and the possible presence of atrophic palsies in other parts of the body, form additional data in these cases.

In *peroneal atrophy* the diagnosis depends on the symmetry of the affection and the preceding or concomitant atrophy of the leg muscles, generally beginning in those supplied by the peroneal nerve (see *Figs.* 20 and 21, p. 60).

Supernumerary cervical ribs may lead to the production of a claw-hand when they cause neuritic changes in the trunk formed by the eighth cervical and first dorsal contributions to the brachial plexus. The muscular atrophy is preceded by pain in the arm and neck, and sometimes by vasomotor changes and diminution of the radial pulse. Analgesia in the distribution of the eighth cervical and first dorsal-root areas may also be detected, but the diagnosis may depend mainly on the skiagraphic discovery of the rudimentary ribs (p. 443). *E. Farquhar Buzzard.*

CLONUS, ANKLE.—(See Ankle-clonus, p. 39.)

CLUBBED FINGERS, or bulbous enlargement of the soft parts of the terminal phalanges, with over-curving of the nails both transversely and longitudinally, are seen characteristically in morbus cæruleus, and also in association with fibroid lung. They are distinguished readily from enlargement due to bony changes, such as those of acromegaly and pulmonary osteo-arthropathy.

Minor degrees may occur with almost any disease that leads to persistent congestion of terminal parts, such as mitral stenosis, mitral regurgitation, emphysema, chronic bronchitis, pleurisy with effusion, empyema, chronic phthisis, some forms of aortic or subclavian aneurysm, asthma, pericarditis, adherent pericardium, mediastinitis, or mediastinal neoplasm. In such cases, however, the clubbing has to be looked for—it does not thrust itself upon one's notice; it may also pass away again when the cause is removed, for instance, when an empyema is cured by operation.

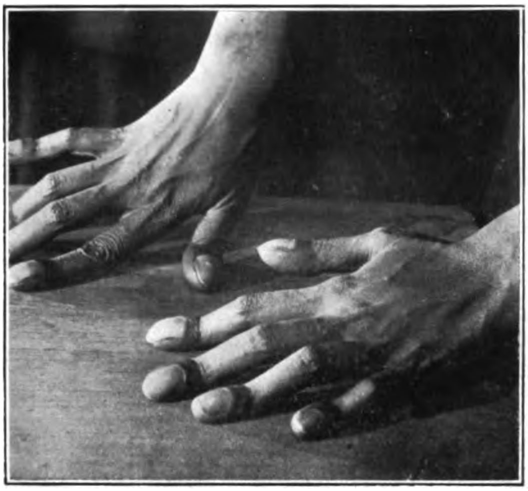


Fig. 46.—Clubbed fingers in congenital pulmonary stenosis with extreme cyanosis.

Obvious and extreme finger-clubbing has only two main causes—congenital heart disease with cyanosis (Fig. 46), especially pulmonary stenosis with or without a perforated interventricular septum; and fibroid lung, especially if associated with bronchiectasis. The distinction between these two will generally be obvious. The former dates from infancy and is associated with extreme cyanosis and a loud pulmonary systolic bruit and thrill; the latter develops later in life, is seldom associated with such extreme cyanosis except when the patient is in extremis, and is accompanied by displacement of the heart and other signs of fibrosis of the lung.

Difficulty may arise in those rarer cases of congenital heart disease in which there is no bruit—for instance when the heart gives off a single large vessel, the place of the pulmonary arteries being taken by intercostal vessels—but even here the fact that the lividity is out of proportion to the dyspnœa, and the history that the cyanosis and the finger-clubbing date from soon after birth, afford immediate clues to the diagnosis. Congenital heart disease without cyanosis—patent ductus arteriosus, for instance—does not give rise to clubbed fingers. In lung cases the diagnosis is either obvious from the physical signs; or else, if the abnormal physical signs are so slight as by themselves to suggest little more than bronchitis, the existence of marked clubbing of the fingers is important evidence that the lung trouble is more extensive than this, and that there is really much fibrosis, and probably bronchiectasis, too deep-seated to permit of the usual physical signs being detected at the surface of the chest. A moderate degree of clubbing of the fingers is sometimes observed in cases of cirrhosis of the liver, particularly in that type which begins as splenic anæmia—Banti's disease (see p. 37). This suggests that the changes in the finger tips have a chemical as well as a mechanical factor in their causation. *Herbert French.*

CLUB-FOOT, or TALIPES.—Any deformity of the foot not limited to the toes commonly goes under the name of club-foot, or talipes. The diagnosis of the different forms of talipes is extremely difficult, owing to the number of causes and the complicated nature of the deformities. It may be well, therefore, to define briefly the chief varieties of simple deformity.

1. **Talipes Equinus.**—In this condition the fore part of the foot cannot be raised to the normal degree. Any healthy adult is able, with the knee straight, to dorsiflex the ankle to such an extent that the ball of the great toe is two or three inches higher than the prominence of the heel. The degree of dorsiflexion is even greater in infants, but with

advancing years the movement becomes limited, so that old people may hardly be able to dorsiflex the foot beyond the right angle.

2. **Talipes Calcaneus.**—In this condition the heel is depressed and the fore part of the foot elevated. Extension of the ankle is limited, so that the fore part of the foot may not touch the ground in walking.

3. **Talipes Valgus.**—The foot is everted and abducted at the ankle-joint, so that the inner malleolus is too prominent.

4. **Talipes Varus.**—The foot is inverted and adducted at the ankle-joint, so that the outer malleolus is too prominent. In this condition, however, there is more serious deformity at the medio-tarsal joint, at which the fore part of the foot is adducted and inverted.

5. **Talipes Cavus.**—The arch of the foot is too high or hollow. This may be due to depression of the fore part of the foot, of the heel, or of both.

Club-feet may be divided into (I) **The Congenital,** (II) **The Acquired.**

I. CONGENITAL TALIPES.

Congenital talipes is usually quite easy to diagnose, because of the history of the presence and the nature of the deformity at birth. There are two chief varieties of it: (1) Equino-varus; (2) Calcaneo-valgus.

Sometimes the history is lacking or misleading, and the shape of the feet has been so altered by treatment or neglect that it is very difficult to distinguish the condition from paralytic talipes, especially that due to paralysis of the lower neuron. In making the distinction it is important to remember that the shortening is usually very much less in congenital cases, and that wasting of the muscles, apart from tight splinting, is also much less. Trophic ulcers, and cold and blue feet, which are common in cases of paralysis, do not occur in congenital talipes. Moreover, the toes are not hyper-extended at the metatarso-phalangeal joints, a condition commonly present in paralytic talipes. The reaction of degeneration is not present in congenital cases, thus distinguishing it from talipes due to comparatively recent paralysis of the lower neuron. The reflexes are not exaggerated, thus distinguishing it from talipes due to paralysis of the upper neuron. In congenital equino-varus the small conical heel is not only raised but also turned inwards in a characteristic way, and it is generally separated from the inner aspect of the foot by a deep furrow. There is also a curious flattening on the outer side of the foot, just in front of the external malleolus, where the skin is dimpled and loose. There is also a furrow on the inner side of the foot opposite the medio-tarsal joint. The varus is always worse than the equinus, whereas in paralytic cases the equinus is usually worse than the varus. With care the overstretched weak muscles can be shown to be capable of voluntary contraction.

II. ACQUIRED TALIPES.

This condition may be subdivided as follows: (1) The paralytic, due to: (*a*) Disease of the upper neuron; (*b*) Disease of the lower neuron; (*c*) Primary muscular disease; (2) Postural, e.g., talipes valgus; (3) Due to fibrosis of muscle, with retraction; (4) Due to bone disease; (5) Due to joint disease; (6) Due to contracting scars; (7) Due to hysteria.

1. **The Paralytic.**—(*a*) In talipes due to destruction of the *upper neuron* the reflexes are exaggerated and the plantar reflex is extensor; whereas in talipes due to disease of the lower neuron the reflexes are unchanged, diminished, or lost. Reaction of degeneration may be present with lesions of the lower neuron, and absent with lesions of the upper. Coldness and blueness of the feet are only common in lesions of the lower neuron, and the same is true of trophic ulcers. The shortening and wasting are generally much greater in lesions of the lower neuron, and the distribution of the paralysis is much more irregular than in those of the upper. When the disease of the upper neuron is in the brain, it is usual for the arm as well as the leg to be paralyzed (*infantile hemiplegia*), or both feet may be involved symmetrically (*congenital spastic paraplegia*). Occasionally there may be a cerebral monoplegia. In any case the deformity due to disease of the upper neuron is almost characteristic, and is mostly equinus, usually with a little valgus, but occasionally with slight varus; whereas when the lower neuron is affected the deformity is nearly

always equino-varus or talipes valgus. In distinguishing various destructive lesions of the upper neuron, the history and the nature of the deformity may help. In *hemiplegia* or monoplegia there may be a history of difficult labour, with delivery by forceps, indicating injury to the cerebral cortex, or meningeal hæmorrhage with secondary fibrosis of the motor area. The deformity may not be obvious for a year or more after birth, and it is usually noticed first when the child begins to walk. In other cases it may be due to *thrombosis of the cerebral veins* following measles or influenza, or to rupture of some of the cortical veins during whooping-cough or violent fits of passion. *Congenital spastic paraplegia* is distinguished by its symmetry, and by the amount of spasm as shown by the unexpected degree of flexion of the ankles that can be produced by firmly pressing upwards the fore-parts of the feet. Moreover, there is usually some mental incapacity, and often the history of nervous disease in the family. When the lesion is in the spinal cord, there may be a history of spinal injury or evidence of spinal caries, or of growth causing a spastic paraplegia. In *amyotrophic lateral sclerosis* there are signs of paralysis and wasting of the upper limbs. *Friedreich's disease*, or *hereditary ataxy*, is an occasional cause of talipes equinus or equino-varus. It can be recognized by the inco-ordination, the nystagmus, the slurring of speech, the age of onset, which is usually about six to nine years, the absence of knee-jerk, and the hallux erectus.

(*b*). *Lesions of the lower neuron* may be in the cord (infantile paralysis), or in the cauda equina (spina bifida), in the lumbo-sacral cord or sacral plexus (e.g., carcinoma of the rectum), or in the peripheral nerves (peripheral neuritis, injured sciatic nerve, or Tooth's neuro-muscular paralysis). *Infantile paralysis* results from acute anterior poliomyelitis, and is distinguished by its irregular distribution, reaction of degeneration, and its vasomotor and trophic lesions. It is frequently possible to show that the patient is unable to use certain muscles or groups of muscles, especially the anterior tibial and peroneal group. It is unusual for the paralysis to be limited to the leg; the thigh is often affected to some extent, and often the opposite leg. It is important to examine for *spina bifida*; talipes due to this is not necessarily symmetrical; one foot may be involved more than another, and the deformity is often progressive. I have seen several cases of talipes calcaneo-valgus associated with it, and also pure cavus, and one very bad case of equino-varus of one foot, and equino-valgus of the other. The foot may drop in *peripheral neuritis* due to diphtheria, lead poisoning, or alcoholism. In each of these conditions there is other evidence of the disease. In many cases of growth in the pelvis the foot may drop owing to invasion of the sacral plexus by the growth, which may be either sarcoma of the pelvis or carcinoma of the rectum. Wounds of the thigh, or the pressure of tight splints in the treatment of fracture, or the forcible extension of a contracted knee, may lead to paralysis of the sciatic nerve, especially of its external popliteal branch. This may lead to talipes equino-varus. A similar deformity may follow injury of the lumbar spine with secondary hæmato-rhachis, or growth anywhere in the course of the sciatic nerve. I have known it follow the use of a Hodgen extension apparatus. *Tooth's neuro-muscular paralysis* (*Figs.* 20 and 21, p. 60) causes paresis of the anterior tibial and peroneal muscles, with talipes equino-varus and marked cavus, and deformity of the toes. It may be distinguished from infantile paralysis by the symmetrical affection of both feet, by the wasting of the thenar eminences, and the history of similar deformity in the family, and from the primary muscular dystrophies by the occurrence of reaction of degeneration.

(*c*). *Primary Muscular Disease.*—In primary muscular paralysis (see ATROPHY, MUSCULAR, p. 59) talipes may be developed late in the disease; but as a rule the patients do not live long enough for the deformity to become a striking feature. The family history assists the diagnosis, and in the pseudo-hypertrophic form there is the characteristic way in which the patient raises himself from the supine position by rolling into the prone position and then lifting himself on his toes and hands, and working his hands up the fronts of the thighs.

2. **Postural.**—Acquired talipes valgus may be due either to posture or to paralysis of the tibiales muscles. When a patient attempts to adduct and invert the fore-part of the foot, the tendons of these muscles can be seen to stand out when they are not paralyzed. The foot may be forced into a cramped position by tight boots, and a form of talipes cavus may thus develop, with marked deformity of the toes, which are hyper-extended at metatarso-phalangeal joints and flexed at the others. This condition must not be

confounded with a similar one due to paralysis of the small muscles of the foot, especially the interossei and lumbricales.

3. **Fibrosis and Contracture of the Muscles of the Calf.**—Very rarely the calf muscles may contract as a result of an ischæmia analogous to that occurring in the fore-arm, and leading to contracture of the wrist and fingers (Volkmann's contracture, *Fig.* 58, p. 141). The same condition may develop as a result of cellulitis of the calf muscles, often associated with compound fracture of the leg, or with acute necrosis of the tibia. In all these conditions it is important to prevent the development of talipes equinus.

4. **Bone Disease.**—Injury or inflammation of the tibia near the epiphyseal lines in youth may lead either to arrest or over-growth of the affected bone. This is not uncommonly a cause of talipes, which can be recognized if care be taken to make comparative measurements and *x*-ray examinations of the bones.

5. **Joint Disease.**—In fractures into the ankle-joint, such as Pott's and Dupuytren's fractures, a very bad form of talipes equino-valgus may form unless care be taken to correct the deformity and to keep the ankle moving. Talipes equinus may arise as a result of the maltreatment of sprains or arthritis of the ankle, either septic or tuberculous, unless care be taken to keep the joint dorsiflexed during treatment.

6. **Contracting Scars.**—Occasionally talipes equinus follows severe burns or lacerations of the skin of the leg or foot. The diagnosis is usually obvious from the scars. There may be some wasting of the muscles from want of growth of the limb from disuse.

7. **Hysteria.**—Hysterical club-foot may be suspected from the associated symptoms and confirmed by the absence of any change in the electrical reactions, by the variation of the deformity, and the disproportionate amount of spasm, which passes off during sleep and under an anæsthetic.

Finally, it is to be remembered that if a normal muscle is left in one position over a long period with its points of origin and insertion unduly approximated, it may presently be found to be impossible to lengthen it out properly again ; it is in this way that contractures of muscles are apt to occur during the course of long febrile illnesses—enterica for instance—when the patient may remain curled up in bed for weeks. If the limbs are passively extended and flexed each day, no contracture results, but it sometimes happens that the neglect of this precaution is followed by persistent contracture of what had hitherto been normal muscles, and one of the likely results of this is club-foot. *R. P. Rowlands.*

COITUS, PAINFUL.—(See DYSPAREUNIA, p. 193.)

COLIC.—This is a word often used very loosely for any severe abdominal pain, especially of a kind which tends to wax and wane in intensity. Such pain may be associated with disease in almost any one of the abdominal viscera, and the word colic is applied quite commonly to the pain caused by the passage of a calculus down the bile-duct (biliary colic) or the ureter (renal colic). The name ' mucous colic ' is also used by some writers for the disease usually known as muco-membranous colitis. It is better, however, to restrict the term colic, used without a qualifying adjective, to pain caused by contraction of the intestine, of a cramp-like nature, caused by local irritation or by general poisoning, in the absence of any organic disease of the bowel. Diagnosis therefore mainly consists (1) *In excluding such organic affections* ; and (2) *In ascertaining, so far as possible, the cause of the local spasm.*

In order to exclude organic disease a careful examination of the whole abdomen is needed, as well as observation of the general condition of the sufferer. It must be remembered that in simple colic there may be vomiting, sweating, and some degree of collapse owing to the severity of the pain. The patient's temperature is not, however, usually raised ; the abdominal walls move freely on respiration ; and there is little or no local tenderness, pressure being often a relief to the pain, so that the sufferer tends to press his abdomen against a pillow or other support. Though the face exhibits an expression of pain, there is not the pinched, anxious facies so characteristic of grave abdominal troubles ; and the patient is likely to throw himself about instead of lying still as in such conditions as peritonitis or intestinal obstruction. The pulse is not often markedly affected ; it may even be unduly slow, but in nervous subjects the anxiety and pain may cause some rise in its frequency.

The different affections which may give rise to abdominal pain liable to be called colic by patients are: Acute intestinal obstruction, intussusception, appendicitis, and possibly even perforative peritonitis; colitis and ulcerative diseases of the colon; malignant disease of the intestine; pancreatic disorders, acute and chronic; gastric pain, especially that encountered in cases of pyloric obstruction; intestinal neuralgia, and referred pains in spinal caries and in cases of pressure by tumours or aneurysms; gastric and intestinal crises in locomotor ataxy; chronic plumbism (p. 34); and renal and biliary colic.

Taking the diagnostic features separately:—

Rise of temperature above 100° F. will indicate the existence of some inflammatory affection, such as appendicitis. The possibility of thoracic disease, such as pneumonia or diaphragmatic pleurisy, causing abdominal pain, must be borne in mind; but such pain is not really colicky in character. (See PAIN, ABDOMINAL, p. 424.)

Vomiting that is repeated and severe does not occur in simple colic. It suggests the existence of intestinal obstruction, if the temperature of the patient is normal or subnormal, or of some form of peritonitis if there be fever. In the former condition a fæcal odour may be noted in the vomit; in general peritonitis the vomiting may be characteristic, large quantities of fluid being brought up with little effort; but these signs occur late in the course of these conditions (see VOMITING, p. 763). The colicky pains associated with gastric dilatation due to pyloric obstruction are likely to end with the expulsion of a large quantity of foul fermenting material. The dilatation of the stomach may be ascertained by noting the existence of splashing in the organ when the fingers are 'dipped' sharply in the epigastric region; by eliciting an increased area of tympanitic resonance; by observing the peristaltic movements of the hypertrophied walls of the stomach, as seen by inspection of the abdomen; by discovery in the vomit of food taken some days previously, as well as of organisms of fermentation (torulæ and sarcinæ, *Fig.* 121, p. 241), the vomited matter being generally foul and frothy; and by examination with the *x*-rays after exhibition of a bismuth meal (*Fig.* 128, p. 268).

Tenderness and rigidity of the abdominal wall are usually absent in colic. When conjoined, they point to affection of the peritoneum; tenderness alone indicates disease of some viscus, as in colitis, when it is found along the course of the colon, in intestinal or gastric ulceration, and so forth.

Slight fullness of the abdomen may exist in cases of colic, but it is usually inconspicuous; more often the abdominal walls are retracted. Considerable distention indicates some organic trouble, such as cirrhosis of the liver, intestinal obstruction, or peritonitis. A contracted portion of bowel may sometimes be felt. This must be distinguished from an actual tumour or inflammatory mass, and from the elongated swelling felt in intussusception. The spasmodically contracted gut of colic is of small diameter, and may be felt to relax as the pain subsides and to harden again with a fresh exacerbation.

Constipation is the rule in patients suffering from colic, and if a motion is passed it is small and hard. The appearance of diarrhœa will point to some affection of the bowel, such as colitis. In mucous colitis, which is associated with severe pain, hard scybala may be passed along with casts of the intestine (*Fig.* 172, p. 398) or large shreds of mucus: these may take the form of rolls resembling segments of tape-worm, but can easily be floated out if placed in water (see below). The appearance of any blood per anum will show that something more than mere colic is present (see BLOOD PER ANUM, p. 75).

Attacks of severe abdominal pain occur in gouty and arteriosclerotic subjects, accompanied by giddiness, nausea, and sometimes vomiting ('angina abdominalis'); there may be slight jaundice. Examination of the pulse will reveal increased tension and possibly disease of the arterial wall, and the trouble yields rapidly to nitroglycerin tablets and iodides.

Pain associated with a movable kidney (*Dietl's crises*) might be described by the sufferer as colic. Such attacks are characterized by sudden pain, nausea, faintness, and collapse; there may be blood in the urine (see HÆMATURIA, p. 280). In some instances the kidney is enlarged as well as movable, owing to developing hydronephrosis.

Intestinal neuralgia may be difficult to distinguish from colic, as both are alike functional disorders without organic disease. Neuralgia is likely to occur in an anæmic, ill-nourished person of neurotic type; it arises without obvious exciting cause, and may recur at the same time of the day with some regularity. The pain has not the cramp-like character of colic, but is aching, boring, or darting. It is a very rare disorder, and can only be recognized by exclusion of all organic disease and of the intestinal spasm associated with colic.

The *gastric* or *intestinal crises* of locomotor ataxy may be indistinguishable from colic, except by recognition of the other symptoms of the disease—absence of knee-jerks, ataxy, Argyll Robertson pupils, lightning pains and girdle-sensation. Examination of the blood and cerebrospinal fluid may reveal the presence of the Wassermann reaction, and excess of lymphocytes may be found microscopically in the latter fluid.

In children, who are specially liable to suffer from attacks of colicky pain due to indiscretions in diet, it is important to bear in mind the possibility of *appendicitis*, on the one hand, as a cause of abdominal pain, and on the other of *Pott's disease*, which may give rise to pain referred to the front of the abdomen. Examination of the spine in these latter cases may reveal the existence of rigidity and tenderness, perhaps some prominence of one or more vertebral spines, and examination with the *x*-rays may give positive evidence of caries of the bodies of the vertebræ.

Appendicular Colic.—This term is sometimes applied to attacks of pain in the right iliac fossa. Their association with disease of the appendix is doubtful. Appendicitis may ensue subsequently, but it is as likely that the original attacks may have been due to colitis (typhlitis), which afterwards spread to the appendix, as that this organ was at fault throughout. Unless the signs of appendicitis are present (p. 454), the condition cannot be recognized with certainty. In all cases of doubt as to the cause of colicky pains, an examination per rectum is advisable ; it may reveal the presence of inflammation in the appendicular region, or of an intussusception, in quite unsuspected cases.

The term **mucous colic** is sometimes used as a synonym for mucous colitis. The disease is characterized by obstinate constipation and by attacks of abdominal pain, during or after which shreds and rolls of mucus, or even casts of large portions of the bowel (*Fig.* 172, p. 398), are evacuated along with scybalous masses. The casts float out in water and are often spoken of as 'skins' by patients who suffer from this malady. Microscopically they consist of mucus with few leucocytes or epithelial cells.

Biliary Colic.—The passage of a calculus down the bile-ducts gives rise to severe and even agonizing pain in the right hypochondrium. It is of a colicky character, but it is apt to be more intense than that of simple colic. It may be accompanied by vomiting, sweating, and collapse. Shivering is frequent, and if present is suggestive of this trouble. The pain is likely to pass round into the right side and to the angle of the right scapula ; it may even be referred to the tip of the right shoulder. If the calculus lodge in the common bile-duct, jaundice will result. Its depth will vary with the degree of obstruction, and while the colic lasts it is not likely to be very intense. Palpable enlargement of the gall-bladder is quite exceptional in cases of gall-stones. Actual proof of the cause of the colic may sometimes be obtained by finding a stone in the fæces, by passing them through a coarse sieve under a current of water. Attacks of gall-stone colic are liable to recur, and a history of previous illness of the same kind may aid in the diagnosis. Women are rather more subject to gall-stones than men, and fat subjects suffer more than thin. The malady is most often encountered in middle life. In some instances examination with the *x*-rays may afford confirmatory evidence of the existence of calculi in the gall-bladder ; but failure of such confirmation does not exclude their presence, as their substance is not very opaque to these radiations.

Pancreatic Colic, due to passage of a calculus along one of the ducts of the gland, may occur, but can scarcely be diagnosed. It is characterized by severe, deeply seated pain in the epigastrium, sometimes extending to the back and loins. Exactly similar attacks of pain occur in *chronic pancreatitis*, and may be accompanied by shivering, or actual rigors. Intense jaundice may also be seen in this malady, and an enlarged gall-bladder can usually be felt. The condition can only be recognized when there are present other signs of pancreatic disease—wasting, pigmentation of the skin, and the passage of bulky, offensive stools, containing large quantities of fat. Chemical examination may show that much of this fat is neutral (unaltered) fat, with less than the usual proportion of fatty acids (p. 101). Microscopical examination may reveal the presence of unaltered meat-fibres in the motions. The urine may contain sugar, and Cammidge's Test (p. 100) may be applied to it, though the trustworthiness of this reaction is not yet established.

Renal Colic.—The distinguishing features of the *passage of a calculus down the ureter* are similar to those of biliary colic, but the pain starts in one loin and radiates downwards to the thigh and to the testicle in the male, to the labium majus in the female. The urine may contain blood, and also epithelium, from the pelvis of the kidney and from the ureter. Frequency of micturition is often marked, but the quantity of urine may be small; it may even be suppressed temporarily. If the calculus become impacted in the ureter a swelling may subsequently appear in the loin, due to the formation of a hydronephrosis. The pain may cease suddenly when the stone passes into the bladder. The x-rays are of considerable value in detecting the concretion (*Fig.* 192, p. 455), provided the bowels be empty so that shadows due to scybala can be avoided.

The pain due to the presence of a *calculus in the kidney* can hardly be mistaken for colic, but occasionally the symptoms of this condition may precede an attack of renal colic. A history, therefore, of pain in the loin, frequency of micturition, and the appearance of blood in the urine, may help in the diagnosis of the latter condition. Tuberculous disease of the kidney, in which the symptoms may be very similar, though apt to be accompanied by more wasting and by evening pyrexia, may give rise to colicky attacks if blood-clots or caseous masses lodge in the ureter. Pus and tubercle bacilli may be found in the urine. Dietl's crises have been referred to above.

The principal causes of **Intestinal Colic** are *indigestible food, alcoholic excess*, and *lead-poisoning*. This last should be eliminated first. It is characterized by symptoms described on p. 34. There will usually be a history of some occupation involving contact with lead—painting, glazing, type-setting, or manufacture of some compound of lead; but the possibility of poisoning by drinking-water or by beer which has stood in contact with leaden pipes must be remembered—the latter especially in potmen. The chief signs of alcoholism are given on p. 726. In cases due to indigestible food, a history of the consumption of fried fish, shell-fish, pork, raw fruit, or other suspicious matter may be obtained. The pain is more likely to move along the course of the colon than to remain fixed in the centre of the abdomen or at some special point, as it usually does in lead colic. In infants, colic may be caused by hard curds of milk, and be indicated by drawing up of the legs and screaming. In older children, unripe apples, plum-stones, and similar delicacies are often the source of the trouble, and fruit-stones may be discovered subsequently in the motions.

W. Cecil Bosanquet.

COLOUR BLINDNESS.—(See Vision, Defects of, p. 762.)

COMA is a state of unnatural, heavy, deep and prolonged sleep, often accompanied by slow stertorous or irregular breathing, and frequently ending in death. It may be due to a large number of different causes, which may be classified into two main groups, namely: (*A*) Cases in which coma is not a prominent symptom early in the malady, but only in a late stage, when the nature of the disease has already been suggested by other symptoms; and (*B*) Cases in which coma comes on early and may be the most prominent feature of the case.

Group A includes—

1. **Certain Severe Fevers** in which coma may occur as a terminal phenomenon:

Typhus fever
Typhoid fever
Cholera
Dysentery
Measles
Scarlet fever
Rheumatic fever
Yellow fever
Blackwater fever
Malignant malaria
Infective endocarditis.
Diphtheria.

2. **Acute Inflammatory Lesions of the Brain or the Cerebral Meninges:**

Acute encephalitis
Suppurative meningitis
Tuberculous meningitis
Posterior basal meningitis
Epidemic cerebrospinal meningitis, or spotted fever

3. **Certain Less Acute Lesions of the Central Nervous System:**

Cerebral tumour
Cerebral abscess
Post-epileptic state
General paralysis of the insane
Disseminated sclerosis
Syphilis of the brain

4. **Diseases in which General Metabolism is probably at Fault:**

Uræmia
Diabetes
Cholæmia
Addison's disease
Raynaud's disease
Myxœdema

Group B includes the following conditions—

1. **The Results of Head Injury:**

Compression by meningeal hæmorrhage
Concussion
Depressed fracture
Fracture of the base of the skull.

2. **Vascular Lesions of the Brain:**

Embolism
Hæmorrhage
Thrombosis: (*a*) arterial, (*b*) of a venous sinus such as the superior longitudinal.

3. **The Acute Effects of Drugs,** particularly:

Alcohol
Opium
Morphia
Carbolic acid
Oxalic acid
Carbon monoxide
Absinthe
Chloral hydrate
Veronal
Sulphonal
Trional
Tetronal
Bromides
Chloroform and other anæsthetics.

4. **The Chronic Effects of Chemicals,** especially plumbism: (Saturnine encephalopathy).

5. **The Effects of Extremes of Temperature:** Heat stroke | Excessive cold.

6. **Excessive Loss of Blood** from:

Ruptured tubal gestation
Post-partum hæmorrhage
Hæmoptysis
Hæmatemesis
Duodenal bleeding
Intestinal bleeding
Ruptured aneurysm.

7. **Stokes-Adams' Disease.**

8. **Sudden Nervous Shock.**

9. **Hysterical Trance.**

Although it is generally possible to make a broad distinction between the two groups enumerated above, it is necessary perhaps to point out that some conditions which usually give rise to other symptoms before they produce coma, sometimes pass unrecognized until coma supervenes. This applies, for example, to certain cases of diabetes mellitus, uræmia, suppurative meningitis, or cerebral abscess or tumour; whilst, conversely, some conditions which usually exhibit coma early, may not do so until after there have been other symptoms to indicate the nature of the case. It is not necessary to enter into the differential diagnosis of those conditions in which other prominent symptoms have preceded coma.

When coma is either the first or the most prominent symptom in the case, it is important to arrive as near the correct diagnosis as may be at the earliest possible moment, the case being relegated to one or other of the following four classes, which differ from one another radically as regards treatment:—

1. Cases in which immediate trephining is required, *e.g.*, for meningeal hæmorrhage.
2. Cases in which active treatment by lavage of the stomach or by the administration of antidotes is required, as in opium or other poisoning.
3. Cases in which active medicinal or physical treatment is required: for instance, diabetic coma requiring the administration of alkalies, or uræmia requiring venesection.
4. Cases in which absolute rest is indicated, especially in cerebral hæmorrhage.

When investigating a case, notice first whether there is any evidence of *unilateral paralysis*: the pupils may be markedly unequal, one cheek may be more puffed out on expiration then the other, one arm or leg may fall more limply than the other; there may be differences between the two knee-jerks or the two plantar reflexes; there may be conjugate deviation of the eyes. If there is distinct evidence of unilateral paresis or paralysis, there is almost certainly a cranial or intracranial lesion—hæmorrhage, embolism, fracture, tumour, abscess, thrombosis, or meningitis. Next, examine the head with particular care to see if there are any signs of injury; the presence of a scalp wound or even of a fracture does not of course prove that this is the primary cause of the coma, for the patient may have become unconscious, from a cerebral hæmorrhage for example, and in falling may have struck his head, in which case the injury is due to the coma, and not the coma to the injury. Some of the greatest difficulties in diagnosis arise on this account, particularly when the patient has previously taken sufficient alcohol for his breath to smell of it, and to suggest that he is drunk. Careful observation for several hours may be required before the diagnosis can be settled, and even then errors are sometimes unavoidable. A clear history is generally lacking, but if available it often assists materially in deciding the nature of the case. The ears and nose should be examined with care to see whether cerebrospinal fluid or blood is coming from either, as an indication that there is a fracture at the base of the skull; blood coming forward into the subconjunctival tissue may afford similar evidence.

Cerebral hæmorrhage is much more common in an elderly than in a young person, whilst the reverse is true of embolism. The latter may occur instantaneously, whilst hæmorrhage produces coma rather more gradually; and thrombosis, syphilitic or otherwise, often leads to hemiplegia so gradually that no coma occurs. The presence of albuminuria with casts, with a high blood-pressure as measured instrumentally; the history, in an elderly man, of a previous seizure of a similar kind with definite hemiplegia, especially if there is also an enlarged heart with a lumpy first sound at the impulse, or perhaps a local systolic bruit there, with a ringing aortic second sound, would all indicate cerebral hæmorrhage, associated with defective arteries and perhaps with granular kidney. Albuminuric retinitis should be looked for. Strong evidence in favour of *cerebral embolism* would be afforded by a previous history of acute rheumatism and the existence of a presystolic or other bruit indicative of organic heart disease, especially if there are signs (p. 34) suggesting that fungating endocarditis has supervened.

Supposing there is no evidence of a unilateral paralysis, it does not immediately follow that none of the above conditions are present; one form of cerebral hæmorrhage in particular that may cause no unilateral paralysis is *pontine hæmorrhage*; this might be suggested at once by the very small, almost pin-point pupils, though similar pin-point pupils may be due to *opium poisoning*. The thermometer affords a means of diagnosis between these, for opium poisoning leads to a subnormal temperature, whilst hæmorrhage into the pons Varolii causes the temperature to rise even to the point of hyperpyrexia. The diagnosis of other varieties of coma due to poisoning can seldom be arrived at accurately unless the circumstances of the case either allow of an analysis of the gastric contents, or else point to the patient having taken an over-dose of one of the drugs mentioned in the above list, either accidentally or with suicidal intent. The bottle may be found near the patient.

Coma due to poisoning by *carbon monoxide* is sometimes obvious from the patient's bright cherry-red colour; it is impossible to convert the carboxyhæmoglobin in his blood into reduced hæmoglobin by the ordinary ammonium sulphide method; and there is generally direct evidence of the mode of poisoning, such as the fact that the patient is found in a room with the windows shut and the gas turned on, or has been subjected to the fumes of slow combustion from a stove, brazier, limekiln, or some other fire which has been burning with an insufficient supply of oxygen.

Saturnine encephalopathy is very variable in its symptoms; it may take the form of epileptiform convulsions; more or less dementia; continued coma; acute mania; indeed, its multiformity is one of its chief features. The occupation of the patient may point to the diagnosis forthwith, or there may be a blue line upon the gums or other signs of lead poisoning (p. 34). Not infrequently, however, the nature of the case gives rise to much perplexity before the diagnosis is ultimately made. One method of arriving at the latter is to collect an abundance of urine, evaporate it to dryness, and apply the tests for lead to the residue; or to test for lead in the fæces. The case is apt to be mistaken for either cerebral hæmorrhage, cerebral tumour, or general paralysis of the insane. Optic neuritis may be due directly to plumbism, and this makes the differential diagnosis still more difficult, unless there is clear collateral evidence of lead poisoning.

Myxœdema is generally diagnosed from the facies (p. 38) and general state of the subcutaneous tissues, or from the results of thyroid treatment; occasionally, however, one meets with a case in which the mental symptoms so far outweigh the others that the nature of the malady is apt to escape attention altogether. An attack of coma is rarely the first sign, though it may be; more often there is a longish history of progressive mental slowness, sometimes with delusions, and often associated with attacks of irascible excitation alternating with fits of depression; or with bouts of mental lethargy stopping short, as a rule, of actual coma.

Coma due either to *heat stroke* or to exposure to *excessive cold* is generally indicated by the collateral evidence, especially as regards the temperature of the patient's surroundings, or his having been exposed to very strong sun's rays when at work. The chief difficulty will be to make certain that there is not any vascular lesion of the brain. When there is doubt, the course of the case may indicate its nature, heat-stroke generally recovering rapidly, or ending fatally with hyperpyrexia ; but sometimes, even in a fatal case, the diagnosis may remain in doubt until a post-mortem examination has been made.

Acute encephalitis is a disease of children rather than of adults ; its general symptoms are those of acute meningitis ; the patient becomes unconscious more rapidly, however, than is usual with the latter, and yet, notwithstanding the apparent severity of the illness, recovery may occur, either within a few days or a week or two. The diagnosis rests upon the course and recovery, for in the earlier stages it will nearly always have been regarded as acute meningitis. The same applies to acute *thrombosis of the superior longitudinal sinus*, the diagnosis between which and acute encephalitis or meningitis is generally one of opinion only, unless operative measures are resorted to, or a post-mortem examination made. Optic neuritis, as well as headache, vomiting, and general convulsions, may occur in all three.

General paralysis of the insane does not as a rule give rise to coma and epileptiform convulsions until the nature of the case has been indicated already by the mental and physical changes—particularly the ideas of grandeur, the loss of highest cerebral control in one way or another, the changes in disposition, and the inability to perform the finer movements required for writing, dancing, playing the piano or violin, painting, and so forth, in which the patient may at some time previously have been an adept. Occasionally, however, notwithstanding some alterations in the mental character, the diagnosis of general paralysis may not have entered one's mind in a given case until a sudden syncopal seizure, with or without convulsions, attracts particular notice to it. It is not impossible that such a case may even then be mistaken for one of severe cerebral hæmorrhage, and it may be treated as such until it is found that the coma, severe though it may have been, passes off rapidly in a way that would not have been the case had it been a hæmorrhage of corresponding severity. The recurrence of these attacks will make the diagnosis certain, even if it remains in doubt for a time, and examination of the cerebrospinal fluid for excess of small lymphocytes or for Wassermann's serum reaction will serve to clinch the diagnosis in most cases.

Severe *hæmorrhage* other than cerebral as a cause for coma is usually indicated at once by the sudden extreme blanching, not only of the patient's cheeks, but also of his lips and mucous membranes. The pulse-rate rises to 100, 120, or even 150, according to the amount of blood that has been lost ; if there has been external evidence of the hæmorrhage, the differential diagnosis will be arrived at as discussed under such headings as HÆMATEMESIS, HÆMOPTYSIS, METRORRHAGIA, etc. If the bleeding has been internal in a healthy person, the commonest cause is duodenal ulcer in a man, pelvic hæmatocele or ruptured tubal gestation in a woman ; similar blanching in cases of typhoid fever would point to intestinal bleeding. The coma in such cases comes on suddenly, but it does not long remain profound. It is often preceded by amaurosis, and may be accompanied by epileptiform convulsions, so that acute uræmia may be simulated.

When an aortic aneurysm ruptures either into a bronchus, the œsophagus, trachea, stomach, or bowel, the amount of blood-loss seldom leads to coma, but rather to sudden death ; sometimes, however, when the bleeding is into some closed space such as the mediastinum or retroperitoneal tissue, the blood-escape is checked to some extent, and acute blanching with coma precedes further bleeding and death. Rupture of an aortic aneurysm into the pericardium causes sudden death before the amount of blood lost has been sufficient to produce marked blanching.

The phenomena of Stokes-Adams' disease are described on p. 83.

Hysterical or functional *trance* is an affection of young women, and it is not very common ; the diagnosis is arrived at by a process of exclusion, and until the case has been watched for some time its nature may not be obvious unless there have been other hysterical symptoms previously (p. 465). It is a dangerous diagnosis to make until every other possible cause for coma has been considered and satisfactorily excluded, for it is not difficult to jump to the conclusion that coma in a girl or young woman, really arising perhaps from a cerebral tumour or abscess, is due to a neurosis. It is most important to examine the optic discs with great care, lest there should be optic neuritis, the latter never being functional.

Herbert French.

CONJUNCTIVITIS.—(See EYE, ACUTE INFLAMMATION OF, p. 231.)

CONSTIPATION.

I. CHRONIC CONSTIPATION.

The indigestible residue of a meal normally reaches the descending colon in less than sixteen hours, and in defæcation all the contents of the large intestine beyond the splenic flexure are evacuated. Some of the residue of a meal taken eight hours after defæcation should be excreted at the next defæcation in individuals whose bowels are opened every twenty-four hours. If, however, the bowels are only opened on alternate mornings—a condition which is not necessarily pathological—forty hours instead of sixteen would elapse before some of the residue of the meal would be excreted. Constipation may therefore be defined as *a condition in which none of the residue of a meal, taken eight hours after defæcation, is excreted within forty hours.* Constipation thus defined can be recognized by giving three charcoal lozenges with food eight hours after defæcation ; if a blackened stool is not passed within the next forty hours the patient is constipated. The abnormal action of the bowels in constipation may manifest itself in three different ways :—

1. *Defæcation may occur with insufficient frequency.* A daily action of the bowels is merely a matter of convenience, and many people in perfect health only defæcate once in two or three days. As a rule, however, an individual may be regarded as constipated if his bowels are not opened at least once in forty-eight hours.

2. *The stools may be insufficient in quantity and a certain amount of fæces is retained,* although the bowels may be opened once daily or more often. This condition (cumulative constipation) can be differentiated readily by the charcoal test from that in which the bowels are properly emptied but the fæces are very small in quantity owing to the diet or to the unusually active absorptive power of the intestines.

3. *The bowels may be opened daily, yet the fæces are hard and dry, owing to prolonged retention before excretion* ; the deficient quantity of water in the stools also renders them less bulky than normal. The stools may be similar in character when an excessive quantity of fluid is lost by other channels, as in diabetes. By means of the charcoal test it is easy to determine whether constipation is also present.

After constipation has been diagnosed, it is necessary to determine its cause. The first essential is to distinguish between two great classes of constipation : that in which the passage through the intestines is delayed whilst defæcation is normal—*Intestinal Constipation* ; and that in which there is no delay in the arrival of fæces in the pelvic colon, but their final excretion is not performed adequately—*Pelvi-rectal Constipation or Dyschezia.*

A.—DIAGNOSIS BETWEEN INTESTINAL CONSTIPATION AND DYSCHEZIA.

A rectal examination should be made in the morning, after an attempt has been made to open the bowels without the assistance of medicine, enemata, or suppositories. If more than a very small quantity of fæces is found in the rectum, dyschezia may be diagnosed. If the rectum is almost or quite empty, the constipation must be due to delay in the passage through the intestines, except in uncommon cases of dyschezia in which there is inability to pass fæces from the pelvic colon into the rectum. The latter condition can be recognized on rectal examination, if the pelvic colon is felt through the front wall of the rectum to be filled with solid fæces ; the presence of fæces in the pelvic colon can also be proved by sigmoidoscopic examination made at once, without preparation of the patient by washing out his bowels.

At the same time the abdomen should be palpated. If scybala are felt in any part of the colon, intestinal constipation must be present. This is, however, not necessarily the case if fæces are felt in the iliac or pelvic colon, as the rectum in dyschezia may be so full of fæces that retention occurs secondarily in the pelvic colon and rectum ; such a condition would be recognized by the rectal examination.

When a patient feels that there is something in his rectum which he cannot expel at all, or that after defæcation the relief is incomplete, dyschezia is probably present. The absence of this symptom does not exclude the possibility of dyschezia, as the rectum is often so insensitive in such cases that no sensation is experienced, even when it is filled tightly with fæces. The frequent passage of very small pieces of hard fæces (fragmentary constipation), or the occurrence of pseudo-diarrhœa—in which small fluid stools, sometimes containing hard fragments of fæces, are passed, although the charcoal test shows the presence of constipation—are both symptoms suggestive of dyschezia.

Some indication, which is not, however, absolutely reliable, can be obtained from the results of previous treatment. Patients who have found that diet and mild aperients readily give them relief are probably suffering from intestinal constipation. Those who have obtained better results with enemata, and particularly with suppositories, probably have dyschezia. Dyschezia is of course also present in those patients who have to dig out the fæces from the rectum with their fingers.

Examination with the *x*-rays is the only method by which the two classes of constipation can be separated with absolute certainty, and by which the predominant condition can be discovered in cases in which both are present together. Two ounces of barium sulphate mixed with porridge or bread and milk are taken at breakfast, and at intervals during the next two or three days observations are made of the shadow produced on the fluorescent screen. The colon should be emptied as completely as possible by aperients and enemata for two or three days, but no medicine should be given the day before the examination, on the morning of which an enema must be used if the bowels have not acted naturally. During the period of observation no aperients or enemata should be given, and the patient should be allowed to continue his usual occupation and to take his ordinary diet. In intestinal constipation, delay is observed in the passage through some part or all of the colon, and occasionally the small intestine ; in dyschezia there is no delay in the intestines, but the act of defæcation does not empty the pelvic colon and rectum completely (*Figs*. 47, 48).

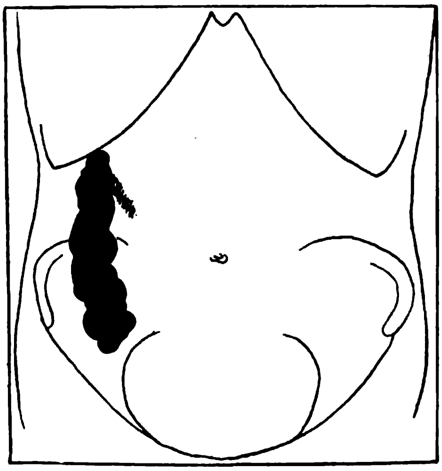


Fig. 47.—Habitual Constipation. Twenty-four hours after bismuth breakfast. No bismuth has reached beyond the first two inches of the transverse colon. Subsequent examinations showed that a similar delay occurred along the whole of the large intestine.

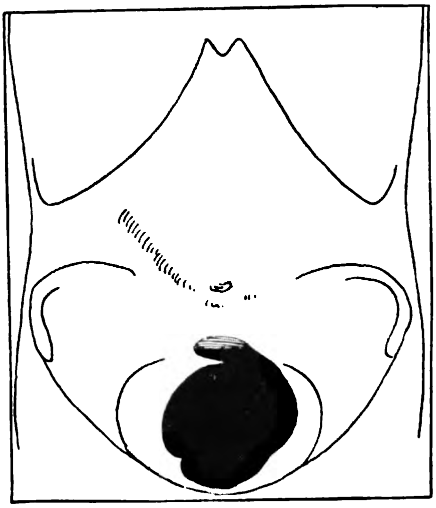


Fig. 48.—Dyschezia. Twenty-four hours after bismuth breakfast. All the bismuth has collected in the dilated colon and rectum, except traces which remain in the transverse colon. In spite of this the patient felt no desire to defæcate.

B.—DIAGNOSIS OF THE CAUSE OF INTESTINAL CONSTIPATION.

Intestinal constipation may be due to (1) *The motor activity of the intestines being deficient* ; or (2) *The force required to carry the fæces to the pelvic colon being excessive*. In the first group of cases aperients are generally much more effective than in the second ; in the latter there may be a history that purgatives are producing less effect than formerly, or that they now completely fail to act, but that enemata still give a more or less satisfactory result. The increased activity of the intestines in their attempt to respond to the excessive demands in the second class often leads to colic.

1. Deficient Motor Activity may be due to:—

(*a*). **Weakness of the Intestinal Musculature.**—When constipation has existed from infancy, especially if it is present in several members of the family, it is likely to be due to congenital hypoplasia of the intestinal musculature. Constipation developing gradually as old age approaches is generally due in part to senile intestinal hypoplasia. When constipation occurs in chlorotic girls, in cachectic conditions, in rickets, and in fevers, it may generally be assumed to be due to weakness of the intestinal musculature secondary to these conditions.

When the abdomen is constantly distended and tympanitic, and the patient complains of attacks of colic, which are relieved by the passage of flatus, it may be assumed that the constipation is due, in part at least, to the incapacitating effect of distention on the intestinal musculature. The Flatulence (p. 240) may be primary, or it may be secondary to the constipation, in which case some other cause of the condition must be looked for.

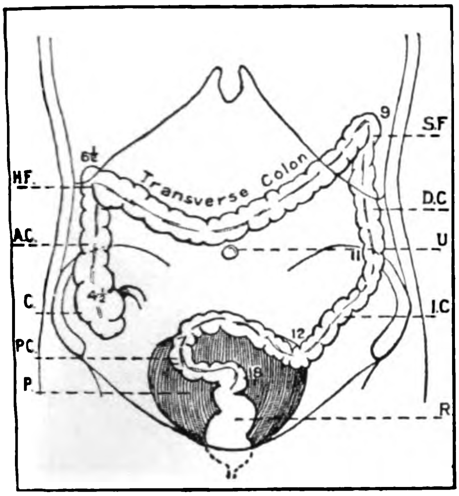


Fig. 49.—Diagram of the normal large intestine. The numbers, in this and the following figures, represent the hours after a bismuth breakfast at which the different parts of the colon are reached. **C** Cæcum; **AC**, Ascending colon; **HF**. Hepatic flexure; **SF**. Splenic flexure; **DC**, Descending colon: **IC**, Iliac colon; **PC**, Pelvic colon; **R**, Rectum: **U**. Umbilicus; **P**, Pelvis.

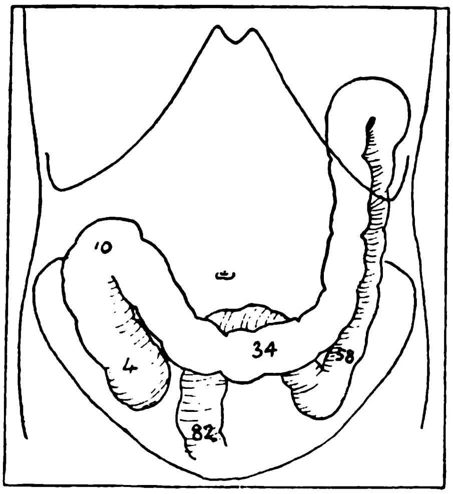


Fig. 50.—Post-dysenteric atony and paresis of the colon. Compare the lumen of the colon and the slow passage of fæces through it with *Fig.* 49.

The constipation of fat people is due in part to the inefficiency of the intestinal musculature resulting from fatty infiltration.

In some of these conditions atony of the colon can be recognized with the x-rays by its abnormally large lumen, in addition to the slow passage of fæces (*Figs.* 49 and 50).

(*b*). **Deficient Reflex Activity of the Intestines.**

Insufficient Stimulation of Intestinal Movements.—Careful enquiry should be made into the patient's diet and habits, as many cases are due to too little food being taken, or to the food containing too little mechanical or chemical peristaltic stimulants, and some are due to deficient exercise. Other cases result from a 'greedy colon,' the absorption of food being unusually complete. In spite of enough food of a sufficiently stimulating character being taken, and in spite of the fact that the abdomen is retracted and no accumulation of fæces can be felt in either the colon or the rectum, yet a very deficient quantity of fæces is excreted. This is the type of case in which benefit results from the use of agar-agar or petroleum. In constipation due to an unsuitable diet or to a greedy colon the stools are generally small, dark, and dry, and smell less strongly than normal. In œsophageal or pyloric obstruction constipation is always present owing to the small quantity of food-residue which reaches the colon. The other symptoms generally prevent a mistake in diagnosis being made; but occasionally in pyloric obstruction the patient complains of nothing but some slight indigestion or weakness in addition to the constipation. The passage of a stomach-tube twelve hours after a large meal, when the stomach should be completely empty, and an x-ray examination, will clear up the diagnosis in doubtful cases.

Deficient Sensibility of the Intestinal Mucous Membrane.—This is the probable cause of the constipation when there is a history of excessive tea-drinking or of the long-continued use of large doses of aperients ; it is also partly responsible for the constipation associated with catarrhal colitis—in which excess of mucus is passed with the stools—whether this is primary or a result of constipation due in the first instance to some other cause.

Depression of the Nervous System.—In neurasthenic, hypochondriac, and insane patients, the condition of the nervous system is the chief cause of the constipation which is almost invariably present ; but an improper diet is generally an additional factor.

(*c*). **Inhibition of the Motor Activity of the Intestines.**—This group of cases can often be recognized by the fact that sedatives, such as opium and belladonna, give relief, whilst purgatives are required in unusually large doses, and produce an unusual amount of colic unless given with a sedative. The x-rays show that the small intestine as well as the colon is traversed slowly ; this is unusual in other forms of constipation (*Fig.* 51). Inhibition may be *direct*, *central*, or *reflex*.

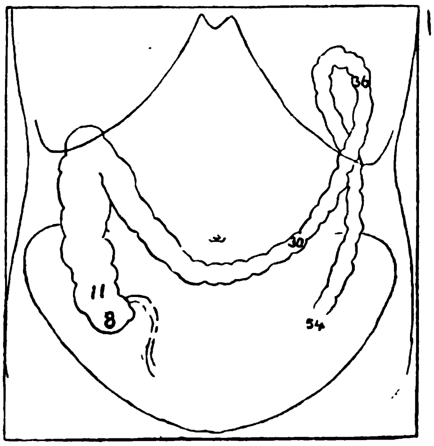


Fig. 51—Constipation due to lead poisoning. The passage through the small intestine as well as the colon is slow, owing to the inhibitory action of the splanchnic nerves.

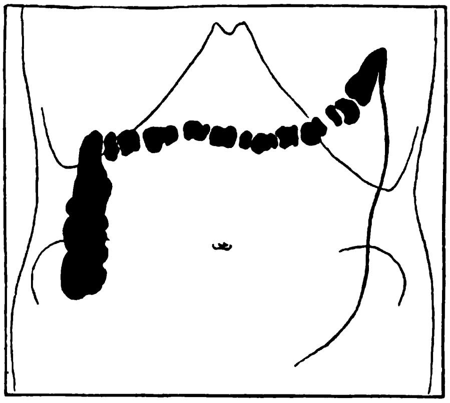


Fig. 52.—Constipation with muco-membranous colitis, showing spasm of descending and iliac colon.

Direct Inhibition in Lead Poisoning.—The diagnosis is suggested by the occupation of the patient, a blue line on his gums, or other symptoms of plumbism (p. 34).

Central Inhibition.—A history of a recent shock or worry is obtained.

Reflex Inhibition.—Constipation is a frequent symptom of painful diseases of abdominal and pelvic viscera, other than the intestines themselves. It can then be cured only by treating the primary condition, so that it is essential to ascertain the cause of the pain. Constipation is particularly liable to result from disease of the vermiform appendix, female genital organs, stomach, duodenum, and gall-bladder.

(*d*). **Irregular Spasmodic Contraction of the Intestine: Spastic Constipation: Enterospasm.**—When constipation is associated with pain, especially if the pain comes on in attacks during which the difficulty with the bowels is increased, the possibility that it is due to spasm of the colon must be considered. The pain is situated in the course of the large intestine, most frequently in the iliac and pelvic colon, but occasionally in other parts. The affected parts of the colon can generally be felt as a contracted, tender cord, in which scybala may be detected and the narrow lumen can be recognized with the x-rays (*Fig.* 52). When the pain is in the right iliac fossa appendicitis may be simulated ; the long duration of the attacks without any pyrexia, the occasional history of similar pain on the opposite side, and the contracted condition of the ascending colon and sometimes of the cæcum (though in other cases the cæcum may be distended and tympanitic), are distinctive features of spastic constipation. When the pain is in the left side, a tumour of the descending or iliac colon may be suspected : the long history, the absence of visible or palpable peristalsis and of distention above the contracted part, and the absence of occult blood from the stools, are points which distinguish spastic constipation from cancer of the colon. In cases of spastic constipation the stools should always be examined for mucus, as the spasm, especially when it occurs in neurotic women, is often only a symptom of muco-membranous colitis, shreds or membranes of coagulated mucus being passed by the patient (p. 398).

2. Constipation due to Excessive Force required to carry the Fæces to the Pelvic Colon may be due to :

(*a*). **Obstruction by Fæces.**—Dry, hard fæces, which require abnormally strong peristalsis to carry them to the pelvic colon, result from : (i) Insufficient consumption of water—a common cause of constipation in women ; (ii) Excessive loss of water by other channels—one cause of the constipation of diabetics, and of individuals who perspire freely and are only constipated in hot weather.

(*b*). **Narrowing of the Intestinal Lumen.**

Organic Stricture.—Unless this is due to a palpable tumour it may be very difficult to distinguish from constipation due to less serious causes. More or less colic is generally present, and its situation often gives a clue to the localization of the obstruction. An *x*-ray examination, when the barium is given by mouth, rarely gives any help in the early stages of the disease, although occasionally the actual narrowing of the intestine is observed and stasis occurs in the proximal portion of the bowel (*Fig.* 53). Much more valuable information can be gained by the administration of a barium enema. Six ounces of barium sulphate are suspended in a pint and a half of water to which has been added an ounce and a half of acacia mucilage and an ounce of methylated spirits. The fluid is run slowly into the bowel from a funnel at a pressure of one foot. In normal individuals some of it reaches the cæcum almost immediately, but even in the early stages of organic obstruction the passage is more or less obstructed at the seat of the stricture, owing apparently to a superadded spasm.

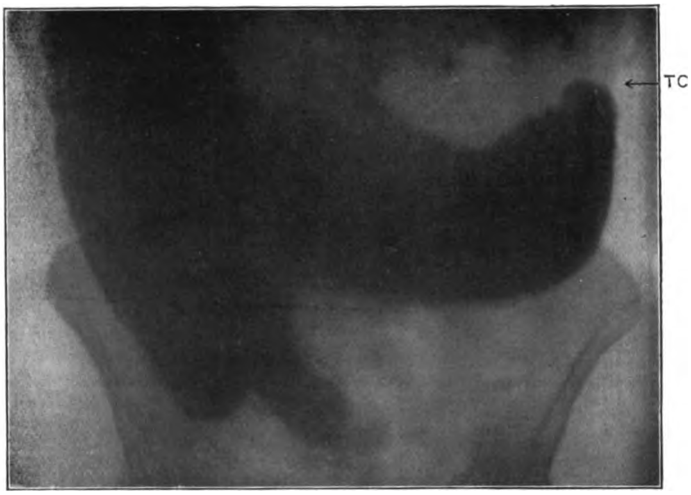


Fig. 53.—Skiagram to show the arrest of bismuth by a malignant stricture of the last 3 or 4 inches of the transverse colon. At the subsequent operation a very hard stricture of this exact portion of the transverse colon was found and excised. The nature of the condition was quite unsuspected until bismuth and the *x*-rays were employed : gastric trouble, possibly an ulcer, was thought probable previously. Male, age 33. TC. Complete arrest of food in transverse colon twenty-seven hours after it was given by mouth. (*Skiagram by Dr. C. Thurstan Holland.*)

Non-malignant strictures of the colon are rare. If there is a history of tuberculous or dysenteric ulceration, the possibility of obstruction due to cicatrization should be considered, though this is a very unusual occurrence. Hyperplastic tuberculous infiltration of the intestine, especially of the cæcum, causes obstruction, but the tumour present is difficult to distinguish from cancer. Obstruction to the iliac or pelvic colon may follow the pericolitis which results from the formation of *diverticula* in old people who have long suffered from constipation. This condition may also be indistinguishable from a growth, but the possibility should be borne in mind in the case of elderly patients with a tumour in the iliac or pelvic colon, where there is a long history of constipation : the sigmoidoscope may help in the diagnosis. If a vesico-colic fistula develops in association with chronic constipation, it should be remembered that pericolitis due to ulceration of diverticula is a more frequent cause of this condition than cancer.

Organic stricture of the colon is most commonly due to *cancer*. The possibility of cancer should always be considered when an individual above the age of forty, whose bowels have been regular previously, develops constipation of increasing severity without change of diet or habits, or when a patient who is habitually constipated becomes more so without obvious reason. The constipation is at first intermittent and may alternate with diarrhœa ; drugs become steadily less effective, and enemata, which at first give greater relief than drugs, also lose their effect slowly. A tumour is often not palpable, but an examination under an anæsthetic reveals the presence of one in many doubtful cases, especially in fat individuals. The tumour may vary in size, and even disappear after the bowels have been opened well, because a mass of fæces may become impacted above a cancerous stricture which is itself impalpable. Hence, although the presence of a tumour is an important aid in diagnosis, its absence or disappearance does not exclude the possibility of cancer ; only when its disappearance under treatment is accompanied by complete and lasting cure of all symptoms can cancer be excluded. The tumour is hard, and cannot be altered in shape by pressure, as is the case with fæcal tumours. Slight attacks of colic occur frequently, but they are not often severe until the obstruction is almost complete ; the colic may be accompanied by visible and palpable peristalsis and spasmodic contractions of the intestine. The latter is a most important sign, as it never occurs in colic associated with lead-poisoning or colitis, and very rarely with obstruction due to fæcal impaction. Progressive loss of weight and strength, anorexia, and anæmia are late symptoms, and it is important to make a correct diagnosis before they have appeared. The obvious presence of blood in the fæces is an important symptom, but it is often absent. Much more frequently traces are found which are only recognizable by chemical tests (p. 81). In the absence of hæmorrhoids and of hæmorrhage from the mouth, throat, or nose, the presence of 'occult' blood in the fæces is strong evidence that ulceration is present in the stomach or intestines ; when symptoms pointing to gastric or duodenal ulcer and gastric carcinoma are absent, and constipation is present, a suspicion of cancer of the intestine receives important confirmation. In doubtful cases a sigmoidoscopic examination should be made, as cancer is much more common in the rectum and pelvic colon—which alone can be investigated by this method—than in any other part of the intestine.

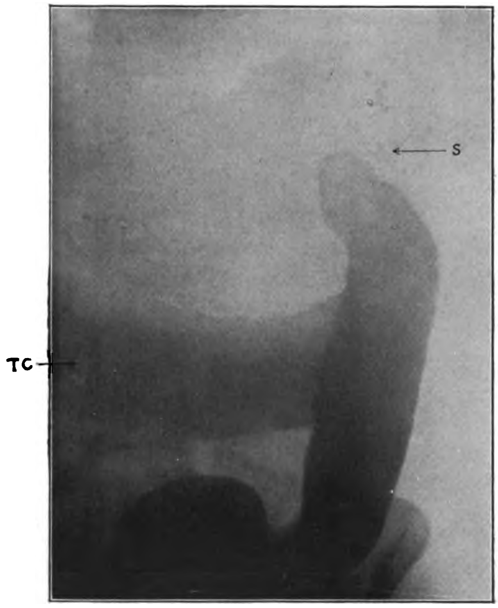


Fig. 54.—The same case as *Fig.* 53. Skiagram after the administration of a bismuth enema seven days after bismuth had been given by the mouth. Arrest of the bismuth by the obstructive carcinoma at the end of the transverse colon. TC. Food still in the dilated transverse colon seven days after it had been given by mouth. S. Splenic flexure: Complete arrest of bismuth enema.
(*Skiagram by Dr. C. Thurstan Holland.*)

A *kink* of the colon is a very unusual cause of constipation. It is sometimes partly responsible for the constipation which is almost always present in visceroptosis (*Fig.* 56), and it should be suspected when an attack of localized peritonitis, due particularly to disease of the female genital organs, appendicitis, or leakage from a gastric or duodenal ulcer, is followed by constipation. An *x*-ray examination should, however, always be made before advising surgical treatment, as, in the vast majority of cases, even if adhesions are present they have nothing to do with the obstruction. The *x*-rays show whether the delay takes place in the neighbourhood of the supposed adhesions, and the presence or absence of adhesions can also be ascertained by seeing how movable the colon is, and whether the two limbs of the various flexures can be separated from each other.

Whatever may be the primary cause of *Hirschsprung's disease* (wrongly called "congenital idiopathic dilatation of the colon"), it is probable that a kink is produced after the dilatation has reached a certain degree by the overhanging of the dilated part of the colon over the undilated section (*Fig.* 55). There is always a history of constipation dating from the first few months of life, although sometimes the bowels may be opened daily but insufficiently. Soon after birth the abdomen becomes greatly enlarged, the size varying from time to time. The outline of the distended colon can be seen, and peristalsis is often visible. The abdomen finally becomes enormous; it is then tense and tympanitic. Attacks of obstruction are liable to occur, and death takes place most frequently between the ages of three and eight.

When a *large abdominal tumour* is present, constipation may be due to its pressure on the colon.

Chronic intussusception may cause symptoms similar to those produced by a stricture; attacks of colic accompanied by visible peristalsis occur with increasing frequency and severity, and they are often brought on by food or aperients. An intussusception should be suspected under these circumstances when a sausage-shaped tumour is palpable, especially if blood and mucus are passed at frequent intervals. In one-third of the cases the apex of the intussusception can be felt on rectal examination.

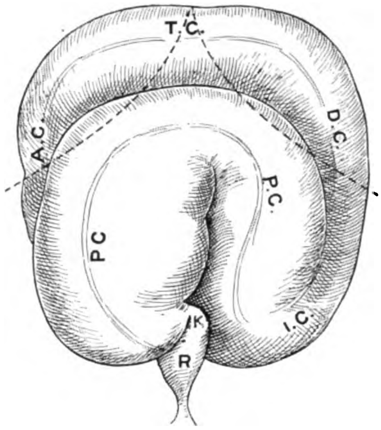


Fig. 55.—Colon in a case of Hirschsprung's Disease. AC, Ascending colon; TC, Transverse colon; DC, Descending colon; IC, Iliac colon; PC, Loop of pelvic colon; R, Rectum; K, Kink at pelvi-rectal junction. The dotted lines represent the costal margins.

C.—DIAGNOSIS OF THE CAUSE OF DYSCHEZIA.

Dyschezia is due to a want of proper proportion between the power of expelling the fæces from the pelvic colon and rectum, and the force required to do this completely. It may therefore be due to (1) *Inefficient Defœcation*; or (2) *An Obstacle to Efficient Defœcation.*

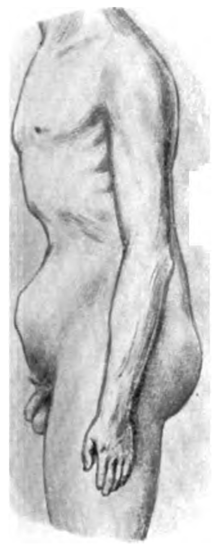


Fig. 56.—Visceroptosis.

1. INEFFICIENT DEFÆCATION may be due to :—

(*a*). **Weakness of the Voluntary Muscles of Defæcation.**—This should be suspected when constipation dates from pregnancy, or is associated with ascites, large abdominal tumours, or obesity. It is often easy to ascertain the condition of the abdominal muscles by simple palpation in the horizontal position; the discovery of a movable kidney or a dropped liver would also suggest that the abdominal muscles are weak. The patient should next be told to raise her head from the couch; the recti muscles contract and their strength can be ascertained, and any separation between them recognized. Finally, the patient should be examined standing up; bulging of the abdomen below the umbilicus (*Fig.* 56) shows that visceroptosis is present and that the abdominal muscles are weak. The patient often complains of abdominal discomfort, which is relieved by lying down or by pressing the lower part of the abdomen upwards. In all cases in which a woman, whose bowels have previously been regular, becomes constipated after the birth of a child, the condition of the pelvic floor should be investigated, as well as that of the abdominal wall. The anus is normally slightly retracted; the retraction is increased and the anus moves slightly forward when the levator ani muscles are contracted by making the movement which is required when it is attempted to restrain a commencing defæcation. If they are weak, the retraction in the condition of rest is absent or diminished, and on contracting the levator ani muscles, the retraction and forward movements are slight or absent. On straining, the whole perineum projects much further than it should do, and in severe cases the uterus may be more or less prolapsed: in such cases no further evidence is required to show that the dyschezia is partly due to weakness of the levator ani muscles.

When constipation is present in asthmatic or very emphysematous people, it is partly due to the fact that the great rise in intra-abdominal pressure required in defæcation cannot be produced by contracting the diaphragm, as the latter is already almost as low as it can go.

(*b*). **Habitual Disregard of the Call to Defæcation.**—When dyschezia is not associated with weakness of the muscles of the abdominal wall or pelvic floor, the history will generally show that it has resulted from habitual disregard of the call to defæcation—a very common cause in girls, and a not uncommon one in schoolboys and business men, who allow themselves too little time between getting up and beginning the day's work. The call is also often neglected if for any reason defæcation is painful.

(*c*). **Unfavourable Posture during Defæcation.**—Enquiry should be made as to the height of the seat in the water-closet, as when this is too high it is impossible to assume the proper crouching position, and defæcation may consequently be inefficient. Weakness of the voluntary muscles of defæcation, habitual disregard of the call, and the assumption of an unsuitable position during the act, all lead to the same results—the loss of the defæcation reflex, and atony and paresis of the musculature of the pelvic colon and rectum. The loss of the defæcation reflex is shown by the fact that the patient never experiences a desire to defæcate, even when examination shows that the rectum is full of fæces. The atony of the rectum is shown by its abnormally large size and the very slight resistance offered when the finger presses upon its walls; the atony of the pelvic colon is shown by the abnormally large shadow it forms when examined with the *x*-rays (*Fig.* 48, p. 122). The paresis of the pelvic colon and rectum is shown by the patient's inability to defæcate by an effort of will, when the rectum is full of fæces.

(*d*). **Primary Weakness of the Defæcation Reflex.**—This is sometimes the cause of constipation in infants; it is probably the case when defæcation occurs on exaggerating the natural stimulus by the mechanical effect of the introduction of a finger into the rectum, or by the combined mechanical and chemical effect of the introduction of a piece of soap.

(*e*). **Organic Nervous Diseases.**—When constipation occurs in the course of organic nervous diseases, such as *tabes dorsalis*, *myelitis*, or *meningitis*, it is due to disturbance in the defæcation centre in the lumbo-sacral cord or the tracts connecting it with the brain. When constipation and difficulty in micturition appear simultaneously, the possibility of some organic nervous disease should be considered, even if no other symptoms are present.

(*f*). **Hysteria.**—When dyschezia occurs in nervous individuals it is often due to the patient having suggested to himself that he cannot open his bowels at all, or unless he takes a purgative or an enema. The diagnosis can be confirmed by the result of treatment: if such a patient can be persuaded after a thorough examination that there is really no reason whatever why he should not obtain a daily action of the bowels without artificial aid, he will have no difficulty in curing himself at once.

2. OBSTACLES TO EFFICIENT DEFÆCATION may be due to:—

(*a*). **Hard and Bulky Fæces.**—When the fæces are abnormally hard as a result of intestinal constipation or of the excessive loss of fluid from diarrhœa, hæmorrhage, or other cause, the force required to expel them may be so great, especially if they are bulky, that dyschezia results. This condition can be recognized easily by a rectal examination, which shows that fæces of abnormal hardness are impacted in the rectum.

(*b*). **Spasm of the Sphincter Ani.**—When defæcation is painful it is rendered difficult as well by reflex spasm of the sphincter ani. The anal canal and rectum should be examined after the introduction of a cocaine suppository, or if necessary under a general anæsthetic, so that any local cause of the pain, such as an anal ulcer or inflamed hæmorrhoids, may be discovered. In the absence of these, the genito-urinary organs should be examined thoroughly for reflex causes of spasm.

(*c*). **Organic Stricture of the Rectum and Anus.**—In every case of constipation a digital examination of the rectum should be made, and in cases of doubtful origin the rectum and pelvic colon should be examined with a proctoscope and sigmoidoscope. *Congenital* *narrowness of the anal canal* is recognized easily ; it is rare, but may give rise to no symptom until several years after the child is born. *Fibrous stricture of the rectum* is an occasional cause of dyschezia, especially in women ; it results from inflammatory infiltration of the submucous tissue secondary to infection of an abrasion of the mucous membrane. The condition is generally painful, and often associated with active inflammation and ulceration ; it can be distinguished readily from malignant stricture by means of the proctoscope. *Cancer of the rectum* or *pelvic colon* is a common cause of dyschezia ; when constipation develops after the age of forty without any obvious cause, especially if it is accompanied by a sense of fullness in the rectum and of incomplete relief after defæcation, by loss of weight and strength, or by discharge of mucus and blood, the possibility of cancer of the rectum should always be considered, and a thorough examination made by the finger and proctoscope or sigmoidoscope.

(*d*). **Pressure on the Rectum from Without.**—Pressure on the pelvic colon and rectum by a *gravid uterus* always produces some dyschezia. Apart from this the possibility of a pelvic tumour, such as *distended tubes*, *cancer*, or *fibroid of the uterus*, and *ovarian tumours*, should be remembered in dyschezia occurring in women, especially if there is any pelvic pain. A retroverted but otherwise normal uterus cannot be regarded as a sufficient explanation of dyschezia.

(*e*). **Invagination.**—When a constipated patient, whose general health is so good that cancer seems improbable, complains that after defæcation he feels as if something were still present in the rectum, especially if mucus and occasionally a little blood are passed, the dyschezia may be due to obstruction caused by the invagination of the mucous membrane of the upper part of the rectum into the lower part. The condition is generally associated with lumbar pain. The invaginated mucous membrane can be felt on digital examination, especially when the patient strains.

II. ACUTE CONSTIPATION.

Acute constipation may be (A) *Due to acute intestinal obstruction* ; or (B) *A symptom of* (*a*) *some general disease*, or (*b*) *some other acute abdominal disease.*

A.—ACUTE INTESTINAL OBSTRUCTION.

1. The following points help in the distinction between acute intestinal obstruction and severe cases of acute constipation of other origin : (i). *Visible and palpable peristalsis* or stiffening of the intestines is never present except in obstruction. (ii). *Vomiting* is never fæculent, except occasionally at a very late stage, in non-obstructive cases. (iii). In other conditions the *constipation is incomplete* :—

(*a*). Flatus, and even a small quantity of fæces, may be passed spontaneously.

(*b*). A purgative may give a result ; it is, however, very unwise to administer purgatives in such cases, but frequently the patients have already tried them on their own responsibility.

(*c*). A rectal examination should always be made. In organic intestinal obstruction the rectum is empty ; if it contains fæces there may be obstruction due to fæces, but it is exceedingly rare for this to produce symptoms at all comparable in severity with those due to acute obstruction. With this exception, the presence of any quantity of fæces would show that there was no intestinal obstruction.

(*d*). In doubtful cases two enemata should be given, with an interval of an hour : the first generally brings away a certain amount of fæces, even if obstruction is complete ; the second only results in the passage of fæces or flatus if there is no complete obstruction, or if the obstruction is high in the small intestine. If there is complete obstruction the second enema is either retained or escapes unaltered and with abnormally small force.

2. Before considering any other possibility, all the hernial apertures should be examined, even in the absence of local pain, as a *strangulated hernia* gives all the signs of acute intestinal obstruction.

3. The following points should be considered in determining the cause of the acute intestinal obstruction :—

(i). *Age.*—Intestinal obstruction in the new-born is almost invariably due to a congenital malformation: as this is generally in the rectum (p. 586) the latter should be examined first, and only after it has been found to be normal should the possibility of congenital obstruction in the duodenum or ileum be considered. In infants the common cause of intestinal obstruction is intussusception ; at a somewhat older age obstruction may arise in connection with a Meckel's diverticulum ; but in children and young adults the most common cause is obstruction by bands or adhesions resulting from local peritonitis, due to appendicitis, tuberculous peritonitis, or caseous mesenteric glands. Acute obstruction occurring in an infant or child under ten years of age, in whom there is a history of constipation and abdominal distention dating from soon after birth, is most probably due to Hirschsprung's disease (p. 126). After the age of forty the possibility of cancer of the colon should always be remembered, and in fat patients, especially women, obstruction by gall-stones. In patients over sixty acquired diverticula of the colon are likely to give rise to symptoms and signs which are generally mistaken for cancer.

(ii). *History.*—A previous attack of appendicitis, or a history of tuberculous peritonitis, or of inflammatory pelvic disease in females, suggests the possibility of obstruction by bands or adhesions ; the same diagnosis should be considered if the patient has some weeks or months before had a strangulated hernia reduced. A history of biliary colic or of the less striking symptoms which may result from cholelithiasis indicates that obstruction may be due to impaction of a gall-stone. When acute obstruction follows a period of increasing constipation in middle-aged patients, cancer is probably present.

(iii). *State of the Bowels.*—The passage of blood and mucus without any fæces is very suggestive of an intussusception. In older patients it may be due to cancer. The passage of stools during the early stages, in spite of other evidence of obstruction, indicates that the latter is situated in the small intestine.

(iv). *Abdominal Examination.*—

(*a*). *Distention.*—Great distention generally means that the obstruction is in the colon : if it is present very soon after the onset of symptoms, it is probably due to cancer or volvulus ; if it has been present to a less extent for some time before the onset of acute symptoms, a growth is likely ; but if it has developed very acutely, a volvulus is more probable. In infants and small children great distention suggests Hirschsprung's disease (p. 126), if the abdomen is tympanitic ; if it is partially dull, and if free fluid or irregular masses are present, tuberculous peritonitis is the probable diagnosis. Well-marked distention in both flanks suggests origin in the pelvic colon or rectum ; if in the right flank only, in the hepatic flexure or transverse colon ; if the flanks are comparatively undistended and the central part of the abdomen is most affected, the obstruction is likely to be in the ileum or the cæcum ; distention is slight when the obstruction is in the duodenum or jejunum.

(*b*). *Visible Peristalsis and Stiffening of the Intestine.*—The position and direction of visible peristalsis and the position of stiffening coils of intestine may show the localization of the obstruction. When a series of more or less parallel contracting coils is visible in the central part of the abdomen, the obstruction is in the small intestine ; if it appears to culminate in the right iliac fossa, this is likely to be the seat of disease. Stiffening of a length of intestine, which can be seen to rise up and felt to harden, most often occurs in the colon, and especially when there is a growth near its lower end. The most marked peristalsis and stiffening occur when acute obstruction is a sequel of chronic obstruction ; they may be completely absent in very acute primary cases.

(*c*). *Tumour.*—The diagnosis of intussusception can be made with certainty only when the characteristic sausage-shaped tumour situated somewhere in the course of the colon is felt. In acute obstruction due to cancer the tumour is often not palpable, as it is generally hidden by the dilated intestine ; but large tumours are felt sometimes, especially when present in the right or left iliac fossa : the former are generally due to cancer of the cæcum, the latter to cancer of the iliac colon and inflammatory thickening round acquired diverticula —a condition which may closely simulate cancer. Gall-stones can hardly ever be felt.

(v). *Rectal Examination.*—A growth of the rectum can be recognized easily, and sometimes a growth of the pelvic colon can be felt through the front wall of the rectum. In infants, the end of an intussusception may be felt in the lumen of the rectum, and more frequently the tumour can be felt on bimanual examination. Obstruction due to pelvic adhesions can often be recognized by the presence of tender masses and the fixity of some of the pelvic viscera. The presence of more than traces of fæces in the rectum in cases of undoubted obstruction indicates that its situation is probably high up in the small intestine. A very ballooned rectum suggests obstruction high up in the rectum or in the pelvic colon, but this is not an invariable rule.

(vi). *Pain.*—When the pain is localized, or moves in a definite direction to reach its greatest severity at a certain point, the latter is likely to be near the seat of the obstruction. When the pain is situated in the middle line, the obstruction is probably in the small intestine if it is above the umbilicus, and in the colon if below.

(vii). *Vomiting.*—The more frequent the vomiting and the earlier the onset of fæculent vomiting, the higher in the intestine is the obstruction likely to be. It is most severe in small intestine obstruction due to bands or internal hernia ; its onset is later and its occurrence less frequent and sometimes only after food in cases of growth and volvulus.

(viii). *Borborygmi* are sometimes most marked over the seat of the obstruction.

(ix). *Shock* and *Collapse* are more marked the higher the obstruction. They are also much greater when obstruction is accompanied by strangulation owing to bands or hernia than when strangulation is absent, as with gall-stones and cancer.

B. SYMPTOMATIC.

In Acute General Diseases.—Constipation beginning acutely is a frequent symptom of a large variety of acute infective and other diseases. It is never so severe that it cannot be overcome by purgatives or enemata, and the other symptoms are so much more striking in the majority of cases that the presence of constipation has little influence in forming a diagnosis.

In Acute Abdominal Conditions.—Constipation is a prominent symptom in most acute abdominal conditions. Other symptoms are often so well marked that the question of intestinal obstruction hardly arises. Thus, the diagnosis can generally be made by the early tenderness and rigidity, its localization, and the early pyrexia in acute peritonitis due to appendicitis or the perforation of an ulcer ; the characteristic situation and radiation of the pain in renal and biliary colic, and the frequent hæmaturia in the former and jaundice in the latter ; the presence of a tumour when an ovarian cyst is twisted ; the melæna and occasional hæmatemesis, and the presence of a primary disease in the heart or abdomen in *mesenteric embolism* and *thrombosis* respectively. Some cases of *acute pancreatitis* are clinically almost indistinguishable from intestinal obstruction, but flatus is generally passed ; there may also be a history of biliary colic, and the patient is generally fat, middle-aged, and alcoholic. The diagnosis is seldom made with certainty until the typical fat-necrosis is seen on opening the abdomen. In *lead colic* the constipation is not absolute, and the occupation of the patient and the blue line on the gums (p. 34) suggest the correct diagnosis.

Arthur F. Hurst.

CONTRACTIONS, Athetotic, Choreiform, Fibrillar, Spasmodic, and Tetanic—are all to be defined for present purposes as involuntary and painless contractions occurring in the voluntary muscles. From CONTRACTURES (p. 138) they may be distinguished by their short duration, longer or shorter intervals in which the affected muscles are relaxed occurring between the separate contractions. From *cramps* they differ by being painless, or comparatively so, and also by their short duration. But in many cases it is impossible and also unnecessary to draw any hard-and-fast line showing where, for example, tetanic contractions cease and tetanic cramps begin. In all cases the occurrence of the contractions mentioned above may be taken to indicate some disease of the nervous system, usually organic but sometimes functional.

ATHETOTIC CONTRACTIONS, ATHETOSIS, OR MOBILE SPASM.

Athetosis is a form of involuntary movement affecting the fingers, hands, and wrists most often ; less often the toes and feet, and in rare instances the face. It is usually unilateral, but in exceptional cases bilateral—the 'double athetosis' of French neurologists. The movements are spontaneous and incessant, and may even continue while the patient is asleep ; in other instances they tend to cease, but are started anew or exaggerated when voluntary movement is attempted. In the hand, the movements consist of a succession of slow and serpentine flexions, extensions, hyperextensions, and lateral motions, all combined to cause the fingers and thumb to execute the most curious and complex clutching or spreading movements (*Fig.* 57). The wrist is held more or less flexed; the fingers may move about together, or wander each individually. Analogous movements are observed when athetosis occurs in the lower extremity, or the mouth and face. No great regularity characterizes the motions of athetosis; as a rule they are steady rather than violent; a large amount of voluntary control over the affected parts is retained. Mobile spasm is due to varying degrees of central irritation of muscles that are incompletely paralyzed and somewhat spastic.

Primary, idiopathic, or *primitive athetosis* is a rare disease of childhood or of adult life, in which bilateral athetotic contractions first make their appearance in a previously healthy person, either for no particular reason, or after a chill or a nervous shock. It may be associated with epilepsy or insanity. This form appears not to be connected with any gross changes in the nervous system, thus differing from all other conditions in which athetosis is seen.

Athetosis is common in the various *spastic paraplegias of infants and children*, which may be either congenital or acquired: in **Congenital cerebral diplegia,** also known as *Little's disease* when the legs are affected chiefly, the nervous structures suffer from an inherited taint (alcoholism, syphilis, insanity), and either fail to develop properly, or degenerate early in life. The onset of Little's disease is gradual, and usually early, but it may be delayed until the child is as much as six or eight years old. The patient is backward or mentally deficient, probably unable to walk, and afflicted with bilateral spastic paralysis. This may affect the legs, the legs and arms, or even the whole body, and may be more marked and more spastic on one side of the body than on the other; speech is defective, optic atrophy common, and the gait is clumsy and stiff, 'cross-legged' or 'scissor.' Involuntary movements occur in the affected members, and are athetotic or choreiform; tremor or intention-tremor is also not infrequent. Although it may not appear for some years after birth, this is really a congenital disorder, and it is to be distinguished, for reasons connected with its pathological anatomy and etiology, from certain other forms of spastic paralysis in infants and children that may closely resemble it clinically. These are the *acquired cerebral paralyses of infants*, the spastic infantile hemiplegias, monoplegias, diplegias, triplegias, paraplegias, that result from more or less localized cerebral inflammations or hæmorrhage occurring at birth or in infancy. *Porencephaly*, or the occurrence of lacunæ in the tissues of the cortex or brain, may be found in either the congenital or the acquired cerebral paralyses; it is really a post-mortem-room term, and requires no special consideration here.

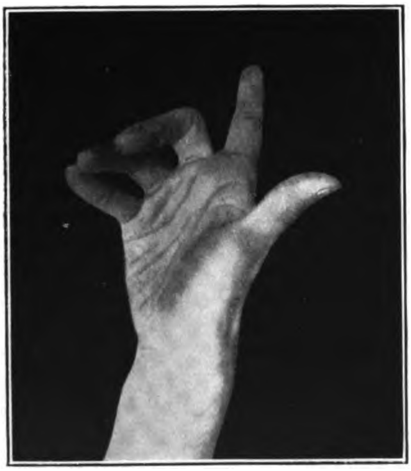


Fig. 57.—The hand in athetosis.

Acquired spastic paraplegias fall into two categories, according to their etiology:—

1. *Birth palsies:* due to meningeal or cortical hæmorrhage caused by prolonged labour or the use of instruments. Many of these infants have been born prematurely.
2. *Acquired palsies:* due to—
    Encephalitis after an acute specific fever, or infective in origin.
    Polio-encephalitis, the cerebral analogue of acute poliomyelitis in the anterior cornua of the cord.
    Cerebral embolism.
    Cerebral or meningeal hæmorrhage or thrombosis.

The *birth palsies* are due to injuries received in the process of birth, and the rupture of meningeal or cerebral blood-vessels, with the escape of blood; they develop at once, and the history of the case should make diagnosis easy. The diagnosis of the exact cause of an *acquired spastic paralysis* in an infant or child may be less easy. The paralysis due to *encephalitis* generally appears during the first two or three years of life, but may come on at almost any age. *Cerebral thrombosis* in children is said to happen oftenest at about the age of six. *Cerebral embolism* is likely to be seen in infants or children with acquired heart-disease, the embolus being derived from vegetations on the mitral or aortic valves, or from thrombi that have formed in backwaters of the dilated left auricle or ventricle. These infantile hemiplegias or diplegias are of sudden onset, and are characteristically spastic. Athetotic movements, with or without choreiform contractions, trophic lesions, and tremors, are common in the affected limbs; the children often grow up to exhibit mental defect, imperfect speech, or epilepsy. As a rule, the face is less involved than the arm or leg, and the athetotic movements, confined to the affected parts, may not begin until years after the occurrence of the original cerebral lesion.

Post-hemiplegic athetosis, which cannot be marked off sharply from post-hemiplegic chorea (see p. 134), is an uncommon sequela of hemiplegia in the adult; but common—being seen in about a third of the cases—in the congenital and acquired hemiplegias just considered. In the adult it occurs oftenest when the lesion is situated near the posterior part of the internal capsule or the optic thalamus. These athetotic movements of the extremities have been described already; in the adult, they may be combined with choreiform contractions involving the whole arm and shoulder, and the face. The diagnosis should not be difficult, as the history of a stroke will be obtained and the physical signs of a hemiplegia will be present.

CHOREIFORM CONTRACTIONS.

These are similar to the contractions seen in chorea. They are involuntary and inco-ordinated movements, purposive in character, but aimless and ineffective in performance. They are jerky, rapid, and highly irregular; groups of muscles are put into action successively, as if the original intention were given up, or changed, as soon as the complex movement began. They may affect one side of the body only, or both. When mild, they amount to no more than excessive fidgetiness, involving perhaps only the hands and arms, or the hands, arms, and face, in wriggling and grimacing. When severe, they give the patient no rest; he is tossed about, perhaps with the utmost violence, by combined but irregular contractions, in which any of the voluntary muscles may participate. Choreiform contractions bear no resemblance to *tremors*, whether coarse or fine. From *intention-tremors* they are distinguished by the facts that they continue when the patient is at rest, that they are purposive, and resemble ordinary voluntary movements misapplied. From *ataxia* they are distinguished by occurring at rest as well as on attempted movement; the muscular contractions of ataxia are merely inco-ordinated, apparently ill-designed and clumsily executed, types of normal movements.

Choreiform contractions are seen in the following conditions:—

Chorea minor, or St. Vitus's dance; chronic or Huntington's chorea; chorea major, or pandemic chorea; hysteria.

Pre-hemiplegic chorea; post-hemiplegic chorea; spastic paralyses of infants; cortical sclerosis; chorea electrica (Henoch).

Chorea, chorea minor, acute chorea, or *St. Vitus's dance*, is an acute disease of childhood or adolescence, commoner in girls than boys, and closely connected with a history of rheumatism, and with rheumatic endocarditis. Like rheumatism, it is often a family disease; not infrequently one finds that one or two children in a large rheumatic family have had rheumatic fever or rheumatism, another chorea, and another both rheumatic fever and chorea. It is commonly and erroneously held that severe fright may by itself be the cause of an attack of chorea. It may also occur in adults in connection with pregnancy, when it is sometimes of a severe type, and may run on into insanity. The movements may be confined to one side of the body—hemichorea—or may affect both sides; the muscles are in general weak, speech may be interfered with, respiration is often jerky, and the patient is often unduly irritable and emotional. Except in the severest cases the movements cease during sleep; the disease tends to recovery in the course of perhaps two or three months. Mild cases in which the face is most affected may present a certain resemblance to the more chronic and quite unconnected disorder known as *habit-spasm, habit-chorea*, or *convulsive tic* (see SPASMODIC CONTRACTIONS, p. 136). A facial tic is controlled for a time by strong efforts of the will, whereas the facial movements of chorea will usually be increased by the concentration of the attention on them; the facial movements of chorea are irregular, representing a succession of various purposive but uncompleted actions, while the facial tic consists in the repetition of a single definite and purposive movement, originally designed, no doubt, to give relief to some local irritation.

Chronic, degenerative, or *Huntington's chorea,* is a rare hereditary disease coming on at the age of thirty or forty, associated with slow and difficult speech and with insanity. The involuntary movements are slower and more ataxic than those of acute chorea, and can often be suppressed for a time by exercise of the will. They affect the extremities and face, are continuous, cease during sleep, and are accentuated by excitement, so that at first sight acute chorea may be imitated fairly closely. The diagnosis between this chronic chorea and an acute chorea that had become chronic, as sometimes happens, would turn on the family history, mental symptoms, age at onset, and the course of the disease. Chronic chorea is incurable, and may take twenty years or more to run its course ; mental failure occurs early, and is progressive ; and a family history of chronic chorea can be obtained.

Chorea major, or *pandemic chorea,* is an epidemic hysterical manifestation occurring in the more emotional races of Europe under the influence of religious excitement. Choreiform movements are among the less conspicuous of its motor phenomena ; it is unknown in the more phlegmatic northern races.

In *hysteria* the motor phenomena are notoriously protean. Should a hysterical patient have had chorea herself, or should she have had the opportunity of observing it in others, she may reproduce its characteristic movements with great accuracy. The diagnosis may be very difficult for a time, particularly if the patient's previous history be not known, and hysteria not suspected. Her temperament will probably lead her to develop other signs or symptoms that suggest the true diagnosis ; such as tremors, paralyses, contractures, hemi-anæsthesia, anæsthesia of the stocking and glove distribution, exaggeration of the deep reflexes, or attacks of hysterics. Remission of the choreiform movements and of the local symptoms generally may occur when the hysterical patient thinks she is no longer under observation, or when her attention is diverted elsewhere. The hysterical patient simulating chorea or hemichorea is likely to overdo the part.

Choreiform movements may occur in connection with hemiplegia in two forms. *Pre-hemiplegic chorea* has been recorded in a few cases, twitchings or even choreiform movements beginning in the limbs of one side of the body shortly before the onset of an apoplectic stroke. *Post-hemiplegic chorea* is commoner, and more often seen in children than in adults. After a hemiplegia more or less muscular spasm and movements of one kind or another are habitually seen on the affected side of the body. In many patients these movements take the form of tremors, fine or coarse ; in others they are athetotic ; in others again they are ataxic, occurring only when voluntary movements are attempted ; and in yet others they are choreiform. Which of these forms of muscular contraction is likely to occur in any given case it is impossible to say ; they are all due to combinations of cerebral irritation, muscular spasm, and muscular paralysis, mixed together in varying proportions.

The choreiform movements occurring in the *spastic paraplegias of infants and children,* conditions that have been more vaguely described as *cortical scleroses* on the strength of their post-mortem appearances, are to be regarded as variants of the athetotic contractions already considered above. *Henoch's chorea electrica* is considered below : it is the muscles of the neck and shoulder that are chiefly involved in this rare disorder.

FIBRILLAR CONTRACTIONS.

Fibrillar contractions of the muscles, or fascicular muscular twitchings, are small spontaneous movements visible on the surfaces of muscles, rhythmical or irregular, involving not the whole muscle, but only single muscular bundles in it. They may be confined to a few of the bundles, or may occur irregularly in any of the bundles composing a muscle. They are almost always too feeble to produce visible movements at the joints ; they are increased in fatigue, and when the muscle is mechanically stimulated. Similar, but coarser, twitchings may be seen in normal muscles when they are over-fatigued, or on exposure to cold. The finest fibrillar contractions are said to occur only in cases of organic disease in the central nervous system. They are seen most freely in muscles that are degenerating or undergoing atrophy, or are shortly about to atrophy, as the result of disease in the lower motor neuron ; they cease to appear when the muscle is much wasted. They are most evident in the extremities and tongue, and no doubt are due to irritation of motor nerve-cells in the cord or bulb that are hyper-excitable because they are degenerating.

From a diagnostic point of view, fibrillar contractions are important because for practical purposes they do *not* occur in the *myopathies* or *primary muscular dystrophies* that are due to lesions in the muscles themselves and not in the spinal cord. In only a few recorded cases have these fibrillations been seen in cases of myopathy where lesion of the central nervous system could be excluded. Neurologists and myologists have devoted much attention to primitive myopathy, with the result that it has become burdened with a highly elaborate classification and nomenclature. Thus the condition generally has been described as primary progressive myopathy, progressive muscular dystrophy (Erb), idiopathic muscular atrophy and hypertrophy, primitive progressive myopathy, muscular dystrophy, myopathy.

Special forms of it have been raised to the dignity of ‘types,’ the chief of which are the—

Simple atrophic (Erb)
Pseudo-hypertrophic
Juvenile (Erb)
Facio-scapulo-humeral (Landouzy and Dejerine)
Distal (Gowers)
Myotonia atrophica
Mixed and transitional (Leyden and Moebius; Zimmerlin).

Distinctions between these various forms must be sought in special manuals. Their importance for present purposes consists in this—that fibrillary contractions may occur as a rare exception in most of them.

Contrariwise, fibrillar contractions are observed habitually in the course of the *progressive muscular atrophies of neuropathic origin*, variously known under such names as—chronic anterior poliomyelitis, amyotrophic lateral sclerosis (Charcot), progressive bulbar paralysis, progressive muscular atrophy, toxic degeneration of the lower motor neuron, Werdnig-Hoffmann progressive muscular atrophy of infants, according to their special characters. In all of these, the lower motor neurons are primarily at fault, exhibiting slow or rapid degeneration ; in many cases the upper motor neurons are also affected, either simultaneously, or before or after the lower. As a rule, no cause for the degeneration can be discovered ; but many—perhaps a half—of the patients have previously had acute poliomyelitis. Occurring in *infants* or *children*, this neuropathic muscular atrophy is generally of the Werdnig-Hoffmann type, affecting the legs first, and spreading upwards to the body and arms ; the hands and feet are affected late, and the deep reflexes vanish. The condition may at first sight resemble rickets, but in rickets there is no real muscular atrophy, the deep reflexes are retained, and fibrillar contractions do not occur. It may be indistinguishable from one of the primary myopathies considered above ; but the occurrence of fibrillar contractions would make the diagnosis of neuropathic muscular atrophy the more probable.

In *adults* the disease may conform to one of several types, according to the distribution of the atrophy. In some instances the lower motor neurons of the hand, arm, and neck are attacked, when the Claw-hand (p. 109) may result ; in others, the lower extremities may first show the degeneration. Charcot's amyotrophic lateral sclerosis is characterized by spasticity of the legs combined with atrophy of the muscles of the hands and arms. In making the diagnosis of neuropathic muscular atrophy it must be remembered that the onset is gradual, that fibrillar contractions are present, that the atrophy proceeds *pari passu* with the loss of power, and that sensation and the sphincters are not involved. The electrical changes in the muscles are of assistance, too, the partial Reaction of Degeneration (p. 582) being exhibited ; the nerves react normally to faradism, and to galvanism so long as there are muscle fibres left to respond to the stimulation, while the muscles react sluggishly, and A.C.C. is often greater than K.C.C.

Bulbar paralysis is due to lesions of the medulla oblongata, and the nerves mainly affected are the motor part of the fifth, the seventh (facial), the eleventh (spinal accessory), and twelfth (hypoglossal). In other cases ophthalmoplegia is observed as well. It is only in the chronic cases of bulbar paralysis that fibrillar contractions are seen, and they are particularly well shown in the tongue, which has been described as looking ‘ like a bag half full of worms.’ The main symptoms will be difficulty in articulation, phonation, mastication, and, most of all, in swallowing.

SPASMODIC CONTRACTIONS.

In general parlance, the epithet 'spasmodic' implies suddenness and short duration. These characteristics are not implied by the word as it is used clinically. Hence it is necessary to distinguish between spasmodic contractions or muscular spasms of three kinds, according as they are :—(1) *Short and single*—the muscular twitch ; (2) *Short and repeated*—clonus or clonic spasms ; (3) *Tetanic*—commonly and improperly known as *tonic* spasms ; these are long-sustained.

Single Spasmodic Contractions of a muscle or group of muscles, over in a fraction of a second, may occur in *normal persons* who are suffering from great fatigue, overwork, or nervous exhaustion. For no apparent reason, and frequently just as the person is going off to sleep, a sudden violent twitch in one or more of the limbs occurs, and wakes him up. In other cases these sudden starts may occur when the patient is resting by day. In *abnormally nervous or excitable patients* such sudden spasms are seen more frequently, and often result from some sudden and unexpected sensory impression—a sound, sight, or touch. The diagnosis of such spasms in nervous or jumpy patients should not be difficult, the affection being very chronic, and no doubt familiar to the patient and the patient's entourage. Coming on suddenly, this jumpiness may be a minor sign of various nervous disorders, such as hysteria, acute chorea, delirium tremens, general paralysis, or Graves's disease.

Single twitches of muscles or of groups of muscles form the outstanding feature of the simpler forms of a series of affections known as *habit spasms* or *spasmodic tics*. A habit spasm consists in the involuntary repetition of some ordinary co-ordinated purposive act. In many instances the tic was at first a natural reflex act, designed to allay some transient irritation. Thus a blinking tic may have been initiated by the pain caused by a foreign body in the eye, or conjunctivitis ; a sniffing tic by some temporary itching about the nares, or it may be associated with the presence of adenoid growths in the nasopharynx ; a shoulder-shrugging tic by some irritation of the neck due to a tight or rough collar. By voluntary repetition such an act ultimately becomes automatic, when it is spoken of as a habit spasm or tic. These motor tics exist in great variety, oftenest affecting the face, less often the jaws, neck, or limbs ; they are so common as to escape comment in their minor manifestations—mannerisms and stereotyped acts—being set down merely to 'individuality.' Most tics can be controlled by mental effort with some distress, are increased by emotion, cease during sleep, and are curable only with great difficulty when well established. In all cases the patient is supposed to exhibit a certain psychical weakness.

More violent and shock-like muscular spasms are seen in the rare condition known as *myoclonus* or *paramyoclonus multiplex*. Myoclonic movements are particularly sudden and violent, occurring bilaterally, or first on one side of the body and then on the other ; they are painless, but may give rise to much inconvenience by their violence. They are increased by emotion and cease during sleep. They may be single, but more often are clonic, repeated perhaps fifty or a hundred times in a minute. In paramyoclonus multiplex there are no mental, sensory, or sphincter changes, and this rare disease is described as both familial and hereditary. In *hysteria*, myoclonus is seen exceptionally, accompanied by other hysterical manifestations. In certain rare forms of *epilepsy*, the so-called myoclonic epilepsy, these paroxysmal asynchronous bilateral lightning-like movements have been recorded ; the diagnosis will be easy here, as the patient exhibits the phenomena of major epilepsy—loss of consciousness, relaxation of the sphincters, etc.—in addition to the sudden and forcible myoclonic movements. In certain cases of *minor epilepsy*. or *petit mal*, the affection may take the form of spasmodic twitches of the muscles of a limb, or of the face, associated with a brief absent-mindedness or a few seconds of loss of consciousness without loss of automatic control over the body generally.

Clonic Spasmodic Contractions, clonic spasms, or clonus, are in reality interrupted tetanic contractions, consisting in the rhythmical and more or less rapid repetitions of the single brief muscular spasm or twitch. A typical clonus of muscles in the arms or legs may often be produced in health by the adoption and maintenance of some strained position. Thus ankle-clonus is soon produced if a normal person sits in a chair and strains the heels up while the toes are held pressing on the floor. Such clonus is physiological, being due to heightening of the muscle tone or normal state of tonic muscular contraction by the application of mechanical tension to the calf muscles.

Pathologically, clonic spasms are seen typically in the second or clonic stage of *major epilepsy*, where they succeed the initial tetanic (or tonic) stage. Here they are universal and bilateral as a rule, although one side of the body may be involved more than the other, or the arms more than the legs. Consciousness is lost, and the sphincters are often relaxed. Mild and limited clonic spasms of a few muscular groups, without loss of consciousness and lasting for only a few seconds, may be seen in patients with major epilepsy, and are often described by them as 'warnings.' Such attacks are identical with those of minor epilepsy. In certain epileptic patients they seem to be to some extent under control, so that their threatened onset can be prevented if the patient can sit or lie down, for example, or can press on or constrict the limb in which the spasms are about to appear. The diagnosis of *hystero-epilepsy* is sometimes made in these patients ; but the term is not a good one, and is often misleading. Very similar convulsive seizures may be met with in patients with chronic nephritis (*uræmic convulsions*) and in pregnant women (*eclampsia*). The clonic stage of epilepsy may be imitated unconsciously by patients with hysteria, or frankly mimicked by the malingerer. In *hysteria* the onset of the fit is gradual, not sudden ; consciousness is impaired, not lost ; the pupil reacts to light, and is not immobile as in epilepsy ; screaming and purposive movements occur throughout, and the fit is often protracted ; the sphincters are not relaxed, and the tongue is not bitten. The *malingerer* is red and heated by the effort of producing the clonic spasms, his consciousness is fully preserved, and he reacts to painful stimuli that leave the epileptic unmoved. Both the hysterical patient and the malingerer show quivering of the eyelids, and are likely to resist attempts to open the eyes.

In *Jacksonian epilepsy*, clonic convulsions occur without loss of consciousness ; they are usually unilateral, starting in some given muscle and spreading thence until both limbs or half the body are convulsed. Transient paresis from exhaustion may be noted afterwards in the affected muscles. In severe or long-established cases the whole body may be convulsed, or a tetanic stage may occur after the clonus ; in these instances consciousness may be lost. Jacksonian or focal epilepsy may result from any form of local irritation of the motor cortex—trauma, hæmorrhage, new growth, the effects of syphilis, chronic inflammation. It leads in the long run to paresis and atrophy of the affected muscles.

As the names imply, the very rare conditions known as *myoclonus* and *paramyoclonus multiplex* exhibit typical clonic contractions. The clonus occurs in single muscles or muscle-groups, such as the biceps and supinator longus, the quadriceps femoris and semitendinosus ; rarely in the face : from 50 to 150 contractions a minute may occur. Henoch's *chorea electrica* is the same as myoclonus. It is said that animals from which the parathyroid glands have been removed may exhibit identical spasms. For the diagnosis of myoclonus, see above. Clonic spasms of the neck-muscles, particularly the sternomastoids, are common in *torticollis* or wry-neck.

Tetanic Contractions, tetanic or the so-called tonic spasms. Physiologists and clinicians both make use of the two terms 'tetanic' and 'tonic,' but unfortunately employ them in different senses. Physiological 'tetanus' is the apparently steady state of muscular contraction exhibited by the voluntary muscle at work, maintained by the fusion of separate muscular twitches or spasms due to a rapid succession of nervous stimuli. It may be seen in a single muscle or in many together. Clinically, however, 'tetanus' or 'tetanic contractions' have come to be associated with pain, besides being of some duration, and the terms are used only when a large number of muscles are involved simultaneously ; tetanus of a single muscle is referred to clinically as a *cramp* (p. 139 and 150). To the physiologist, the normal resting muscle is already in a state of 'tonic contraction,' and exhibits 'tonus.' This muscle-tone is maintained partly by local or peripheral stimulation (mechanical tension, the venosity of the blood, drugs such as digitalis or veratria), and partly by nervous impulses that reach the muscles more or less continuously from the motor neurons of the central nervous system. This central element of muscular tonus is really of reflex origin and due to posture, the maintenance of the erect attitude ; the motor impulses descending in answer to ascending impulses received by the central nervous system from the muscles and joints concerned. But the clinician applies the terms 'tonus' and 'tonic contractions' to the severe and pathological muscular contractions seen, for example, in the first stage of major epilepsy, which are physiologically and scientifically speaking tetanic, not tonic. This clinical misuse of the word 'tonic' is well-established and time-honoured, but only serves to promote confusion. The terms 'tonic spasm' and 'tonic contraction' should be reserved for states of muscle-tone that are raised only within physiological limits, and are not pathological. The contractions or spasms that the clinician calls 'tonic' are almost always pathological, and in the interests of uniformity should be described as 'tetanic,' not 'tonic.' Exaggerated states of physiological tone and the milder degrees of pathologically heightened muscular tonicity are described clinically as spastic states or spasticity, falling short of tetanus in degree, and differing from both tetanus and cramp by being painless. They are detailed under the heading CONTRACTURES (p. 139).

Typical tetanic (or tonic) spasms are seen in *tetanus*. Here the patient has become infected by *Bacillus tetani* (*Plate XXVIII, Fig. T*, p. 614), through some known or unknown wound. He first notices stiffness of the neck and jaws; soon, increasing tetanic spasm of the muscles of mastication brings on trismus or lockjaw. Spasm of the facial muscles next brings on the painful grin known as the *risus sardonicus*, and presently paroxysmal tetanic spasms of great violence occur in practically all the voluntary muscles, although in mild cases in children the spasm may proceed no further than the muscles of the face. If the spasms are strongest in the extensors of the back, the body is arched backwards till, perhaps, the heels touch the head (opisthotonus). If the flexors contract most powerfully, the body is bent forwards (emprosthotonus); in some cases the body remains straight and stiff (orthotonus) when the flexors and extensors are balanced. These acutely painful paroxysms last for perhaps a few seconds, and recur at varying intervals on any kind of stimulation; they may cause death by asphyxia or heart-failure. In the intervals between them, a milder but still painful tetanic (the so-called tonic) contraction of the muscles is maintained; or, in milder cases, nothing more than an exaggerated physiological muscle-tone. In mild or chronic cases of tetanus, the signs and symptoms will be far less severe than those described above; but trismus and painful muscular contractions will still occur. In some chronic cases, the chief sign may be a recurring but transient risus sardonicus, perhaps with some stiffness of the neck; not a few of these patients have been treated for *habit spasm* or *hysterical grimacing* for a time, until the suspicion of tetanus arose, or spread of the tetanic spasms to the trunk-muscles made the diagnosis more obvious. The diagnosis of tetanus may have to be made in other instances from *impacted wisdom tooth*; or from *muscular rheumatism*, which may cause stiff-neck but is hardly likely to set up trismus; or from *spinal meningitis*, in which there is fever, while the tetanic spasms occur on exertion, and do not primarily affect the muscles of the jaws, and great pain is felt on moving the head and neck.

In *strychnine poisoning* trismus is absent or occurs very late, the extremities are first and most markedly affected, the muscles are quite relaxed between the paroxysms, and the symptoms develop rapidly—within an hour or two of the administration of the drug. In *tetany* (p. 2) the distribution and duration of the tetanic contractions should suffice to prevent any confusion with tetanus. In *hydrophobia* there should be a history of a bite by some animal, most often a dog; mental symptoms are prominent, and the spasms affect the muscles of respiration and deglutition most, while trismus is absent. In *hysteria* a patient may exhibit trismus, tetanic spasms, and opisthotonus; but no true picture of tetanus will be presented, and other evidences of hysteria will be found on examining the patient, or will develop if the case be kept under observation.

A. J. Jex-Blake.

CONTRACTURES—are lasting bodily deformities resulting from a great variety of causes. For clinical purposes they may be divided roughly into two classes, according as they are (1) *Active*, or (2) *Passive*. The division is not sharp, as active contractures when long established tend to become passive.

1. **Active Contractures**: resulting:—

(*a*). *From lesions of the upper motor neuron:*

Cortical lesions
Hemiplegia
Friedreich's ataxia
Myelitis
Transverse lesions of the cord
Subacute combined degeneration
Lateral sclerosis
Spastic ataxia
Spastic paraplegia
Hæmatomyelia

(*b*). *From lesions of the lower motor neuron:*

Acute poliomyelitis
Chronic poliomyelitis
Progressive muscular atrophy
Neuritis
Injury of nerves

(c). *From disuse :*

Hysteria	Torticollis

2. **Passive Contractures:** seen in :—

Late stages of the active contractures
Local organic diseases of the bones, joints, muscles, fasciæ, skin.

Active or Spastic Contractures.—In these, certain groups of muscles are thrown into a state of permanent contraction, or else the balance of power between antagonistic sets of muscles is upset. In either case bodily deformity (flexion, extension, curvature) results ; but the deformity can be redressed temporarily either by steadily maintained mechanical traction, or by the forcible electrical stimulation of the weaker set of the antagonistic muscles involved. In passive contractures, on the other hand, no amount of electrical or other stimulation avails to correct the deformity, nor can the application of force without rupture of the tissues.

Active contractures must be distinguished from certain other forms of muscular contractions, particularly cramps and tetanic (or so-called tonic) contractions or spasms of the voluntary muscles. *Cramps* may resemble contractures by their relatively long duration—thus those of tetany have been known to persist for days and even weeks ; but pain is a constant feature of cramp, whereas it has no connection at all with contractures *per se*. *Tetanic contractions* of muscles (see CONTRACTIONS, p. 131)—commonly called *tonic* by the misuse of a word that already has a definite and different physiological meaning (p. 137)—resemble cramps by being painful, and differ from them only by being more generalized. The normal resting muscle is, physiologically speaking, in a constant state of *tonic contraction*, and exhibits a certain reflex tone or tonus (muscle-tone) due to the combined action of two factors, one local and one nervous. Any muscular spasm, rigidity, or spasticity set up by increase of this normal tone within physiological limits, may properly be referred to as a condition of tonic contraction. But when a spasticity is pathological, as are all the 'tonic contractions' of the clinician, it should no longer be referred to as a state of tonic contraction, especially as it corresponds satisfactorily with the physiological 'tetanic contraction' or 'tetanus.' A typical pathological spasticity or active contracture is seen in Sherrington's 'decerebrate rigidity,' the extensor spasm observed in the limbs of the cat or rabbit after removal of the cerebral hemispheres and basal ganglia. This rigidity lasts for several days, and is due to the removal of the inhibitory impulses normally reaching the cord from the cortex and thalamus. A similar rigidity, though, of course, with a different distribution, is seen in such disorders as hemiplegia, cortical losses, lateral sclerosis, Friedreich's ataxia, subacute combined degeneration, and transverse lesions of the cord.

The active contractures following *hemiplegia* or *cortical lesions* in the motor area are confined to the affected side of the body, and should not be difficult to diagnose. There are three varieties of rigidity after hemiplegia, but only the last of these is usually described as a contracture: (1) Initial rigidity, present at the outset and lasting only for a few hours ; (2) Early rigidity, beginning within a few days of the stroke and lasting for a week or a few weeks, possibly due to the irritation of blood-clot at the site of the cerebral lesion ; (3) Late rigidity or contracture, first appearing several weeks or months after the stroke, and due to the fact that while all the muscles of the affected limbs are spastic, certain groups of them are stronger than their antagonists. Thus the thumb is flexed and pressed into the palm, the fingers clenched, the wrist and elbow flexed, the forearm pronated, and the arm adducted. The thigh is adducted, the knee extended, and the heel drawn up, the foot inverted, and a characteristic spastic gait results. The deep reflexes are increased on the hemiplegic side, where, too, ankle-clonus and Babinski's extensor reflex can be obtained. The lapse of years converts these active contractures into passive in consequence of the structural changes that take place in the muscles, fasciæ, and joints.

Contractures are highly characteristic of *congenital* and *acquired cerebral diplegias* or *hemiplegias due to cortical lesions*, cortical sclerosis, or porencephalus (see CONTRACTIONS, ATHETOTIC, p. 131). The patients show bilateral spastic paralysis; one side is sometimes affected more severely than the other. If the legs only are affected the condition is known as Little's disease, and the gait is 'cross-legged' or 'scissor,' the feet being pointed and inverted, and the thighs adducted. Kyphosis is often seen, and the arms, if involved, are held in the position of a hemiplegic arm (see above). In the acquired cases the spastic paresis is oftener unilateral than bilateral; the nutrition of the affected limb suffers conspicuously, and its growth is retarded and incomplete.

In *Friedreich's disease*, a familial disorder beginning usually between the ages of seven and seventeen, and seen oftenest in males, characterized by ataxia, intention-tremor, nystagmus, and hesitating or syllabic speech, active contracture sets up scoliosis or scoliokyphosis, pes varus or equinovarus, and 'main bote'—an analogous deformity of the hand with hyperextension of the terminal phalanges. These contractures are partly due to muscular atrophy, partly (in the case of the foot) to over use of certain muscles in attempts at equilibration; the heel is drawn up, the dorsum of the foot arched, the sole hollowed out, the toes flexed at the interphalangeal joints and hyperextended at the metatarsophalangeal; prominent hypertrophy of the extensor longus hallucis has been found.

In *subacute combined degeneration*, in which may be included *lateral sclerosis* if the degeneration mainly affects the upper motor neurons, there will be contractures. The earliest symptoms are connected with sensation; but the patient, usually an anæmic adult in the second half of life, presently develops spasticity in his legs. The limbs tend to draw up as he lies in bed, from flexor spasm; the gait becomes spastic, and walking is soon impossible—the condition becoming one of *spastic paraplegia*. The deep reflexes are increased, and Babinski's sign is present; segmental areas of anæsthesia can be made out, and control over the sphincters is weakened. After some months, this spastic stage gives place to flaccidity, control over the sphincters is lost, and the patient rapidly runs down hill. In cases of *transverse myelitis* or *transverse lesions of the cord*, and in certain cases of *hæmatomyelia* of insidious onset, in which the hæmorrhage perhaps takes place into an already dilated central canal, spasticity with increased deep reflexes, loss of sensation, and loss of control over the sphincters is the rule. The diagnosis is facilitated by the fact that no symptoms occur in parts of the body innervated from above the cord lesion; at the level of the lesion there is evidence of nerve-irritation (girdle pain, hyperæsthesia). In these cases the flexors of the leg overpower the extensors; the limbs draw themselves up again, sooner or later, as often as they are extended for the patient.

So far, the active contractures considered have all been due to lesions of the upper motor neuron. A second class contains those resulting from lesions of the lower motor neuron and the subsequent muscular atrophy. These contractures arise either from the unbalanced action of the muscles that normally antagonize those that have atrophied, or from late shrinkage of the paralyzed muscles themselves; and a spinal curvature may come on from the adoption of some posture that facilitates locomotion or the occupations of life when the spinal muscles are intact. Acute and chronic poliomyelitis, neuritis, and lesions of the nerves have to be discussed in this connection. *Acute poliomyelitis*, or infantile paralysis, begins suddenly with malaise, pains, and an acute febrile attack; the flaccid paralysis appears early, and contractures begin to show themselves within a few months, as a rule. The limbs are most involved, isolated muscles or groups of muscles being paralyzed; and it should be noted that the paralysis is distributed in accordance with the nuclear grouping of the muscles in the anterior cornual regions of the cord. Sensation is affected only in the rarest instances. If many muscles in a limb are paralyzed, its growth is much impaired. Contractures are common in *chronic poliomyelitis* and the various forms of *progressive muscular atrophy of neuropathic origin* (see CONTRACTIONS, FIBRILLAR, p. 134), the hands and feet being mainly involved, with the production of various forms of club-foot and claw-hand. All the muscles are involved together, and there is no selection of certain groups for paralysis as is the case in acute poliomyelitis. In addition, fibrillar contractions can be seen in the degenerating muscles, provided that they are not covered too thickly with subcutaneous tissue. The onset is insidious, and the disease occurs most often in middle age; the commonest type is that in which the hands are first and most involved, but in other cases the legs, and in others the upper arm and shoulder, first give evidence of the disease. Contractures are seen occasionally in *alcoholic neuritis* of the motor type, and more frequently in *arsenical neuritis*, talipes equinovarus or flexor contracture of the wrist, with excessive muscular hyperæsthesia, being noted; such deformities are rare in other forms of neuritis, such as those due to lead, diabetes, influenza, diphtheria, etc. Secondary contracture of the muscles on the affected side in Bell's facial paralysis may occur, and gives rise to the impression that the sound side of the face is paralyzed while the face is at rest, for the face as a whole is pulled over to the affected side (*Fig.* 198, p. 492): on voluntary movement, however, the healthy side will be found to move normally, while the paralyzed side remains comparatively still. Contractures usually follow severe *injury of nerves,* unless satisfactory healing of the wound and regeneration of the nerve-trunks take place.

Active contractures from disuse may occur in otherwise healthy subjects who for any reason may have been kept too long in one position. Patients who have lain on their backs in bed for long periods may have a temporary talipes equinus when they get up—an active contracture due to the weight of the bedclothes resting on the toes and keeping the feet extended. Fractured or injured limbs that have been splinted and kept too long in one position, often exhibit active contractures when the splints are removed (e.g., Volkmann's ischæmic contracture of the forearm, *Fig.* 58). In some cases the contracture is due to fixation of the muscles, tendons, or muscle-sheaths by inflammatory products that have become organized, in others to adhesions or bony deposits that have formed themselves in or about the joints, while in others mere disuse, without inflammatory changes, may underlie these contractures: all of these would be avoided by the timely use of massage and movement.

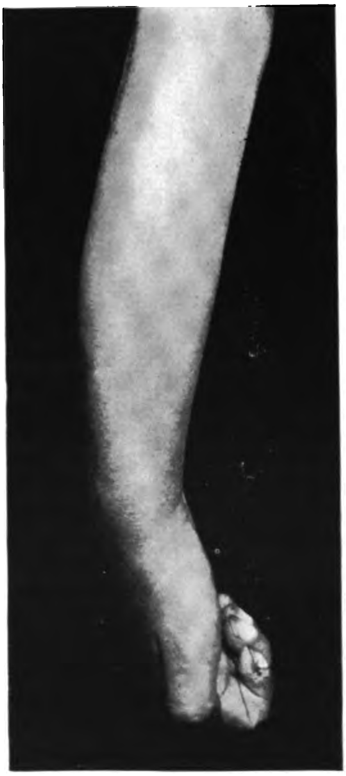


Fig. 58.—Volkmann's ischæmic paralysis following the use of an anterior splint for fracture of the bones of the forearm. Note the clenched fingers and the trophic sore on the forefinger. (Reproduced by permission from a paper by Mr. R. P. Rowlands in the *Guy's Hospital Gazette.*)

Paralyses occur in perhaps 25 per cent of all patients with *hysteria,* in two main types: the rarer flaccid, the commoner spastic, and often marked enough to produce active contracture. In hysterical contracture the affected muscles are not wasted except in severe cases of long duration; the deep reflexes are increased; ankle-clonus may be present; but Babinski's sign is probably never observed. The limbs are most affected (hemi-, mono-, or paraplegia), less often the muscles of the face, eyelids, lips, or tongue. Certain attitudes are highly characteristic of hysterical spastic paralyses; the elbows, wrists, and fingers are kept flexed, the arms are adducted; the hip and knee are extended, and the foot is held in a position of talipes equinovarus; ptosis may be seen, from spasm of the orbicularis; torticollis from contracture of the sternomastoid. In the less severe cases the stiffness and paresis are neither complete nor marked enough for the condition to be referred to as a contracture. In all instances the deformity produced is the result of active muscular spasm, and in severe cases it cannot be overcome by exercise of the patient's will, galvanization, or by the application of mechanical force. The contracture often persists during sleep, and is abolished only by deep anæsthesia—a character distinguishing hysterical contractures from those due to organic disease. Hysterical contractures often last for months, or even years: and in cases of long standing muscular atrophy and structural changes about the joints may establish permanent passive contracture from disuse. Highly characteristic of hysterical contracture is the patient's use of antagonistic muscles to prevent passive or active correction of the deformity exhibited. If, for example, the arm is semiflexed by contracture of the biceps, the triceps can be felt to contract and resist the movement when the attempt is made to flex the arm further. A similar contraction of the triceps can be felt or seen if the patient is asked to bend the joint herself; with the result that the joint remains unmoved, although all signs of great effort to bend the arm may be displayed. Pain and tenderness in the contracted muscles are usual; and other hysterical stigmata such as hemianæsthesia, paræsthesia, clavus or globus hystericus, and the hysterical temperament generally, will not be wanting. Special forms of hysterical contracture may give rise to great trouble in diagnosis by imitating definite conditions or diseases. Thus a painful 'hysterical hip' or 'hysterical knee' may pass on from surgeon to surgeon, until one is found to operate upon the normal joint for tuberculous arthritis; hysterical spasm of some of the abdominal muscles may lead to the diagnosis of pregnancy or new growth—pseudo-cyesis or phantom tumour; hysterical contracture of muscles in the neck or shoulder may be diagnosed as new growth, the palpable tumour vanishing only when the patient has been anæsthetized and is on the operating-table awaiting incision.

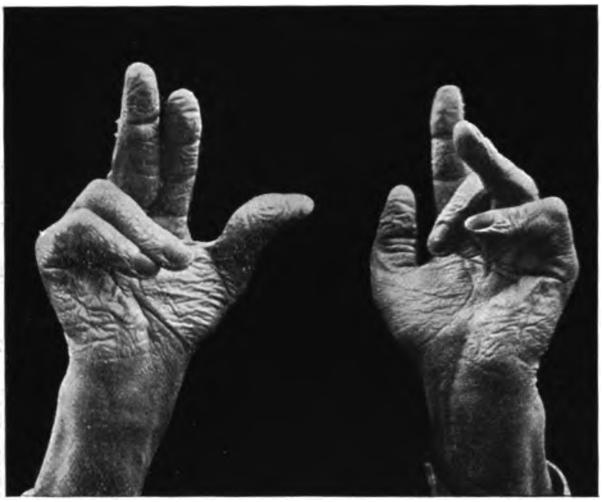


Fig. 59.—Dupuytren's Contracture.

Torticollis, or wry-neck, in adults, may be regarded as a functional disease, and is a form of tic, characterized in its later stages by contracture of the affected muscles of the neck. The muscles chiefly affected are those supplied by the spinal accessory nerve. Its clonic variety is easy to diagnose; but where the spasms are tetanic (or tonic) rather than clonic, the diagnosis must be made from such conditions as cervical caries, rheumatic myositis, or deep inflammation in the glands of the neck. *Congenital torticollis* dates from birth, usually affects the right sternomastoid muscle, and is often associated with facial asymmetry—when it is perhaps due to congenital defect of the centres in the bulb. The face is smaller on the side of the affected sternomastoid. Congenital torticollis is distinguished from the form of wry-neck produced in infants by rupture of the sternomastoid muscle at birth during delivery, by the fact that in the latter a callus is to be felt at the site of the rupture.

Passive Contractures are those due to affections of the bones, joints, or soft tissues, that mechanically obstruct correction of the deformities they produce. They also result from *long continuance of the active contractures* considered above, by a gradual process of transition. The contracted limbs can only be straightened by surgical measures, or by manipulations severe enough to rupture the obstructions.

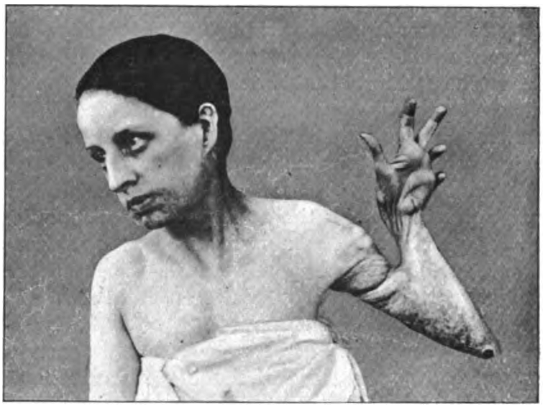


Fig. 60.—Cicatricial contracture after a burn.
(From Professor Rutherford Morison's *Introduction to Surgery.*

Passive contractures may result from the most varied *local organic diseases* of the affected parts. *Dupuytren's Contracture* of the palmar fascia, leading to deformity of the little and ring fingers (*Fig.* 59) is so characteristic that it can seldom be mistaken. It is prone to occur in gouty subjects and in those who use the palms of their hands most, as in the case of coachmen and those who use spades, etc. In diseases of the *joints*, such as rheumatism, rheumatoid arthritis, spondylitis deformans, tuberculosis, gonorrhœa, etc., the patient may lie in bed or go about for weeks or months in some bent or contorted position that involves the minimum of discomfort; ankylosis of the affected joints often results from the growth of adhesions, ecchondroses, or exostoses in and about the edges of the joints, that permanently limit their range of movement. Corresponding shortening will take place in the muscles that are relaxed, and a passive contracture results. The growth of a tumour in or about a joint may produce identical results. Traumatic or inflammatory lesions about the *muscles* or their *tendons* may establish inflammatory products locally that permanently limit the movements of these structures. Large superficial *scars* due to extensive burns or losses of skin and the superficial tissues, being composed mainly of fibrous tissue, may contract, and so bring about marked contractures (see *Fig.* 60).

Spondylitis deformans, a chronic malady of the spinal column (p. 648), often results in contractures and partial ossification of the ligaments and muscles of the back; and extreme deformity may arise from *myositis ossificans*, a rare but easily diagnosed affection in which the muscles all over the body gradually become rigid from calcification; the patient has generally been normal up to adult life, and then becomes the subject of acute attacks of pain in various muscles, accompanied by local myositic swelling and some pyrexia; after the local inflammation subsides, calcium salts are deposited in the site that has been inflamed and the affected muscle becomes stiff and hard. Weeks or months may elapse between successive attacks of this kind, but the number of calcified muscles slowly mounts up, until in extreme instances the patient is rigid almost from head to foot—the 'ossified man.'

The diagnosis of the cause of a passive contracture will obviously depend upon the results of the physical examination of the affected part, and upon the success with which a true history of the onset and course of the case can be elicited. *A. J. Jex-Blake.*

CONVULSIONS, or CONVULSIVE SEIZURES, are paroxysms of involuntary muscular contractions. They may be divided into two classes, according as they are *local* or *general*; *local convulsions* have been considered under the heading CONTRACTIONS, SPASMODIC (p. 136), and the following account deals mainly with *general convulsions*. The general convulsions without loss of consciousness that constitute RIGORS are described under that heading (p. 594); with this exception, general convulsions are almost always accompanied by loss of consciousness, excepting in some few cases of partial epilepsy and of hysteria.

In most cases of convulsions, both sides of the body—face, neck, arms, trunk, and legs—are convulsed equally. But it sometimes happens that though their cause is apparently general, the movements are unilateral, or much more marked on one side of the body than the other; for present purposes such convulsions may still be termed general. Usually convulsions are clonic, less often tetanic or tonic.

'Fits' may be defined roughly as any sudden paroxysms or seizures occurring in the course of any disease. In common usage, however, a 'fit' is a convulsive fit, or fit of convulsions, and if unqualified, the term usually means an epileptic fit, but not always.

Certain clinical features are common to almost all convulsive seizures in which consciousness is lost. If the onset is sudden, as it usually is, the patient is apt to fall down and injure himself unless already recumbent. If the muscles of the mouth and jaws are involved and saliva is secreted freely, the mouth foams; if the tongue or cheeks are bitten, the foam becomes stained with blood. Clenching of the jaws will make the breathing laboured, stertorous, and ineffectual. If the muscles of respiration are greatly affected, cyanosis, with congestion of the face, neck, and exposed parts, will be observed. The convulsive movements are typically clonic, limited in range, purposeless, and accompanied by more or less rigidity. If the rigidity is marked, the amplitude of the movements will be reduced correspondingly, so that the condition may even become one of stiffness and tetanic (or so-called tonic) spasm. It is characteristic of epilepsy that the fit should consist of a brief tetanic stage followed by a longer stage of clonus; but convulsive attacks of every sort may occur in epilepsy, and either the tetanic or the clonic stage may be absent or so brief as to pass unnoticed. Control over the organic reflexes of micturition and defæcation is often lost, the bladder and rectum being emptied involuntarily. As a rule the reflexes cannot be obtained while the convulsions last, and are lost or diminished for some hours after they are over, or are unequal on the two sides of the body. When the fit is over and the patients have recovered consciousness, they often complain of headache and lassitude, showing diminished sensibility to all impressions, mental hebetude, and great sleepiness. Less often, the patient becomes excited or terrified after a fit, or even maniacal, and he may also exhibit automatism for hours or even days ; in none of these conditions will he be responsible for his actions. The duration of general convulsions is commonly to be measured in seconds or minutes ; but in severe cases they may go on for hours if untreated, and in the status epilepticus may last for days with only brief intermissions. Prolonged convulsions due to any cause may raise the temperature several degrees ; when they are unilateral, the temperature is raised more on the affected side than on the other. Albuminuria after a fit is very common, and may last for a day or two ; it is by no means necessarily evidence that the fit was uræmic.

The morbid conditions in which *local* or *partial convulsions*, and in rarer instances *general convulsions* also, occur without loss of consciousness, have been considered under CONTRACTIONS, SPASMODIC (p. 136), but for the sake of clearness may be recapitulated :—

Fatigue
Nervous exhaustion
Habit spasm
Spasmodic tic
Myoclonus
Hysteria
Jacksonian epilepsy
Chorea electrica (Henoch)
Tetany
Tetanus
Hydrophobia
Strychnine poisoning
Malingering.

The convulsions commonly accompanied by loss of consciousness will be considered here under the following heads :—

1. **General Convulsions of Infants and Children,** seen in :—

Hereditary syphilis
Congenital heart disease
Cerebral paralysis
Onset of acute fevers
Meningitis
Drug poisoning
Enlarged thymus
Idiocy
Rickets
Epilepsy, minor and major.

2. **General Convulsions of Adolescents and Adults,** seen in :—

Epilepsy, minor and major.
Jacksonian epilepsy
Epileptiform convulsions—
  Uræmia
  Pregnancy
  Severe heart disease
  Asphyxia
  Stokes-Adams' disease
  Saturnine encephalopathy
Cerebral lesions :—
  Apoplexy
  Meningitis
  Intracranial growth
General paralysis
Chronic alcoholism
Cerebral syphilis
Hysteria
Malingering.

3. **Unilateral Convulsions,** seen in :—

Apoplexy
Intracranial growth
Meningitis
Epilepsy, major and minor
Jacksonian epilepsy
Disseminated sclerosis.

General Convulsions of Infants and Children.—Among the commonest of all convulsive seizures are those occurring in children of tender age, known as *infantile convulsions*. The sexes are affected equally ; about a third of the cases take place during the first year of life, two-thirds during the first two years ; and they are rare, apart from epilepsy, after the age of five or six. They are of more serious import in infants under six months than in older children, and also in anæmic and weakly infants. In *hereditary syphilis* convulsions often prove fatal during the first week of life. For the rest, in about half the patients *rickets* is the predisposing cause ; in many of the others some local irritation, such as inflammation of the gums in *dentition*, *diseases of the nose* or *ears*, the presence of *irritating food* or *worms* in the intestine, renal or vesical *calculus*, or *phimosis*, can be found ; while convulsions at the onset of *acute infectious diseases*, such as scarlet fever, pneumonia, measles, whooping-cough, or during their course, and in *nephritis*, are not infrequent. Overdosing with *drugs* —strychnine, atropine, santonin, morphia—or with *alcohol*, may bring on convulsions. Fright and over-strung emotions are included among the causes of infantile convulsions ; how far inheritance, the neurotic or neuropathic taint, is responsible for them is uncertain. They occur in children with enlargement of the thymus gland, the so-called *status lymphaticus*, and in these not infrequently a fit has a fatal issue. Finally, it must be remembered that in any child there may be early evidence of *epilepsy*, or of organic disease of the brain. Their diagnosis demands a very careful examination of the child, and also of its diet and the hygiene of its daily life. They may be due to *congenital heart disease*, when there will be enlargement of the heart, a cardiac murmur or murmurs, and some degree of cyanosis. In children with organic disease of the brain (*porencephalus*, *congenital or acquired cerebral paralysis*, *spastic paraplegia*, *etc.*) there will be paralysis, spasm, and muscular atrophy, and probably mental defect. If the convulsions are due to the *onset of some acute infectious disorder*, they will come on suddenly in a child previously well, and will be accompanied by high fever and followed by the characteristic rash. Similar convulsions and fever may occur in *meningitis*, usually towards the end of the disease. They are not rare in *whooping-cough*, particularly in rachitic infants, being precipitated by the asphyxia resulting from the whooping, and not rarely causing death. The diagnosis of fits due to *drugs* or *alcohol*, taken either by the child, or by the mother if the child is being suckled, will depend upon obtaining an adequate history of the case. In what way *enlargement of the thymus* brings about convulsions is not known; the condition is fortunately rare, and is hardly ever diagnosed during life. The fits occurring in *hydrocephalus* and the various degrees of *mental defect* need only be mentioned.

It is to *rickets* that one must look for the explanation of most convulsions occurring between the ages of three months and four or five years. The nervous system is unstable in all young children, the power of cerebral inhibition not being acquired for several years. In rickets this instability is much increased, and finds expression in irritability, fits of screaming, restlessness, inability to sleep well at night, and in the more serious troubles of tetany, laryngismus stridulus, and convulsions. Any child with fits should be scrutinized for evidence of rickets—exaggerated curvatures in the long bones, the rickety rosary, a Harrison's sulcus on the sides of the chest, the large and bulging rickety head, thinness of the hair on the back of the head (due to head-rolling), a tumid and flaccid abdomen, lateness in the closure of the anterior fontanelle, and general muscular debility. Enquiry should be made for other symptoms common in rickets that will come under the observation of the mother or nurse—tenderness of the bones and skull on handling and washing, head-rolling due to tenderness of the skull, much sweating about the head in sleep, broken slumber, proneness to gastro-intestinal upsets, constipation and mucous stools or constipation alternating with diarrhœa, unusual liability to coryza and bronchitis (or 'catching cold'). The feeding and hygiene of the child must be gone into: in low life, rickets is mainly due to deficiency of fat and protein in the diet, with excess of carbohydrate food, whereas in high life the diet is more likely to err by lack of freshness due to too careful sterilization or to the use of patent foods; rickety children all suffer from want of enough exposure to fresh air and sunshine. But if rickets is the main *predisposing cause* of infantile convulsions, it must be remembered that they are actually brought on by some secondary *exciting cause*, such as a gastro-intestinal disturbance with diarrhœa or vomiting, or reflex irritation of any sort. Whether *dentition* is in itself enough to account for convulsions is extremely doubtful, although that 'teething-fits' do occur is one of the things that every woman knows.

Epilepsy is one of the last causes of infantile convulsions that should be thought of, except when the fits occur for the first time in tolerably healthy children more than three or four years old. A bad family history of fits or of insanity would make epilepsy more probable; so would the occurrence of an aura before the fit, and the division of the fit into a tonic and a clonic stage, with biting of the tongue or cheeks. The repetition of fits for which there is no local or general cause, such as those described above, would be in favour of epilepsy, particularly if the sequence extended over a long period of time. But one fit undoubtedly facilitates the occurrence of another soon afterwards, so that the recurrence of convulsions for a few days or weeks in a rickety child is not enough to justify the diagnosis of epilepsy.

General Convulsions of Adolescents and Adults.—The convulsions of *epilepsy*, including both the major and the minor forms, are very variable in extent and duration. In the minor degrees, or *petit mal*, there is usually brief tonic or tetanic spasm, with loss of consciousness, but without clonus or convulsions. In severer cases this is known as *tetanoid epilepsy*, a tetanic spasm convulsing the patient for some seconds, or even for a minute or two, with great risk of death by asphyxia. In *partial epilepsy* the convulsions are confined to part of the body—the face, perhaps, or the arms and face. Midway between minor and major epilepsy Gowers places "*epilepsia media*, in which there is muscular spasm of tonic character, without the clonic spasm which follows when the tonic spasm is more severe." In *major epilepsy* the typical picture is as follows: after experiencing an aura or warning of some sort for a few seconds, the patient is seized with a general tetanic spasm, cries out, and falls to the ground, this tetanic or tonic stage lasting for from five to thirty seconds. This then gives place to the clonic stage, or convulsions, with foaming at the mouth, and clonic jactitations that are often unequal on the two sides of the body. After a few minutes the clonus dies away and the patient is left comatose or stupefied, with a headache that is slept off in the course of the next few hours. Consciousness is always lost in true epilepsy ; the extent and duration of the convulsions, however, are highly variable. The fits of *Jacksonian epilepsy* are rarely generalized ; the condition is considered below. In true epilepsy there is no known organic lesion of the brain ; the loss of consciousness and the convulsions are due to some unknown functional disturbance of its action ; but apparently identical fits may occur in the course of a number of diseases in which organic lesions are present either in the brain or elsewhere, and to these the name *epileptiform convulsions* is given. They are seen most often in *uræmia*, in which the kidneys are severely diseased and toxæmia results ; the patient exhibits the characteristic picture of advanced renal disease, with headache, high blood-pressure, hypertrophied heart, albuminuria, probably retinal changes, and anæmia ; or may have a stricture of the urethra or an enlarged prostate with secondary ascending nephritis ; or may be the subject of renal tuberculosis perhaps. It must not be forgotten that transient albuminuria is commonly present after fits due to any cause whatever. In the intervals between uræmic convulsions the patient may remain unconscious.

The convulsions occurring in connection with *pregnancy* are known as *eclamptic fits*, the condition as *eclampsia*. The majority of such convulsions come on before labour, some during labour, and 15 or 20 per cent during the first week after parturition ; any fits occurring after this are probably due to some cause—uræmia, for example—other than pregnancy or parturition. In many cases the fits occur suddenly and without any warning, or after no more than a brief period of headache or restlessness, or after vomiting. Eclampsia appears to be an auto-intoxication accompanied by a profound disturbance of the protein metabolism ; its primary cause is in the placenta. Its diagnosis can rarely be difficult. There is nearly always albuminuria, and some observers regard puerperal eclampsia as one variety of uræmia.

Epileptiform convulsions may occur in *severe heart* or *lung disease*, and, indeed, in the terminal stages of many disorders, due in part to asphyxia, in part to toxæmia. Like certain obstinate infantile convulsions, they may often be stopped by the administration of oxygen.

In *Stokes-Adams' disease* (p. 83), epileptiform or apoplectiform convulsive seizures occur from time to time, no doubt due to the asphyxia and cerebral anæmia resulting from temporary cessation of the heart's action. The radial pulse is habitually slow in this disorder, but becomes suddenly slower at the time of the 'attacks,' beating perhaps forty or thirty or even only twenty times to the minute ; the cardiac auricles, on the other hand, beat at the normal rate. The patients are usually arteriosclerotic people in the second half of life ; if they are seen in their convulsions, the diagnosis of apoplexy will probably be made, only to be corrected later when it is found that the attack leaves no paralysis or paresis behind it, that similar seizures have occurred before, and that the pulse becomes excessively slow during the seizures.

General convulsions due to direct irritation or disease of the brain may occur in a large number of *cerebral lesions*, unilateral or bilateral, most commonly in the latter. In most of these there will be other signs or symptoms of disease, especially optic neuritis, that should suffice to clear up the diagnosis. Such convulsions may be seen in *meningeal*, *subdural*, or *arachnoid hæmorrhage* ; in *meningitis* due to the *B. tuberculosis*, Weichselbaum's *meningococcus*, or other microbes ; in *cerebritis* ; in congenital anomalies of the brain such as *porencephalus*, *hydrocephalus*, and the abnormalities met with in idiots and mentally defective children generally ; and in *cerebral* or *cerebellar abscess*, *tumour*, or *aneurysm*, when sufficient growth has taken place to raise the intracranial pressure generally. In another group may be placed those cases in which extensive degenerative changes have taken place in the brain ; fits are common in the second and third stages of *general paralysis of the insane*, when other signs, such as defective memory and judgement, grandiose ideas, inequality or reflex immobility of the pupils, blurred speech, tremors of the tongue and face, loss or exaggeration of the deep reflexes, and muscular weakness may be looked for ; in the insanity of *chronic alcoholism*, with its tremors and inco-ordination, its marked sensory perversions, and its paramnesia or illusions of memory ; and in *cerebral syphilis*, where the lesions may be either vascular, gummatous, meningeal, diffuse, or a combination of any or all of these, and the main symptoms are headache, insomnia, attacks of aphasia and hemiplegic or epileptiform convulsions, paralysis of cranial nerves, and in addition dementia in the diffuse cases. *Chronic plumbism* may produce cerebral symptoms of the most varied kind (*saturnine* *encephalopathy*), from simple headache to acute mania, and amongst the phenomena, convulsions of epileptiform type may be prominent. The diagnosis is based upon the history, the occupation, the other symptoms of lead poisoning (p. 34), and perhaps upon the discovery of lead salts in the urine.

Lastly must be mentioned the general convulsions of the hysterical and of malingerers. In *hysteria*, the fits are noisy and protracted performances, the movements more or less purposive and quite unlike clonus ; the patient becomes red in the face rather than blue or white ; consciousness is not lost, attempts to open the eyes are resisted, pressure into the supra-orbital notch causes withdrawal of the head, the sufferer's hand is withdrawn if pressure is made between a nail and its matrix ; the sphincters are not relaxed, and the tongue or cheeks are rarely bitten. The convulsions are brought on by some emotional upset, and tend to cease when unsympathetically received. The *malingerer* may display no little art and skill in his convulsions, which are modelled on those of epilepsy ; here again the sufferer is red in the face rather than blue, although he may breathe stertorously, and, with the help of a little soap, foam at the mouth ; consciousness is not lost, the corneal reflex is present, the head and hand are withdrawn from painful impressions ; the sphincters are not relaxed ; perspiration is usual ; it is said that in epilepsy, if the hands are clenched, the thumb is buried in the palm, whereas the malingerer clenches it outside the fingers ; on the detection of its character, the simulated fit ends as suddenly as it began.

Unilateral Convulsions.—The convulsions in *apoplexy* are habitually limited to one side of the body. The onset of apoplexy, more often gradual than sudden, is generally preceded by headache, dizziness, and tingling or weakness in some part of the body ; and it is more marked in cerebral hæmorrhage than in embolism or thrombosis. The loss of consciousness comes on earlier and persists longer in cerebral hæmorrhage than in the other two conditions. When the convulsions are prominent the case is described as one of *epileptiform apoplexy*. *Cerebral hæmorrhage* is commoner in middle-life, in persons with high blood-pressure and hypertrophied hearts, and in the subjects of arteriosclerosis ; *cerebral embolism* is associated with endocarditis or intracardiac thrombosis, and occurs oftenest in young patients with heart disease ; *cerebral thrombosis* is seen in syphilitic patients, and in those with vascular disease, and is characteristically of slow onset after premonitory warnings.

In *cerebral abscess* and *cerebral tumour* convulsions are not very common, and usually appear only after the diagnosis has been made clear by the occurrence of such cardinal symptoms as headache, vomiting on change of position, optic neuritis (choked disc), and localizing signs pointing to intracranial tumour ; but it may happen that an epileptiform fit with unilateral or bilateral convulsions is the first sign that anything is wrong, or at any rate the first thing that makes the patient consult a medical man. The headache that follows a convulsive seizure is likely to be very severe and prolonged. Of the two, *cerebral abscess* is the more likely in patients with chronic suppurative disease of the ear or nose, or of the facial and frontal sinuses. Meningitis—especially *tuberculous meningitis* in its later stages—often exhibits unilateral or bilateral convulsions, squint and other local paralyses, more or less coma or mental apathy, gastro-intestinal symptoms, Cheyne-Stokes breathing, and irregularity of the pulse-rate and temperature ; lumbar puncture and examination of the cerebrospinal fluid (p. 304) may be required in establishing the diagnosis and in distinguishing between the tuberculous, the suppurative, and the epidemic cerebrospinal—'spotted fever'—forms.

The unilateral convulsions of *Jacksonian epilepsy* are rarely difficult to diagnose. The patient usually gives a history of head injury, and often a cranial scar or irregularity is to be found. There is no loss of consciousness during the attack, except in very severe and inveterate cases ; usually only one limb is involved, and an aura of some sort usually precedes the convulsions, which exhibit a characteristic 'spread'—beginning in a single muscle or group of muscles, and spreading thence to the muscles whose cortical areas of representation adjoin that of the muscle first involved. In Jacksonian epilepsy there is almost always an irritative lesion of the motor cortex or its immediate vicinity, due to trauma, syphilitic meningitis, or new growth ; paresis or paralysis of the affected muscles follows the convulsions, and in the course of time becomes marked. The 'spread' is frequently characteristic ; if the face is involved first, the arm follows, and then the leg ; if the hand is attacked first, the convulsions spread up the arm, then to the face, last to the leg. In the severer cases, where the whole side of the patient is convulsed, consciousness is lost, and then the convulsions may become bilateral.

Unilateral convulsions do not occur often in *epilepsy* or *infantile convulsions*, or *epileptiform convulsions*, and when they do there is a danger lest the diagnosis of apoplexy or some focal organic lesion of the brain be made. There is nothing in the character or distribution of the convulsions in these cases to enable a diagnosis to be made, and it is only after they are over, and it is found that no evidence of organic cerebral mischief is left behind, that their functional nature can be established. They are not followed by any permanent paresis, paralysis, or atrophy of the muscles on the affected side. It must be remembered that unilateral convulsions, the so-called 'apoplectiform' convulsions, may occur exceptionally in some of the conditions detailed under Group 2.

In *disseminated sclerosis*, hemiplegic apoplectiform attacks like those seen in general paralysis are not rare, often accompanied by aphasia. These attacks are both transient and recurrent. The patients are likely to exhibit other evidences of disseminated sclerosis—a childish and optimistic mental attitude, optic atrophy, nystagmus, impaired articulation, intention tremor, undue muscular fatigability ; the deep reflexes are commonly increased, Babinski's extensor plantar reflex is present, sensation is but little affected, and control over the sphincters is rarely lost until late in the disease. *A. J. Jex-Blake.*

CORNEA, ULCERATION OF.—(See ULCERATION OF THE CORNEA, p. 733.)

CORYZA.—(See DISCHARGE, NASAL, p. 178.)

COUGH.—Cough is a signal that something is irritating a branch of the vagus nerve or the cough centre, and is, in fact, nature's effort—often ill directed—to remove that something. Hence, to diagnose the cause of a cough it is necessary to know the branches of the vagus ; they are as follows :—

(1) A small meningeal branch, of no interest as causing cough, though it may possibly account for vomiting in meningitis ; (2) Arnold's branch to the ear—a cause of cough, though a rare one, due to affections (wax, eczema, etc.) of the external ear ; (3) Pharyngeal branch—a frequent source of cough ; (4) Superior laryngeal branch—sensory to base of tongue, larynx, etc., the most frequent source of cough, with or without visible changes ; (5) Inferior laryngeal branch—motor for action of coughing, not a cause of cough, but of inefficiency and other peculiarities in the act of coughing ; (6) Cardiac branches—indirect causes through circulatory failure ; (7) Pulmonary branches—concerned in the cough of gross pulmonary or pleural disease ; (8) and (9) Œsophageal and pericardial branches—possible but most rare causes ; (10) Gastric branches—occasionally dyspepsia causes a cough.

The irritants to which the surfaces of the distribution of these nerves are exposed may be classified into : (1) Foreign bodies, e.g., dust, food, tobacco smoke, etc. ; (2) Excess of natural secretion ; (3) Pressure and inflammation ; (4) Acute or chronic simple debility or increased irritability, e.g., after influenza, etc.

In dealing with the treatment, there is no better division of coughs than into those which are helpful and those which are not, and the same division is most useful in arriving at a diagnosis of the cause of a cough, for if the cough succeeds in its object—the removal of the offending material—we can see, or at least enquire about, its nature, and this will at once give a strong clue to the locality of the irritable point, and very possibly also to the morbid process going on. Hence the first questions to ask a patient with a cough are : " Do you bring anything up ? " " What do you bring up ? "

Cough without Expectoration.—If the answer to the first question be, " No, the cough is just a troublesome dry cough, with no expectoration at all," we at once begin to think of the purely reflex coughs produced by an irritant which the cough itself is powerless to remove, and though we may often make a short cut to a diagnosis by other means of investigation, or observation of the general condition, the following routine should be followed if no prominent clue offers itself :—

1. Examine the external ear for wax, eczema, etc., although this is a comparatively rare cause of cough, except in the special experience of aurists.

2. Enquire whether any ordinary irritant, such as tobacco smoke, etc., brings it on ; this, of course, at once raises the suspicion that the nasopharynx or larynx is unduly

sensitive, and should lead to a careful examination of the region, whereupon a cause may be detected at once, such as chronic inflammation of any sort, or a long pendulous uvula, somewhat œdematous, or showing other signs of acute inflammation. Conditions of undue irritability without anything to see occur after influenza or whooping-cough, and indeed remain long after the acute trouble has passed away from the regions; therefore enquiry must be made for some such illness. Such a cough is often seen when convalescents go into a cold bedroom, or get into cold sheets at night.

3. Ask the patient to cough voluntarily; the curious barking or rough cough of laryngitis and of pressure on the trachea from aneurysm or growth, also the very striking cough of paralysis of the vocal cords, at once betray themselves; there is no mistaking them when they have been once or twice heard in a hospital ward; the same remark applies to the cough of whooping-cough.

4. Examine the chest carefully for heart disease or early phthisis; the cough of both these conditions is commonly dry; so too is the cough of the early hours of an oncoming bronchitis or pneumonia, but these can scarcely fail to give other indications. Children often suffer from very troublesome dry cough, sometimes persisting for months, as the result of reflex irritation from caseous or inflamed bronchial glands; the latter may be impossible of diagnosis from physical signs, but they can often be seen very clearly with the *x*-rays (*Fig.* 61).

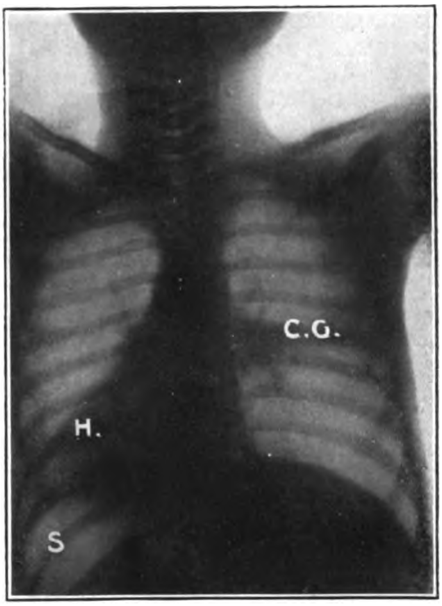


Fig. 61.—Skiagram showing a large caseous gland in a child, causing cough persisting for months without expectoration, and difficult to relieve by medicines. H, Heart; S, Gas in stomach: CG, Caseous gland.

5. If no cause reveals itself by now, the stomach must be thought of, and its functional and physical conditions enquired into and examined, and only after negative results from all these enquiries and procedures may we think of a simple hysterical cough.

Cough with Expectoration.—Expectoration generally makes the task of diagnosis much easier, and from the simple inspection of a spittoon it is frequently possible to make an almost complete diagnosis of a case: the very sticky sputum of any acute inflammation in its early stages, the rusty sputum of pneumonia, the stink of abscess or gangrene of the lung and of bronchiectasis, the nummulation of phthisical sputa, the frothy sputum of bronchitis, are very commonly quite typical and unmistakeable. Small blood-clots make us apprehensive of early —but well-marked—phthisis, or of pharyngeal conditions, or of mitral stenosis; streaks of blood point to acute laryngitis or bronchitis; profuse hæmoptysis almost diagnoses acute phthisis in the absence of signs of an aneurysm or growth. Pus is a factor common to all inflammations of mucous membranes, and therefore in itself is of but little diagnostic value, though its quantity, colour, and odour may be very suggestive of abscess or excavation, or of an hepatic abscess ruptured into the lung, of gangrene, or stinking empyema. With hepatic abscess the sputum sometimes has an almost pathognomonic anchovy-sauce appearance.

In any case of cough with sputum it is wise to have a microscopical examination of the latter, particularly for tubercle bacilli.

The Age of the Patient.—In babies and quite young children most of the more unusual causes of cough can be excluded at once on the mere fact of age, but the presence of a foreign body in the larynx is one of the unusual ones to be remembered, especially if the cough has come on suddenly in the midst of health. Bronchitis, bronchopneumonia, tubercle, pneumonia, whooping-cough, and diphtheria, are far and away the most common causes in these young subjects, and owing to the absence of expectoration they do not reveal their presence without careful examination of the chest and throat. From infancy to middle life, the age of the patient gives but little assistance in determining the diagnosis; but about middle age chronic bronchial troubles, quiet pleurises, growths, aneurysms, etc., become increasingly obtrusive, giving rise to a persistent cough, and only careful routine examination of the chest will reveal their presence.

How long have you had the cough? Much information may be derived from the answer to this question, for a cough that has only lasted a few days, but in that time has become sufficiently severe to cause the patient to seek advice, is practically certain to belong to the group caused by acute trouble, easily detectable when the chest is examined carefully; whereas, on the contrary, a cough that has lasted some months, and yet seems to the patient uncertain in its causation, is very likely to be due to some of the obscurer conditions, pressures of aneurysms or glands, etc., which need care to discover. The *x*-rays are valuable in detecting thoracic aneurysms and new growths, and they are also of service in demonstrating phthisical and other lesions in many cases; skiagraphic evidence must never be relied upon by itself, however; it should always be interpreted in terms of the other clinical data and physical signs. In an obscure case, however, *x*-ray screen observation should not be omitted, as it will now and again be the only means of clearing up the diagnosis.

When does the cough come on? A cough in the morning only is suggestive of bronchial catarrh with accumulation of secretion during sleep. A cough on getting into bed suggests laryngeal irritability or a long pendulous uvula; but one that wakes the patient after he has gone to sleep makes one apprehensive of phthisis in the absence of other indications of obvious acute chest changes. A cough on exertion suggests heart weakness, and in determining the presence of this, the finest discrimination is required in auscultation, for these are typically the cases of morbus cordis without a bruit in which frequency of rhythm and good differentiation of the first and second sounds are all important for a diagnosis. Shortness of breath will generally be a marked symptom associated with the cough in these cases (see BREATH, SHORTNESS OF, p. 87).

Has the voice altered since the cough appeared? Laryngeal inflammations or paralysis of a vocal cord are suggested by an affirmative answer, and the larynx must be examined carefully, the more carefully the more nearly the patient is approaching to the period of life when growths are more common.

Cough and Vomiting.—These two complaints are not infrequently made together by patients, and there is a very useful but often forgotten question to put, viz., "Are you sick independently of the cough? or do you cough till you are sick? Yes to the first suggests stomach trouble; yes to the second suggests bronchial trouble or whooping-cough.

Fred. J. Smith.

CRACKLING, EGG-SHELL.—This is a condition closely allied to CREPITUS (p. 152); if subcutaneous emphysema, arthritis, and tenosynovitis can be excluded, it is nearly always a symptom either of osteosarcoma, if it occurs in connection with a long bone, or of hydrocephalus or craniotabes in the case of the occipital or other cranial bones. The *x*-rays may assist the diagnosis (p. 673); if there is a tumour connected with the end of a long bone which exhibits egg-shell crackling with or without pulsation, it is almost certainly an osteosarcoma.

Herbert French.

CRAMPS are involuntary tetanic muscular contractions accompanied by sharp pain in the voluntary muscles involved. Temporary paralysis of movement, partial or complete, is often associated with cramp. Similar painful spasms of the involuntary muscles are referred to as colic. In most instances, cramps result from *over-exertion* of the affected muscles. The cramp comes on at once, or after a short delay, or when the attempt is next made to use the muscles involved. The most striking example of this is *swimmer's cramp*; in this the victim is overtaken suddenly by painful spasm and paralysis of the muscles of the leg or legs, or of the legs and arms; he is likely to drown unless help is speedily forthcoming. Similar but less extensive cramps are not rarely experienced by persons taking part in the more violent of outdoor games—football, hockey, lacrosse, etc.; some particularly sudden or violent effort may be followed by cramp in the thigh- or calf-muscles. Similar cramps of the legs are familiar to rowing men and ballet-dancers. Certain people have a great proclivity to cramp during the night, and it seems to return with less and less provocation the more often it is experienced. Stokers and iron-founders who do heavy bodily work in much overheated atmosphere are liable to heat cramps, severely painful spasms in the muscles of the limbs and abdomen, in attacks lasting for many hours and followed by great weakness. The diagnosis of cramps due to over-exertion, directly associated as they are with a definite history of muscular strain, should not be difficult. They rarely become so severe as to prevent their victims from continuing to take part in the occupations that provoke their occurrence.

It is quite otherwise, however, with patients who are afflicted with the so-called *professional cramps* or *occupation neuroses* that result from chronic strain and over-use of certain groups of muscles. They occur in such persons as writers, typists, telegraph operators, compositors, painters, tailors, seamstresses, dairymaids (from milking cows), pianists, flute-players, violinists, 'cellists, drummers, blacksmiths, file-makers, cigarette-rollers, and so forth. In all these employments, particular groups of muscles are in constant and special employment. If they are overworked they may become the seat of cramps and aching pains—professional cramps—as soon as they are used ; their movements lose their delicacy, and become inco-ordinated and spasmodic. A fine tremor is very commonly to be observed in the affected limb. It is probable that over-use alone is not enough to set up these cramps. Anxiety, ill-health, local injury or disease, and the inheritance of a neurotic temperament, all contribute to the establishment of professional cramps. These cramps have also been recorded in other occupations, and as affecting other groups of muscles : in treadler's cramp, the hamstring muscles and glutei are affected ; in cornet player's the tongue, in watchmaker's the orbicularis oculi, may be attacked. As a rule, the diagnosis of a professional cramp is not hard, but it is necessary to make sure that neither organic nervous disorder nor local disease is present. Thus the physical signs, though hardly the symptoms, of writer's cramp may be present in such diseases as paralysis agitans, disseminated sclerosis, tabes, general paralysis ; brachial neuralgia might simulate the neuralgic forms of occupation neurosis, but it is free from cramps. Again, affections of the joints or of the tendons at the wrists, such as chronic rheumatism, rheumatoid arthritis, tenosynovitis, tuberculous infection, may all give rise to pain in, and interfere with, the movements of the hand. Again, writer's cramp may be so much feared by nervous patients that their right hand may become so stiff, or weak, or painful, that they can no longer write : objective signs of the cramp, however, are lacking in such cases, which are cured by the re-establishment of the patient's self-confidence.

Cramps are the main feature of *tetany*, a disease characterized by the occurrence of paroxysmal or continued tetanic spasms of the extremities, and increased excitability of the nerves and muscles to electrical or mechanical stimulation. Tetany occurs in many different conditions, and at any age. In infants and young children it is a complication of rickets, improper feeding, and acute gastro-intestinal disorders, either with or without diarrhœa and vomiting. Epidemics of tetany in young adults, probably resulting from food-poisoning, have been described on the Continent, though not, apparently, in Great Britain. In nursing women, tetany may follow prolonged lactation ; or it may develop during pregnancy and recur in successive pregnancies. It may result from the removal of too much or all of the thyroid gland in either sex. Tetany complicates a certain proportion of the cases of gastrectasis, occurring whether the dilated stomach has been washed out or no. A few instances are on record in which tetany followed the acute specific fevers, enteric fever, or poisoning by chloroform, lead, or ergot. In fine, it may be said that tetany is usually due to acute or chronic digestive troubles, the painful spasms being evidence of the absorption of some toxin from the gastro-intestinal tract in most cases. The cramps of tetany are mainly in the extremities and paroxysmal ; they may continue, however, for hours or days, and are very painful. During the spasms, the fingers are extended at the terminal and flexed at the metacarpophalangeal joints and pressed together, while the thumb is adducted and flexed into the palm, so that the so-called 'accoucheur's hand' (*Fig.* 1, p. 3) is produced. The wrist and elbow are flexed, the arms being usually folded over the chest ; exceptionally the elbow may be extended stiffly. The toes are drawn together and flexed, the foot is arched and turned inwards, and the ankles and knees extended. Usually the limbs only are involved, but in severe cases cramps occur in the face, neck, and even the trunk, when respiration may be embarrassed seriously. The rigid muscles are very tender to the touch. Three special signs are present in the intervals between the attacks of tetany, and are valuable in diagnosis : these are Trousseau's sign, or reproduction of the paroxysm by manual compression of the nerves or blood-vessels supplying the affected parts ; Erb's sign, or hyper-excitability of the motor nerves to electrical currents (0·5 to 2·0 milliampères) ; and Chvostek's sign, or reproduction of the spasm in the facial muscles by tapping either on the muscles themselves or on the facial nerve. Tetany must be diagnosed from *tetanus*, in which the spasms begin in the head and neck, and trismus is an early symptom ; and from strychnine poisoning, where the spasms are clonic rather than tetanic, and affect the whole body, and not the extremities primarily or principally. In the *carpo-pedal spasms* of rickety children or of infants with severe gastro-intestinal catarrh, the cramps are similar to those of tetany, but are transient, and perhaps affect the hands only, or the hands and arms. Such spasms may justly be regarded as identical with mild tetany. *Hysterical tetany* occurs, and is to be distinguished from true tetany by its association with other hysterical stigmata on the one hand, and on the other by the absence of Trousseau's and Chvostek's signs. Hysterical tetany may also, perhaps, be distinguished by its failure to respond to the exhibition of calcium salts ; the most recent view of true tetany regards it as the expression of hyper-excitability of the nerve-cells due to lack of calcium salts, and connects it with the parathyroid glands by supposing that they control the calcium-metabolism of the body.

Reference may again be made to the fact that cramps are prone to occur in patients debilitated by the *acute fevers* or *enteric fever* ; severe cramps in the legs and arms are often a highly troublesome feature of the convalescence from *cholera*. In many chronic diseases nocturnal cramps may give rise to no little distress, or may interfere seriously with sleep : in *gout, chronic Bright's disease, uræmia, alcoholic neuritis*, and almost any chronic wasting disorder, complaint of cramp is not infrequent, but in such instances more serious signs or symptoms of disease will be evident.

A. J. Jex-Blake.

CREPITUS is a term generally used to denote the grating or crackling sensation and noise produced when two ends of a broken bone grate together. It is the most conclusive sign of a *fracture* ; but it causes the patient so much pain that whenever the *x*-rays can be employed attempts to obtain crepitus should not be made with any vigour. Apart from fracture of a bone, crepitus is also to be felt and heard in joints affected by dendritic synovitis, or still more so in cases of *osteo-arthritis* ; the term 'silken crepitus' has been used for the sensation felt on moving such a joint, comparable to the rubbing together between the fingers of two pieces of stout silken ribbon. *Tenosynovitis*, especially around the flexor tendons at the wrist, may also produce a very marked feeling of crepitus, especially in cases where the tendon sheaths contain melon-seed bodies.

When there is an enlargement of a bone without fracture, and when on palpation a feeling of crepitus or egg-shell crackling is obtained, it is an indication that the tumour is a deposit of secondary carcinoma or a primary rarefying *osteosarcoma*, which may sometimes be felt to pulsate also. The diagnosis may be assisted by the use of the *x*-rays (*Fig.* 62).

Rarefaction of the bones of the skull, either as the result of syphilitic lesions in adults, or of *hydrocephalus* or *craniotabes*, especially in the occipital region of congenital syphilitic and rickety infants, may make the skull bones so thin that they readily bend on pressure, and sometimes the result is a sensation of crepitus. The diagnosis is generally obvious.

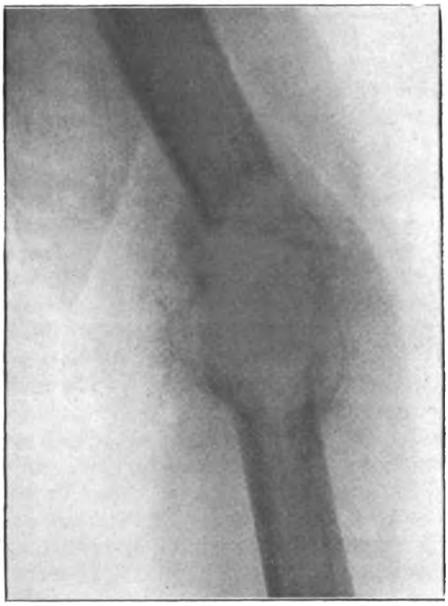


Fig. 62. —Skiagram of a growth in the humerus in a male, age 72, who was sent in as a case of primary sarcoma ; the tumour proved on operation to be a secondary deposit of cylindrical-celled carcinoma ; the seat of the primary growth was undiscovered at the time.
(*Skiagram by Dr. C. Thurstan Holland.*)

Quite apart from bony, arthritic, or synovial changes, a characteristic feeling of crepitus may be felt beneath the skin when gas or air has accumulated in the subcutaneous tissues as the result of surgical EMPHYSEMA (p. 203). *Herbert French*

CRUSTS ON THE SKIN.—(See SCABS, p. 599.)

CUD-CHEWING.—(See MERYCISM, p. 388.)

CURSCHMANN'S SPIRALS consist of a highly refractile central fibre, and a sinuous wavy sheath of mucus. They may be half an inch in length, but they are very slender. They occur in the sputum of patients suffering from true spasmodic asthma, and they may be associated with eosinophile corpuscles and Charcot-Leyden crystals. They are pretty objects, best seen in fresh sputum, but their diagnostic significance is very limited, first because they are so often absent in cases of undoubted asthma, and secondly because they have been found in bronchiolitis without asthma. They seem to be casts of the finest bronchioles. It is probable that, if there were doubt as to whether a given case were one of primary emphysema and bronchitis, or of spasmodic asthma that had led to emphysema and bronchitis, the occurrence of typical Curschmann's spirals would point to the latter. There are, however, other means of arriving at the same conclusion, particularly the history, the age at which the first attack began, and the presence or absence of EOSINOPHILIA (p. 219). *Herbert French.*

CURVATURE, SPINAL.—In the diagnosis, the first thing is to distinguish between lateral and antero-posterior deformities; but in a good many cases scoliosis or lateral curvature is complicated by antero-posterior deformity, kyphosis, or lordosis as well, and in a few instances of angular kyphosis due to caries there is some lateral deviation, which is generally much more abrupt than is the curve of scoliosis. A good way of demonstrating lateral curvature is to pencil the skin over the spinal processes.

LATERAL CURVATURE.

The following are the most important causes of lateral curvature:—

Inequality in the lengths of the lower limbs
Weakness of the muscles of the back associated with bad habits of standing or sitting
Carrying heavy weights with one arm or on one shoulder
Rickets
Wry-neck, or other causes of asymmetry of the head and shoulders, such as Sprengel's shoulder
Paralysis of the muscles of the back, as in infantile paralysis, peripheral neuritis, especially that following diphtheria, and some of the muscular dystrophies
Shrivelling of one side of the chest as the result of empyema or fibroid lung
Hysteria.

Inequality of the lengths of the lower limbs is one of the commonest causes of lateral curvature; therefore it is very important to find out at once if the legs are equal. The most reliable and easy method of determining this is to get the patient to stand up with both knees straight and without resting a hand upon anything. The observer then stoops in front of the patient and places his thumbs, with their extremities upwards, exactly upon the prominence of each anterior superior spine. The eye can then detect even a slight difference in the level of the two spines. This method is far more reliable than measurement from the anterior superior spines to the malleoli. Moreover, the latter method does not show shortening due to flexion and adduction of the hip-joint. Further, the foot may be fixed in a position of talipes equinus, which may make a short limb apparently longer than its fellow, so that the anterior spine on the corresponding side may be elevated. When the anterior spines are on a different level, the trunk leans towards the lower spine, but in order to maintain the erect position the upper part of the body becomes flexed to the opposite side. Thus, the spine in the lumbar region develops a curve with its convexity to the side of the short limb. Lateral curvature due to a shortened limb, in its early stages, is corrected at once by compensating the shortened limb, and it also disappears when the patient sits on a flat level surface. In the absence of inequality of the limbs, *muscular weakness* is by far the most common cause of lateral curvature. The spine does not become straight when the patient sits on a flat level surface; but in the early stages of the deformity the shape can be corrected somewhat by muscular effort.

Asymmetry of the chest following upon empyema or fibroid lung is easily detected. The shrivelled side is generally less resonant on percussion, and there are other signs of pulmonary disease.

Scoliosis secondary to *wry-neck* is usually slight, and limited to the cervical and dorsal regions. In growing youths the *carrying of heavy weights* with one arm or upon one shoulder is a common and important cause of scoliosis, and it is therefore necessary to go into the question of occupation and habits. For instance, nursery-maids and butchers' boys are very apt to develop lateral curvature as the result of carrying burdens upon the right arm.

The lateral curvature due to *rickets* is recognized by the unusually early onset, during the first or second year, and the signs of rickets in other parts, especially thickening of the lower end of the radius. The direction of the primary curve is sometimes explained by the pressure of the arm of the nurse who carries the baby too exclusively on one arm. Actual *paralysis of the spinal muscles* is a rare cause of scoliosis, and is to be recognized by the wasting of the spinal muscles, especially when this is more marked on one side. The sinking of the muscles due to rotation of the spine must not be mistaken for wasting. There is usually paralysis of other muscles, especially those of the leg. Scoliosis is often seen in the various primary muscular atrophies (p. 513), and in Friedreich's hereditary ataxy (p. 512).

Peripheral neuritis as a cause is nearly always due to diphtheria or sore throat. The history may indicate this, or there may have been other post-diphtheritic paralyses, notably that of the soft palate, with nasal voice and regurgitation of fluid through the nose. Cultivations should be taken from the throat, and the Klebs-Löffler bacillus (*Plate XXVIII, Fig. L*, p. 614) may be found if sought early enough. Occasionally the abdominal muscles may also be paralyzed in these cases, and this is a contributory cause of the curvature.

ANTERO-POSTERIOR CURVATURES.

These may take the form of (1) *Kyphosis*, (2) *Lordosis*.

1. **Kyphosis** or 'hump-back,' means a bending forwards of the upper part of the back on to the lower. The curve may be (*a*) *Angular*, and limited to a small portion of the back; or it may be (*b*) *Diffuse*, or even general, extending from the coccyx to the cranium.

(*a*). *Angular Kyphosis*.—The causes of angular kyphosis are:—(i) Tuberculous caries of the vertebræ; (ii) Growth of the spine; (iii) Hydatid disease of the vertebræ.

(i). *Caries* is by far the commonest cause, and it is very important to recognize the disease before the deformity becomes well marked. Unfortunately, it may be treated for a long time as stomach-ache or intercostal neuralgia, because the pain is referred to the abdomen and the intercostal regions. During its active stages it is easy to recognize it from its classical symptoms and signs. The patient avoids all jerky movements, walks with a stooping gait, and grasps with the hands any convenient article of furniture. The spine is tender on percussion, also on pressure upon the head or shoulders. Local rigidity of the back is noticed when the patient attempts to stoop. In later cases, paralysis of the legs may complicate the deformity. In the quiescent stages, the diagnosis is based on the characteristic local deformity and rigidity. Skiagrams, especially those taken from side to side, may afford material help by showing evidence of destruction of the bodies of the vertebræ (*Fig.* 195, p. 460). In some cases, lateral curvature may complicate or follow caries, and then the diagnosis is not easy. The disease may have affected the bodies of the vertebræ unevenly, leading to some lateral deviation, which is usually rather abrupt and associated with the local rigidity characteristic of caries.

(ii). *Growth of the spine* is a rare cause of angular curvature. Rapidly developed curvature in a patient after middle age may be due to secondary carcinoma in the bodies of the vertebræ, and bearing this possible cause in mind, the surgeon should go carefully into the history, and examine every possible source of primary carcinoma, particularly the breast. Primary or secondary sarcoma may also lead to deformity of the spine, and in some cases an *x*-ray examination may give evidence of the development of new bone in the growth, or of the absorption of the vertebræ.

(iii). *Hydatid disease* is a very rare cause of spinal curvature, and it is usually not limited to the spine.

(*b*). *Diffuse Kyphosis.*—The back may be bent forwards in a uniform curve extending from the coccyx to the cranium. This variety is common in *rickets*, owing to the premature assumption of the sitting position when the bones are soft and the muscles of the back are weak. When the patient is lying prone, the deformity can easily be corrected by raising the legs. Moreover, there are other signs of rickets, such as enlargement of the lower end of the radius, beading of the ribs, and delay in the eruption of teeth. A similar deformity arises from *muscular weakness* due to other causes, such as idiocy and congenital spastic paraplegia. In all of these there is an entire absence of rigidity of the spine. An extensive and uniform curve, affecting the cervico-dorsal region, is common during adolescence, and is due to muscular weakness, lazy habits, and the carrying of heavy weights. In its early stages the deformity is easily reducible, and as a rule is compensated by a marked lordosis in the lumbar region, and some tilting backwards of the occiput. It is often associated with lateral curvature, and in some cases may be partly due to shortness of sight. The condition is distinguished from caries by the diffuseness of the curvature, the absence of pain and local tenderness, and the comparative suppleness of the back.

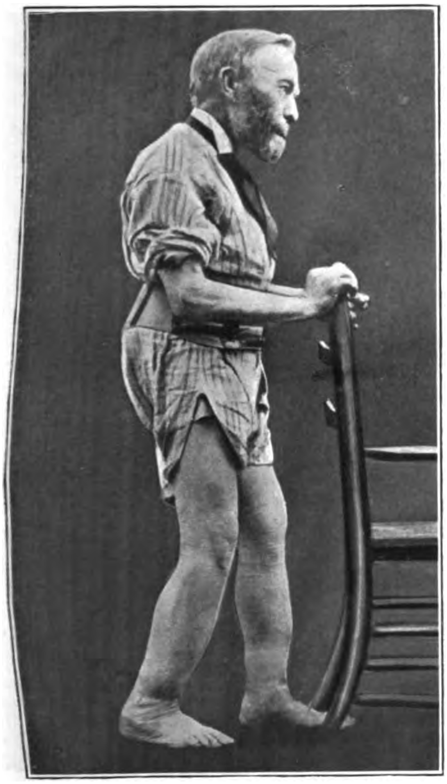


Fig. 63.—Osteitis deformans in a man.

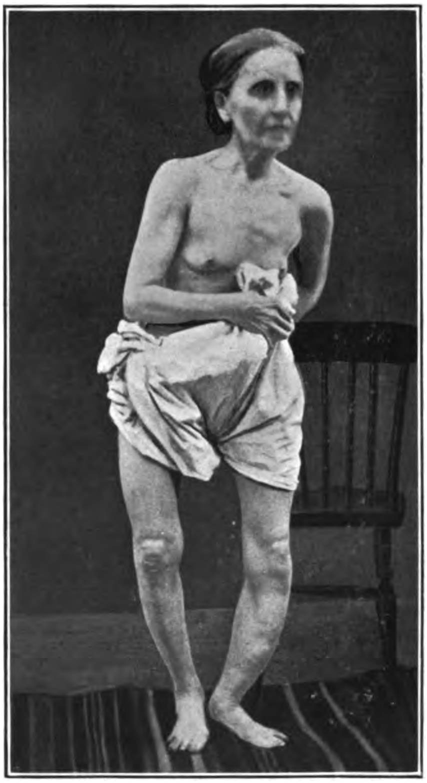


Fig. 64.—Osteitis deformans in a woman.

Kyphosis due to *spondylitis deformans* or to *osteitis deformans* (*Figs.* 63 and 64) is of a more uniform character without complicating lordosis, and the deformity is irreducible. There is generally evidence of the disease in other parts, such as osteo-arthritis, or the bending of the legs, and increase of the size of the head, which are due to osteitis deformans. Porters carrying heavy weights on the upper part of the back prematurely develop the kyphosis which is usually associated with old age. They frequently have a bursa over the seventh dorsal spinous process (*Fig.* 65).

2. **Lordosis, Hollow-Back.**—This deformity is only common in the lumbar and lower dorsal region. The natural hollow of the loin is exaggerated, and usually there is either primary or compensatory kyphosis in the cervico-dorsal region (*Fig.* 66). Lordosis is rarely primary, but it may be so in the early stage of lumbar or lumbo-dorsal *caries* in children, when the real cause of the deformity is apt to be overlooked. The abdomen is very prominent, and the back is not only hollow, but rigid and tender. Pressure upon the head also causes pain in the back. In some cases the deformity is exaggerated by induration or suppuration in the psoas muscle, which complicates this disease. Lordosis is not uncommonly due to *weakness or paralysis of the muscles* of the back (*Fig.* 66). It is particularly important to look for other evidence of primary muscular dystrophy. The upper part of the back is then thrown backwards to facilitate the maintenance of the erect position. Lordosis is often secondary to the flexion of *hip disease*, which must not be overlooked. Limitation of movement —especially of rotation of the hip-joint—and wasting of the thigh, serve to demonstrate the existence of this disease. Lordosis and the waddling gait may be the first indications of *congenital dislocation of the hip*. In this condition, which is almost confined to the female sex, the erect position is maintained only by throwing the shoulders backwards to an unusual degree in order to bring the trunk in a line with the heads of the femora, which are dislocated backwards. The suspicion of congenital dislocation of the hip may be confirmed by skiagraphy, by the gliding movements of the head of the femur upon the pelvis, the unnatural width of the hips, the hollow appearance of Scarpa's triangle, and by palpation of the head of the femur upon the dorsum ilii when the thigh is flexed, strongly adducted, and inverted. *Contortionists* usually have a good deal of lordosis owing to the unnatural suppleness of the lumbar spine and the elongation of the hamstrings. In all these conditions, the back is supple, and can be restored to its natural shape by placing the patient in the supine position and flexing the thighs.

R. P. Rowlands.

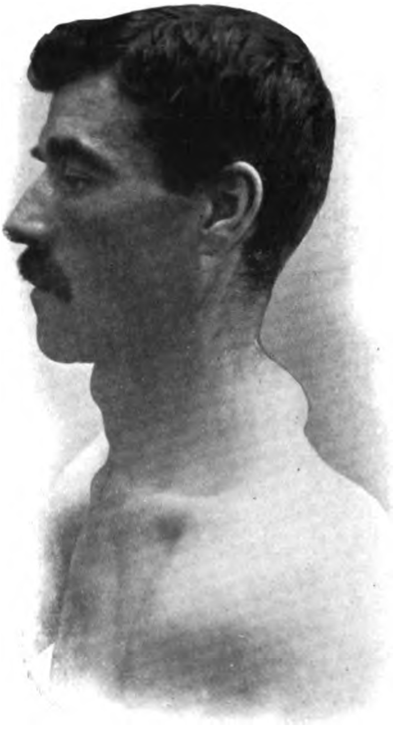


Fig. 65.—Deal porter's bursa over vertebra prominens.

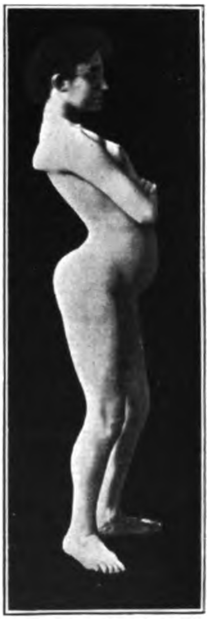


Fig. 66.
Myopathic lordosis.

CYANOSIS, EXTREME.—Extreme cyanosis, blueness, or lividity, is generally most marked in the face; next in the extremities, especially the hands, feet, ears, and penis; and least in the trunk. Cases in which it is a prominent symptom may be divided into two main groups, according as the cyanosis is present at or soon after birth, or occurs later in the life of a patient originally free from it. Congenital cyanosis of extreme degree is nearly always due to *malformation of the heart*, particularly *pulmonary stenosis* (*Fig.* 67). *Patent septum ventriculorum* may also produce the symptom, though not in so marked a degree, whilst *patent ductus arteriosus*, when it occurs by itself, is generally not associated with cyanosis at all. These three conditions all give rise to loud universal bruits, of which that due to pulmonary stenosis is purely systolic, with its maximum intensity in the second left intercostal space close to the sternum; that due to patent septum ventriculorum is also systolic, but has its maximum intensity lower down the sternum, usually between the two third spaces or fourth ribs; whilst the bruit of patent ductus arteriosus is not purely systolic, but continues through both systole and diastole, with its maximum intensity at the time of the second sound, and it is best heard in the third left intercostal space, about half an inch out from the sternum; all these bruits may or may not be accompanied by a corresponding thrill, the latter generally being least marked with patent septum ventriculorum. Extreme **Clubbing of the Fingers** and of the toes accompanies the cyanosis in most cases (*Fig.* 46, p. 111). In addition to these three types of congenital heart disease, there are other cases in which extreme cyanosis, with or without clubbing of the fingers, occurs without any definite bruits, and the diagnosis of the nature of the lesion can only be guessed at. There may or may not be transposition of the great vessels or of the viscera at the same time. Sometimes there is a single large vessel, the pulmonary artery coming off from the aorta; or there may be only one ventricle, or a single auricle. It is almost impossible to decide between the various possible lesions, unless there is one of the definite bruits just described. Anomalous cases seldom survive, but some cases of pulmonary stenosis or patent septum ventriculorum reach adult life, and patent ductus arteriosus often gives little inconvenience to the patient at all. It is to be remembered that patent foramen ovale is quite undiagnosable, that it causes no symptoms, and is present in a large percentage of normal people.

Fig. 67.—Morbus cæruleus: congenital pulmonary stenosis with extreme cyanosis.

Cyanosis developing in children or adults who have hitherto been healthy, is generally due either to laryngeal or tracheal obstruction, to lung lesions, cardiac failure, obstruction to the superior vena cava, or to some alteration of the blood itself, such as is found in splenomegalic polycythæmia, methæmoglobinæmia, sulph-hæmoglobinæmia, or the later stages of diseases associated with extreme loss of fluid from the tissues, especially cholera maligna. The differential diagnosis is usually easy up to a certain point; not a little cyanosis may result from taking certain *drugs* either in large quantities at a time, or in less quantities continually—veronal, trional, sulphonal, and acetanilide in particular. The urine in these cases often reduces Fehling's solution, and may contain methæmoglobin, recognizable by the spectroscope. The diagnosis depends on a knowledge of the drug that is being taken. Cases of *pancreatitis* often exhibit a peculiar cyanotic hue. The fact of *laryngeal obstruction* is generally obvious from the stridor, and from the way in which the larynx moves forcibly up and down with respiration. The cause of the obstruction may be less easy to determine. In a child, a digital examination of the back of the mouth should not be omitted, lest there be a *post-pharyngeal abscess* or a *foreign body*; in the absence of this, the most probable cause is *diphtheria*; though it may be difficult to diagnose forthwith between *laryngitis with intermittent spasm*, *laryngismus stridulus*, *acute obstructive laryngitis*, and *diphtheria*. Swabbings should be taken from the throat as far back as possible, and examined bacteriologically. The bacillus of diphtheria (*Plate XXVIII, Fig. L*, p. 614) may be found on direct examination of films stained by Neisser's method; but sometimes they cannot be found until cultivations have been made, and this takes upwards of twenty-four hours. If there has been no obvious cause for catarrhal laryngitis, such as the inhalation of irritant gases or a recent attack of acute bronchitis affecting the large tubes, it is better to assume that the condition is diphtheria until it is proved not to be so. The occurrence of other cases in the same house, or in the neighbourhood, may assist the diagnosis. Another condition which may simulate diphtheria from the extreme dyspnœa and cyanosis that result is the *inhalation of a foreign body*, such as a button, small shell, piece of food, a tooth, and so on; or obstruction to the trachea by a bulging *caseous* *gland* (*Fig.* 68). In an older person, *acute suffocative laryngitis* due to pneumococci or streptococci is associated with extreme cyanosis of rapid onset. Tracheotomy is necessary, and the diagnosis is arrived at upon bacteriological grounds. When similar acute infective changes occur, not in the larynx only but in the root of the tongue as well, thence infiltrating the deep structures of the neck, as in *angina Ludovici*, cyanosis and dyspnœa may be very marked; the diagnosis is suggested by the acute brawny swelling of the neck and by the changes in the floor of the mouth and tongue. Severe dyspnœa and cyanosis may accompany *goitres*, whether simple, exophthalmic, or malignant; the attacks may be paroxysmal even though the thyroid gland itself does not seem to vary in size; or the cyanosis and dyspnœa may be continuous when there has been rapid enlargement of the gland from rarities such as hæmorrhage into it, acute suppuration in it, or from progressive and extreme fibrosis of the organ such as is seen in *ligneous thyroiditis*, or Riedel's disease (see THYROID GLAND, ENLARGEMENT OF, p. 721). It is difficult to inspect the vocal cords in a child; but in an adult this is easier, and direct examination serves to distinguish between acute or ulcerative lesions of the larynx and laryngeal paralysis; the latter, sometimes the result of syphilitic degeneration of part of the vagal centre in the medulla, is apt to produce *bilateral abductor paralysis* with adductor spasm, which may come on acutely and simulate acute asphyxia from a foreign body. *Tuberculous*, *syphilitic* or *malignant*, *variolous*, *leprous*, *lupoid*, and *traumatic ulceration* of the larynx, may any of them become acutely infected by inflammatory organisms, and lead to comparatively sudden and severe laryngeal stenosis with acute cyanosis; the diagnosis will depend upon the history, bacteriological examination, and direct examination of the vocal cords. *Bright's disease* has

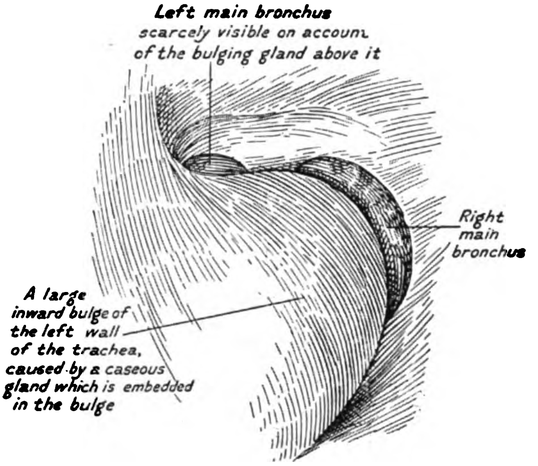

Left main bronchus scarcely visible on account of the bulging gland above it
Right main bronchus
A large inward bulge of the left wall of the trachea, caused by a caseous gland which is embedded in the bulge

Fig. 68.—Bronchoscopic appearance of a case of tracheal compression from enlarged glands. There was extreme dyspnœa and cyanosis in this case, relieved at once by the passage of the bronchoscope.

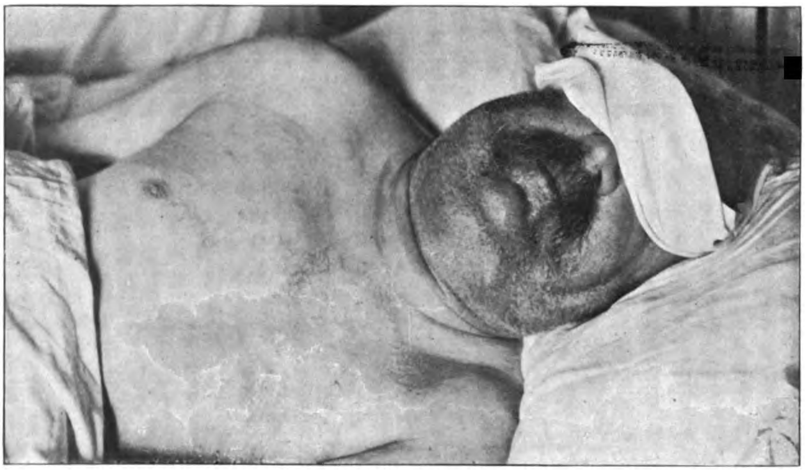


Fig. 69.—Obstruction to the superior vena cava by intrathoracic aneurysm. The patient, who was under the care of Dr. Moorhead, of Blindley Heath, was of normal appearance until, almost suddenly, his face and neck increased enormously in size and became violaceous without being definitely œdematous. There had been a brassy cough for some time previously. He survived the acute obstruction to his superior vena cava a few months. The photograph, taken when he was lying ill in bed, shows the bloated appearance that resulted.

sometimes caused similar symptoms, due to acute œdema of the larynx, and *potassium iodide* may do the same in those who are particularly prone to iodism. Knee-jerks should be tested, and the pupils examined, lest acute attacks of dyspnœa with cyanosis simulating laryngeal obstruction are due to the laryngeal crises of *tabes dorsalis*.

Growths of the lung, particularly if they give rise to pleuritic effusion or to obstruction of a bronchus, may cause progressive cyanosis ; the diagnosis is not as a rule easy in the earlier stages, but if there is evidence of progressive interference with the structures within the thorax, with ultimate stenosis of the superior vena cava, and the results of this, namely, œdema of the face and arms, together with cyanosis of these parts out of proportion to any similar change in the legs, the diagnosis lies between growth, aneurysm, and mediastinal fibrosis. The x-rays will sometimes be of material assistance in deciding. A rare but very alarming complication of thoracic aneurysm is for the latter to open suddenly into the superior vena cava ; the result is acute dyspnœa, extreme cyanosis of the face and hands, and bloated-looking swelling of the head, face, neck, arms, and upper part of the chest and back (*Fig.* 69). The diagnosis is suggested at once by the suddenness of the onset of the graver symptoms ; though these have also been produced in rare cases by such lesions as sudden *hæmorrhage into the mediastinum* or *thymus gland*, or similar *hæmorrhage into an intrathoracic sarcoma* or other new growth.

Phthisis, in the later stages, particularly when it advances rapidly and leads to generalized caseous bronchopneumonia, causes extreme cyanosis in some instances. The diagnosis will generally have been made long previously, from the symptoms, such as hæmoptysis, cough, and wasting ; from the abnormal physical signs which started at the apices of the lungs and were progressive ; and from the discovery of tubercle bacilli and elastic fibres in the sputum, though there are many cases of *miner's phthisis* (*Fig.* 70), or pneumonoconiosis, in which the lung trouble may be extensive, yet tubercle bacilli cannot be found ; there is doubt as to whether this condition is always tuberculous and not sometimes syphilitic.

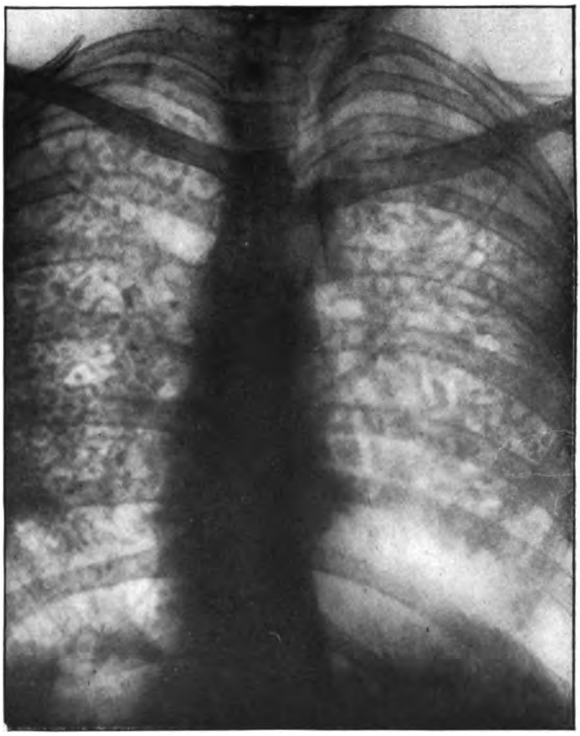


Fig. 70.—Skiagram of a case of miner's phthisis in a man, age 35, who had been a worker in the Rand gold mines. Three years' history of lung trouble, though the man had won a mile race a year ago. Now a typical asthmatic with sputum but without detectable tubercle bacilli. He died a few months after the skiagram was taken.
(*Skiagram by Dr. C. Thurstan Holland.*)

Pneumothorax, when it comes on suddenly in a patient who has had no symptoms hitherto, leads to acute dyspnœa and cyanosis, which presently pass off ; the physical signs are pathognomonic, and the cause is generally tubercle.

Embolism of the lung, if the artery occluded is of large size, may cause sudden death, so that the patient hardly has time to become cyanosed ; when the embolus blocks a smaller vessel, lividity, dyspnœa, intrathoracic pain, and hæmoptysis are the most prominent symptoms ; the diagnosis is suggested by the suddenness of the onset in a case in which there is a cause for embolism, particularly thrombosis of a vein such as the femoral or iliac, or a recent surgical operation in the neighbourhood of a large vein such as those in the abdomen, or otitis media with lateral sinus thrombosis, or a cardiac lesion such as infective endocarditis of the right side of the heart. There may be no abnormal physical signs ; but sometimes the resultant infarct may be detected by the impairment of percussion note, the deficient vesicular murmur, and the development of a rub over it.

In childhood, the commonest lung affection to produce extreme cyanosis is *bronchopneumonia* ; the diagnosis is generally obvious, though it is not always easy to determine whether, in a case in which there is some evidence of laryngitis at the same time, the cyanosis is due mainly to the laryngeal obstruction or to the intrapulmonary lesions. Each may cause extreme sucking in of the intercostal spaces and convulsive movements of the chest as a whole ; but the best measure of the degree of laryngeal obstruction is the violence of the up-and-down movements of the larynx itself. There may or may not be *empyema* associated with bronchopneumonia ; but the degree of cyanosis will not help to distinguish between these two ; needling of the chest will be resorted to when there is ground for supposing that empyema may be present. Severe *bronchitis and emphysema* in middle age often lead to marked cyanosis and orthopnœa, owing no doubt to the failure of the right side of the heart to which the lung trouble gives rise. The over-distended condition of the chest, its small difference between maximum inspiratory and maximum expiratory girths, the deficiency of the vesicular murmur, the rhonchi all over it, and perhaps non-consonating râles at the bases, would indicate the diagnosis, particularly if the patient has inelasticity of the skin of the back of the hands, and has suffered from similar attacks for some years past, especially in the winter. The chief difficulty will be to determine whether the cause of the cyanosis is pulmonary or cardiac (see below). *Lobar pneumonia* as a cause of acute cyanosis is diagnosed chiefly by a history of sudden onset, the continuance of pyrexia for a week or ten days and ending by crisis (*Fig.* 272, p. 642), the rapid respiration-rate in proportion to the temperature, the viscid rusty sputum, and herpes labialis. Occasionally the pyrexia terminates by lysis or in some other atypical way (*Fig.* 71), instead of by crisis ; or it may rise again after the crisis, particularly when empyema follows (*Fig.* 71). Sometimes the diagnosis is made when no abnormal physical signs can be detected ; but if over a large portion of a lobe there is at the same time impairment of note, with bronchial breathing, bronchophony, pectoriloquy, without râles at the height of the malady, but with fine crepitations at the beginning of the attack, and with redux crepitations as the bronchial breathing disappears after the crisis, the diagnosis will be obvious, especially if during the fever there is a great deficiency or complete absence of chlorides from the urine.

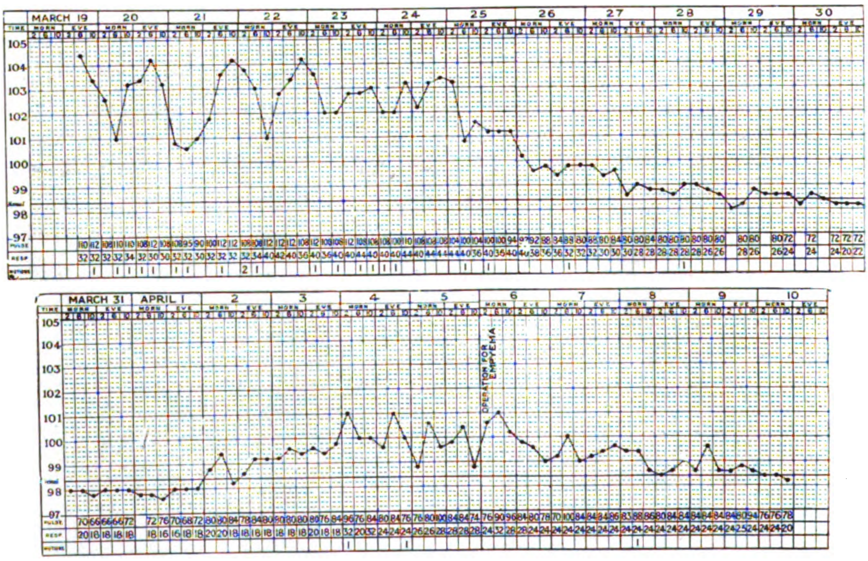


Fig. 71.—Termination of a case of lobar pneumonia by lysis followed by empyema.

Asthma is sometimes very difficult to distinguish from bronchitis and emphysema, because it ultimately gives rise to both the latter (p. 535). It may produce extreme cyanosis during an attack.

Cardiac causes for extreme cyanosis include any of the conditions which lead to *chronic failure of the right side of the heart.* These may be classed into one or other of four main groups, namely : primary *valvular disease of the heart* ; affection of the *muscle of the heart* or pericardium ; failure of the heart as the result of *chronic lung lesions,* especially emphysema, bronchitis, fibroid lung and bronchiectasis ; and cardiac failure when the heart is unable to maintain the *high blood-pressure* due to granular kidney or arteriosclerosis. When a late stage in the failure of compensation has been reached, it is often difficult to determine whether the primary condition is kidney, heart, lungs, or arteries ; the differential diagnosis between these is considered on page 14.

Cyanosis due to *splenomegalic polycythæmia* (*Plate XXIX*, p. 634) is slowly progressive, and the diagnosis is arrived at by finding in the patient a big spleen with POLYCYTHÆMIA (p. 532), and no other very definite lesion.

Cyanosis due to *inspissation of the blood* as the result of loss of fluid from the tissues in fevers, such as cholera, dysentery, yellow fever or typhus, is a late symptom in a disease that will generally have been diagnosed upon other grounds.

Methæmoglobinæmia and *sulph-hæmoglobinæmia* are diseases which have been grouped together under the term *enterogenous cyanosis.* Both are exceedingly rare. The tint of the skin by itself suggests the diagnosis, being altogether different from that of ordinary cyanosis, and yet not to be mistaken for pigmentary affections such as Addison's disease, argyria, ochronosis, or hæmochromatosis. There is no polycythæmia. The diagnosis is established by spectroscopic examination of the patient's blood, a suitably diluted specimen exhibiting a well-defined absorption band in the red (*Fig.* 35, p. 80) in addition to the two bands of oxyhæmoglobin between the D and E lines (*Fig.* 30, p. 80) ; the distinction between sulph-hæmoglobin and methæmoglobin is not easy except in the hands of experts in blood chemistry and spectroscopy. Some cases arise without any obvious external cause, and are to be distinguished from those in which the blood-changes are directly attributable to the effect of taking chlorate of potash, aniline derivatives, and possibly other drugs.

Herbert French.

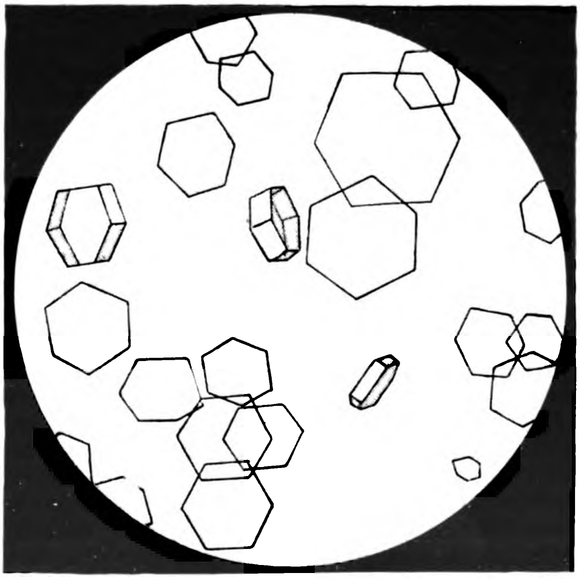


Fig. 72.—Cystin in crystals, as seen under the high power of the microscope : colourless flat hexagonal plates.

CYSTINURIA is the term used to denote the presence of cystin $(C_3H_6NSO_2)_2$ in the urine. The latter is usually pale, turbid, and oily in appearance when passed, slightly acid in reaction, with an aromatic odour resembling sweet-briar ; after standing, alkaline decomposition leads to the formation of sulphuretted hydrogen and a change in colour from yellow to green. The cystin forms a light-yellowish deposit, which consists of colourless microscopic hexagonal plates (*Fig.* 72). The condition is hereditary, and merely indicates a peculiarity of metabolism. The crystals have occasionally given rise to calculi, which are of a light fawn colour when first passed or removed, changing to green when they are exposed to the air. Cystin is not dissolved on heating the urine or by adding acetic acid, but it is by mineral acids and by ammonia ; from the latter it can be recovered by evaporation : a chemical test that has been recommended is to boil some urine with acetate of lead and caustic potash ; if cystin be present, a dark precipitate should form, as the result of the formation of lead sulphide. The best evidence of the condition, however, is the discovery of the typical crystals in the urine microscopically. *Herbert French.*

DEAD FINGERS.—Most individuals are familiar with dead fingers arising in perfectly normal persons who have spent more than the usual length of time in a swimming bath or in the sea; sometimes all the fingers of both hands will go absolutely white under these circumstances; even the whole hand may go dead-white, but more often it is the fingers only. The toes may be affected in a similar way.

Very similar deadness of the fingers results from exposure to cold on land, though the amount of cold required on shore is much greater than that which produces dead fingers in the water. The degree of cold required to produce this deadness of the fingers is much greater in the case of some individuals than in that of others, and the more inured the individual is, as the result perhaps of his occupation or other circumstances, the less easily do his fingers go dead with cold. This being so, it becomes a difficult point to decide just where deadness of the fingers ceases to be a physiological phenomenon and begins to be evidence of a pathological change. At the other end of the chain one has *Raynaud's disease*, which is one of the most characteristic of maladies, the patient's fingers going dead on the least exposure to cold, and sometimes often in quite warm weather. This phase of local syncope often passes on quickly to one of local asphyxia, in which the fingers and generally also the toes, from being white, go more or less purple or even quite black (*Fig.* 125, p. 256) and remain in this deeply cyanotic state for hours, days, or even weeks, unless artificial measures are resorted to to restore the circulation. In the most severe cases some portions of the affected tissues fail to recover their circulation properly, and die in patches, with the result that indolent ulcers develop, healing slowly to form depressed scars, and thus simulating to a minor extent the effects of frost-bite. Even extensive gangrene and loss of fingers results sometimes.

A very similar condition in which dead fingers may be a symptom results from *ergotism* (p. 259); and deadness of the fingers may be one of the phenomena of *pellagra* (p. 225), although here erythema is commoner than acute pallor. Fortunately both pellagra and ergotism are exceedingly rare in this country.

Between the physiological dead fingers of exposure in cold water or to cold atmospheres, and the pathological deadness resulting from Raynaud's disease as the result of exposure to temperatures which ought not to cause deadness of the digits in normal persons, one meets with varying degrees of precisely similar changes to which it is difficult to give an exact name. For instance, an apparently healthy individual complains that whenever he is getting up on a winter's morning he finds one or other of his fingers, generally a ring or little finger, goes dead and white, and it is not until he comes down to breakfast and gets into a warm room with a fire that the circulation becomes restored in it; what name is one to give to this? There is no generalized syncope of all the fingers such as one meets with in Raynaud's disease; and yet the patient suffers from his dead fingers without any cause which should be adequate. The complaint is fairly common; generally it is no indication of disease. Four things in particular need to be thought of, however, before the trouble is put into the category of personal idiosyncrasy, namely: (1) *Cervical rib;* (2) *Arteriosclerosis;* (3) *Occupation;* (4) *Blue-brain.*

Deadness of the ring or little fingers may be one of the earliest symptoms in the case of a person who has a *cervical rib* (*Fig.* 186, p. 443). Later, more generalized neurotic symptoms in the arm and hand may be expected, or even atrophy of the muscles supplied from the ulnar part of the brachial plexus. Although the rib dates from birth, it is remarkable how it often produces no symptoms until adult life is reached; it may produce no symptoms at all even then; when it does so the patient's attention is seldom drawn directly to the neck, but nearly always to something being the matter with the hand or forearm, especially the ulnar aspect of the latter and the little and ring fingers. If one realizes that the cervical rib or the fibrous band which joins the end of a buttress cervical rib (*Fig.* 187, p. 443) to the first rib, is liable to interfere with the lower trunks of the brachial plexus, one can imagine the various vasomotor and other nervous symptoms that may result; and if the possibility occurs to one, the diagnosis is established by means of the *x*-rays. Only when in place of any bony rib there is but a fibrous cord representing it will the *x*-rays fail to show either the entire rib, or more commonly perhaps a stump representing the vertebral end of such a rib, sufficient nevertheless to indicate the cause of the nerve symptoms in the hand and arm.

Arteriosclerosis or *atheroma*, or both, may involve the vessels supplying the hands and produce in the latter various symptoms of deficient circulation, including dead fingers. The patient will generally be past middle life, and as a rule there will be other indications of arterial degeneration, especially raised blood-pressure, though when atheroma rather than arteriosclerosis is the cause the arterial affection may be extensive though the blood pressure is not raised. The condition in the arms and hands comes on as a rule spasmodically, or in paroxysms when the arms and hands are used, and the remarks made on page 440 in regard to intermittent claudication apply here just as they do in the case of the leg. Dead fingers from this cause, however, are not met with frequently.

Occupation as a cause for dead fingers is familiar in two classes of persons in particular, namely first, those whose hands are immersed for many hours a day in waters of different temperature, especially if there are chemical ingredients such as carbonate of soda in the waters. Dead fingers are in this way one of the troubles which washerwomen are apt to suffer from (p. 444). The nature of the patient's employment may suggest this cause if inquiry is made as to exactly how the particular individual carries on his work. The other group of persons who are liable to develop dead fingers in one hand or the other as the result of their occupation, are those who carry heavy loads upon one shoulder in such a way as to depress that shoulder and push the head far over towards the opposite side. Apparently what happens is that the pushing asunder as it were of the shoulder and the neck throws much strain upon the fibres of the brachial plexus, and in some individuals this strain leads to degenerative changes which extend down the nerve of the arm into the hand. Pains may be the most prominent result, and these pains are generally most severe in the region of the shoulder and the upper arm, especially in the parts supplied by the circumflex nerve. In other cases, besides the pain, or without pain, muscular atrophy results. In a few instances vasomotor phenomena predominate, and dead fingers or even a condition similar to that of Raynaud's disease has resulted. That occupation is the probable cause will be suggested by the symptoms being so much more pronounced in one hand than in the other, for it very rarely happens that the man will carry weights first on one shoulder and then upon the other, so as to affect both brachial plexuses alike.

Blue-brain is a descriptive term, coined by Sir James Goodhart to cover a very extensive class of case in which all sorts of peripheral phenomena of a functional type have their root, in his opinion, not in a peripheral cause but in a central one; and as the individuals generally have what is called a poor circulation, with a tendency to blueness of the ears and hands, a liability to chilblains and other phenomena of that kind, he considers that they also have a corresponding tendency to poorness of the circulation in the cerebral centres; just as they have blue extremities, so they have, as he says 'blue-brain.' The patients are not all women, though the majority are; they have aches here and pains there; the abdominal aorta is often unduly pulsatile; the right kidney is often movable; there is suffering at the monthly periods; the knee-jerks are exaggerated; the patients are of the nervous, neurotic, neurasthenic, or even actually hypochondriacal type. Amongst the many symptoms that they may complain of, deadness of the fingers on the slightest provocation may be one; the condition may then simulate Raynaud's disease, and it is a question whether in Raynaud's disease itself the vasomotor anomaly is not central rather than peripheral. Every practitioner has met with dead fingers in patients for whom they can recognize at once that the term 'blue-brain' fits as an appropriate label; for a full description of the types of case in question he should read Sir James Goodhart's original article upon the subject. *Herbert French.*

DEAFNESS.—This is the most constant symptom of disease of the ear. It may be present in one or both ears, and may vary from a slight deficiency, which may be unnoticed by the patient, to a complete loss of hearing. The causes of defective hearing are many. In some cases it can be easily relieved; in others the prognosis may be absolutely hopeless.

The organ of hearing consists of two main parts. The first is a conducting portion consisting of the external auditory meatus, tympanum, drum, and ossicles—the function of which is to collect the sound waves and transmit the vibrations to the endolymph of the internal ear. The second portion contains the labyrinth—cochlea, vestibule, and semicircular canals—in which are situated the terminations of the auditory nerve. Deafness may be caused by a lesion either of the conducting portion of the auditory apparatus, or of the internal ear, which contains the receptive mechanism. The latter—labyrinthine or nerve deafness—is the more serious and usually the more severe, but the former is much the commoner. Rarely, deafness may be due to some disease of the auditory nerve or to some tumour of the brain involving the fibres of the nerve in their intracerebral course.

Tests for Hearing.—In the examination of a deaf patient, a careful investigation of the sense of hearing is necessary: (*a*) To estimate the severity of the deafness; (*b*) To ascertain whether the lesion is situated in the conducting apparatus, or in the labyrinth or auditory nerve. Before carrying out these tests it is well to examine the external auditory meatus with a speculum, to make sure that the deafness is not due to the presence of a plug of cerumen, in which case elaborate hearing tests are unnecessary. The following are the tests usually applied:—

1. *The Whispered Voice Test.*—This consists in noting the distance at which whispered words are heard. Vowel sounds are usually heard better than consonants. The examiner must cultivate a whisper of uniform intensity, and the patient's eyes should be covered to avoid the possibility of 'lip reading.' Each ear must be tested separately, the other external auditory meatus being covered by a finger.

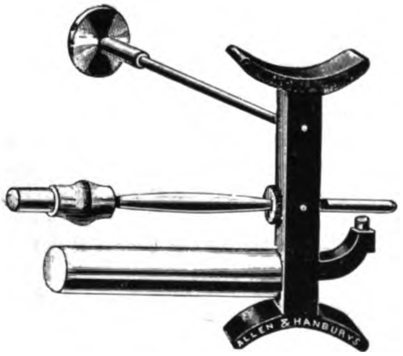


Fig. 73.—Politzer's acoumeter.

2. *The Watch Test.*—Here the distance is measured at which the ticking of a watch is heard. The same precautions must be taken as in the voice test. The observer must first measure the distance at which it can be heard by a normal person. Suppose this to be 30 in., and the patient hears it at a distance of 12 in.; the patient's hearing is then described as $\frac{12}{30}$. Instead of a watch, Politzer's acoumeter (*Fig.* 73), an instrument producing a uniform tapping sound, may be used.

The results obtained by these tests by no means always coincide. Sometimes the whispered voice may be heard remarkably well while the watch is almost inaudible. This is more likely to be the case when the onset of the deafness is late in life. More rarely the watch is heard more easily than the voice.

3. *Tuning-fork Tests* are of the greatest importance, since it is chiefly by these that labyrinthine or nerve deafness can be distinguished from deafness due to a lesion of the external or middle ear. In the latter case the sound waves are obstructed on their way to the receptive apparatus, and cannot be heard when the fork is near to, but not in contact with, the ear; whilst if the base of the fork is applied to the mastoid process, forehead, or chin, the vibrations are heard readily, because they are now conveyed to the normal receptive mechanism directly through the bone. In nerve or labyrinthine deafness, on the other hand, though the vibrations are transmitted by the bone, the sound is heard poorly or not at all, for the receptive apparatus is at fault, and is unable to respond properly to the stimulus of the sound waves, whether they reach it viâ the external and middle ear, or through the bone.

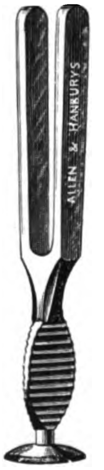


Fig. 74.—Tuning-fork with foot-piece.

The tuning-fork used should be one which vibrates 256 times per second (C^1). It should have a flat foot-piece (*Fig.* 74), so that it can be applied conveniently to the bone, and it may with advantage be fitted with a contrivance to prevent the occurrence of overtones. In addition, tuning-forks vibrating 64 times per second and 1024 times per second should be at hand, for testing the perception for high and low tones. In an elaborate investigation, still higher pitched tuning-forks may be necessary. The following are special tests used in testing bone conduction in a deaf patient:—

1. *Rinne's Test.*—The tuning-fork is struck lightly, and the flat foot-piece is held steadily against the mastoid process. Directly the patient ceases to hear the sound, he

raises his hand, and the fork is then held close to the external auditory meatus. If the sound is heard again, the result is positive; if it is inaudible, the result is negative. The test may also be carried out by holding the fork opposite the external auditory meatus first, and then, when it is no longer audible, applying it to the mastoid. A useful modification of this test is for the examiner to wait until the fork is no longer heard by the patient through the mastoid, and then to transfer it to his own mastoid. In this way the bone conduction of the patient is compared with the bone conduction of a normal individual.

2. *Weber's Test.*—This is especially useful in unilateral deafness. The vibrating fork is applied by the flat foot-piece to the middle of the forehead. The patient is then asked in which ear the sound is heard best. If the deafness is in the external or middle ear, the sound will be best heard on the deaf side (positive); if due to a lesion of the internal ear or auditory nerve, it will be heard in the good ear (negative). Great care has to be exercised in this test to get the correct reply from the patient, as there is often unwillingness to admit hearing in the affected ear.

3. *Gellé's Test.*—The air-pressure is increased in the external auditory meatus by means of a Siegle's speculum. The vibrating fork is then applied to the mastoid, or to the middle of the forehead. In a normal person, bone conduction is diminished. When it is unaffected it is generally considered that the foot of the stapes is fixed.

The hearing of high or low tones is ascertained by using tuning-forks of a rapid or low rate of vibration. Galton's whistle (*Fig.* 75), which produces very high notes, is also used for this purpose. By means of this instrument a note as high as 50,000 vibrations per second can be produced. If notes of more than 20,000 or 25,000 vibrations are not heard, the auditory nerve is probably affected.

Fig. 75—Galton's whistle.

To sum up, labyrinthine deafness is indicated when bone conduction is diminished markedly, i.e., when Rinne's test is positive and Weber's is negative. Generally speaking, in this form of deafness the perception of high-pitched sounds is diminished. It must, however, be remembered that in old people the perception of high notes is generally diminished considerably without any affection of the nerve.

Deafness due to some error in the conducting apparatus is indicated when bone conduction is good, i.e., when Rinne's test is negative and Weber's positive. There is also likely to be poor perception of low-pitched notes.

In carrying out these tests, however, it must be remembered that, in a patient over fifty, bone conduction is normally diminished, so that the tests are often inconclusive in an elderly patient.

Deafness due to a Lesion of the Sound-conducting Apparatus.—When this is the case, either the external or the middle ear may be at fault. Examination with the speculum will readily reveal the presence of a *plug of cerumen*, *polypi*, or a *foreign body*, such as a mass of wool, which is not infrequently inserted and forgotten by the patient.

The cause of middle-ear deafness will be diagnosed by considering other symptoms which may be present, such as pain and tinnitus, together with an examination of the tympanic membrane, and of the nose and nasopharynx. Deafness is more or less marked in all *inflammatory diseases of the middle ear*, *acute* or *chronic*, *suppurative* or *non-suppurative*. It must be remembered that there is not necessarily a correspondence between the intensity of the deafness and the condition of the membrane. The latter may be destroyed and hearing may remain fairly good, while with a small perforation, or in chronic non-suppurative otitis media, with but little alteration in the appearance of the membrane, the deafness may be profound.

Catarrh of the Eustachian tube, or obstruction to this passage by the presence of *adenoids* or *enlarged tonsils*, is a common cause of deafness, especially in children.

In some cases of middle-ear deafness, especially in *otosclerosis*, where the foot-plate of the stapes is fixed, the patient may hear ordinary speech better in a noisy place than in a quiet room. This is known as *paracusis Willisii*. It is generally explained by supposing that the more extensive vibrations caused by the loud noise loosen the joints between the ossicles, which are otherwise abnormally stiff.

In disease of the conducting apparatus, the patient, though deaf, not infrequently hears his own voice very loudly, and also noises in the nasopharynx such as occur on swallowing. This is known as *autophonia.*

Hyperæsthesia acustica is a term applied when sounds produce an actual painful sensation in the ear. It may be present in acute inflammation of the middle ear, fevers, and migraine.

Nerve or **Labyrinthine Deafness** may be due to a *lesion of the auditory nerve* itself, which may be involved in a *growth of the temporal bone,* or may show degenerative changes in *tabes.* It may also result from a definite intracranial lesion such as a *tumour of the mid-brain* or *pons.* A diagnosis in these cases will be made from the coexistence of other nervous symptoms associated with cerebral tumour. In labyrinthine deafness the following actual pathological changes have been found: (1) Degenerative changes in the organ of Corti; (2) Hæmorrhage; (3) Organized inflammatory products; (4) Rise in pressure in the endolymph.

The following are the chief causes of labyrinthine deafness:—

1. Extension from disease of the middle ear, suppurative (pyo-labyrinthitis) or non-suppurative (occasionally in otosclerosis).

2. Apoplectic deafness or Menière's disease, which may be due to hæmorrhage or a sudden rise of intracranial pressure.

3. Following the specific infectious fevers, especially mumps. but also influenza, typhoid, measles, scarlet fever, and others.

4. Syphilis. In the acquired disease, deafness may occur at almost any stage. The onset is usually sudden, the trouble is usually unilateral, and may have all the characters of Menière's disease. In congenital syphilis the deafness usually begins between the ninth and sixteenth years. Eustachian obstruction and retracted membranes are frequently present, but the deafness progresses and is labyrinthine in character. Other signs of congenital syphilis will be present to assist in the diagnosis.

Deafness may follow an injection of *salvarsan.* It may appear after an interval of several days, or as long as three months. The short interval is usually after an intravenous injection, the longer when the drug has been injected into the muscles. The deafness is more or less absolute and has the characters of nerve deafness. By some this is regarded as due to the drug; others regard the lesion as due to the liberation of a large quantity of endotoxin consequent upon the destruction of the spirochætes.

5. In Bright's disease, leukæmia, pernicious and other anæmias. A hæmorrhage is frequently the cause of the trouble here.

6. Certain drugs cause transient deafness of labyrinthine character; notably quinine, and sodium salicylate; possibly alcohol and tobacco. Mercury and lead also are stated to cause deafness sometimes.

7. Traumatic. Labyrinthine deafness may follow blows, falls, or fracture of the base of the skull.

8. Occupations, such as caisson workers, or workers in a continuous loud noise (boiler-makers' deafness).

9. Meningitis, especially cerebrospinal meningitis; and occasionally in epilepsy.

Deafness may also occur in hysterical individuals. This may usually be recognized by the manner and aspect of the patient, and by the absence of abnormal physical signs on examination.

Lastly, it must be remembered that deafness may be complained of by a malingerer; the fraud is usually exposed by contradictory replies to hearing tests with the eyes bandaged, or by speaking into the chest-piece of a binaural stethoscope with the tube to the sound ear plugged with wool. The probability is that the patient will say he hears words spoken into the stethoscope, but on removing this and covering the sound ear with the finger, he will say that he hears nothing. *Philip Turner.*

DEFORMITY OF THE CHEST.—In the differential diagnosis of alterations in the form of the chest, it must be remembered that many slight deviations from its typical form are not produced by disease. A long narrow chest (*alar chest*), or one flattened anteriorly (*flat chest*) is often found in persons predisposed to phthisis; but these also occur in individuals who are never affected by this disease. A long neck and sloping shoulders are also associated with this condition, while a short, thick neck with high shoulders is found in persons subject to apoplexy. The alterations in the form of the chest which may result from disease may be considered under the following headings :—

(1). *Deformities the result of rickets* ;

(2). *General changes* in the form of the chest : (*a*) The barrel-shaped, (*b*) Unilateral enlargement, (*c*) Unilateral shrinking ;

(3). *Local changes* : (*a*) Bulging, (*b*) Retraction.

Rickets.—The following deformities of the chest in an infant are due to rickets :—The chest is somewhat pear-shaped on transverse section, and a long vertical groove is often seen on each side of the sternum. Beading of the sternal ends of the ribs takes place, giving rise to the rickety rosary. The pigeon chest, in which the ribs are flattened on each side in front, so that the sternum becomes unusually prominent, making the chest appear somewhat triangular on transverse section, is always due to rickets (*Fig.* 78). *Harrison's sulcus*, a horizontal groove in the lower part of the rickety chest, is due to the sinking in of the ribs above the attachment of the diaphragm. This groove is exaggerated because the lower ribs are pushed out by the increase in size of the abdominal viscera. All these deformities are associated with other signs of rickets in the child, which make the diagnosis easy.

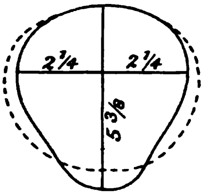


Fig. 76.—Rickety chest—child, aged 15 months.

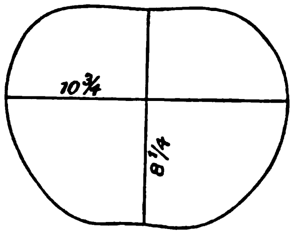


Fig. 77.—Normal adult chest.

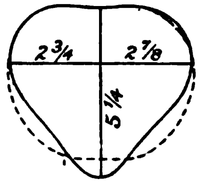


Fig. 78. —Pigeon chest—child, aged 14 months.

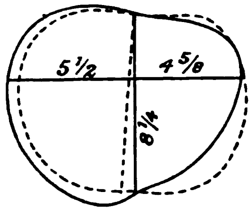


Fig. 79.—Fibrosis of the left lung—man, aged 30 years.

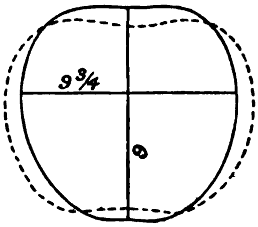


Fig. 80.—Emphysematous chest.

CYRTOMETRIC TRACINGS OF VARIOUS FORMS OF CHEST.

Transverse sections of various forms of chest at the level of the sterno-xiphoid articulation, reduced from cyrtometric tracings. The dotted lines indicate the natural shape at the same age. The figures represent the measurements in inches. (Sawyer's *Physical Signs*, 1908.)

General Changes.—(*a*). The *Barrel-shaped Chest* is found in patients suffering from *pulmonary emphysema* (*Fig.* 80). The chest is enlarged in all directions and gives the appearance which is assumed by the normal chest only after deep inspiration. The antero-posterior diameter is greatly increased. The shoulders are higher and squarer than in health, the intercostal spaces are enlarged and bulging, the dorsal curve of the spine exaggerated. The movements of the chest during respiration are extremely restricted ; there is elevation of the chest as a whole during inspiration, but very little real expansion. The neck appears abnormally short. The apex beat of the heart cannot be felt. On percussion the note over the lungs is hyper-resonant, the cardiac dullness is greatly diminished and often obliterated, and the upper level of the hepatic dullness is lowered. The breath-sounds upon auscultation are enfeebled, and expiration is markedly prolonged. If bronchitis be present also, adventitious sounds are heard, especially sonorous and sibilant rhonchi and coarse bubbling râles. The heart-sounds are difficult to hear.

Whenever any loss of symmetry in the two sides of the chest is found, the vertebral column must be examined carefully, as the alteration may be due to spinal curvature.

(*b*). *A Unilateral Enlargement* of the chest can be produced by an extensive *pleuritic effusion*, a large *empyema*, *pneumothorax*, and when an *intrathoracic tumour* affects the greater part of one side of the chest. The cause of the enlargement is ascertained by the physical examination ; thus with pleuritic effusion, either serous or purulent the movements of the affected side during respiration are restricted, while those of the opposite side are exaggerated ; dullness is found over the effusion, while above it the note is usually of higher pitch than normal, and often skodaic ; vocal fremitus, breath-sounds, and voice-sounds are diminished or absent over the dull area. At the upper level of the fluid ægophony may be present, and the breath-sounds frequently tubular. The presence of fluid is further confirmed by finding the heart pushed over to the opposite side, and the liver depressed when the right pleura is involved. When a *pneumothorax* is present, there is usually a history of a sudden onset, accompanied by a severe pain in the chest ; the affected side does not move as freely as the other with respiration ; the heart is displaced towards the opposite side, and vocal fremitus, breath-, and voice-sounds are diminished or absent, though the affected side of the chest is fully resonant ; if serum or pus be present in addition to air, the note is dull or greatly impaired at the base of the lung, with hyper-resonance but absence of breath-sounds above. When much fluid is present, the note changes considerably with the position of the patient. The metallic tinkling of Laennec is sometimes heard over a pneumothorax ; coughing is generally required for its production ; it resembles the sound which occurs when " a drop of water falls on the surface of a fluid contained in a half-filled decanter." The bell sound or ' bruit d'airain ' is very characteristic of a pneumothorax ; to hear it, auscultation is performed over a portion of the pneumothorax, and a coin placed on another portion is struck with a second coin ; the sound has a ringing metallic quality like that of the tinkling of a small bell, or like the ring that accompanies hammering upon a blacksmith's anvil. Hippocratic succussion may also be obtained when the observer's ear is applied to the chest while the patient's body is shaken or jolted.

(*c*). *Shrinking of the whole of one side of the chest* is due to contraction of one lung, either as the result of a previous compression by a large pleuritic effusion, and especially by an empyema, or on account of *fibrosis of the lung* (*Fig.* 79). The history of the patient often indicates the cause of the contraction of the lung ; a large effusion may have been aspirated, or an empyema may have been drained by surgical means, leaving the scar of the operation. In other cases the empyema may have burst into the lung, and there may be a history of a large amount of pus having been expectorated. With fibrosis of the lung the affected side is retracted and shrunken, the intercostal spaces are very narrow, and the ribs may even overlap. The shoulder is lower on the affected side, and the vertebral column is deviated towards the diseased lung. The heart is drawn over to the affected side, in which there is very little movement during respiration. If the left lung be affected, the heart will be less covered by lung than normally, and so there may be a large area over which cardiac pulsation is visible. The note over the contracted lung is impaired, while on the opposite side it is hyper-resonant. The breath-sounds are deficient or absent, and may be tubular or cavernous, while at the base there may be numerous coarse bubbling râles, especially if there is bronchiectasis. Vocal fremitus may be decreased or exaggerated. The expectoration is generally copious, semi-purulent, and often fœtid. There is often marked clubbing of the tips of the fingers.

Local Changes.—(*a*) *The cause of bulging of any portion of the chest wall* may be difficult of diagnosis, though sometimes it is obvious, as when an empyema points externally ; even this is sometimes mistaken for a localized abscess of the chest wall, unless a careful examination reveals the sign of fluid within the chest. In pulmonary emphysema, bulging is often present in the supraclavicular and infraclavicular regions. Bulging may also be due to an *intrathoracic tumour*, to an *aneurysm of the aorta*, or to a *tumour* or *abscess of the chest wall*. The most common situation on the chest wall for an aneurysmal swelling is to the right of the sternum in the first, second, and third intercostal spaces ; it may erode the upper part of the sternum and so produce a swelling there, while in rare instances it may produce a prominence to the left of the sternum ; a bulging to the left of the vertebral column may be due to an aneurysm of the descending thoracic aorta. The expansile character of the pulsation suggests the diagnosis. A tumour or abscess of the chest wall may occur in any situation. The præcordia becomes prominent in children in cases of *pericardial effusion*, or when the heart is enlarged; the situation of the prominence indicates its cardiac origin. An *enlargement of the liver* (p. 366) may also produce a prominence of the ribs under which it lies; a hepatic abscess, a subdiaphragmatic abscess, or an empyema, sometimes point over the lower part of the chest in front, while a psoas abscess may point over the lower ribs posteriorly. A prominence over the spinal column in the dorsal region may be due to *spinal caries*, or to a *malignant new growth of the spine*. An angular curvature of the spine is most commonly due to spinal caries, and any swelling which is associated with it may be produced by an abscess arising from the disease. Bulgings which give an impulse on coughing, and which wax and wane with respiration, suggest *hernia of the lung*, sometimes of considerable size in marasmic children suffering from whooping-cough, or in emaciated phthisical subjects with incessant cough.

(*b*). *Retraction* or localized shrinking of the chest wall occurs in any condition in which there is a portion of lung contracted by disease. When present over one or both apices of the lungs, as shown by retraction in the supra- and infraclavicular regions, it is nearly always due to *phthisis*. Unilateral shrinkage is also found with *fibroid conditions of the lungs* which are not tuberculous, or after the absorption of a pleuritic effusion or the removal of the pus from an empyema.

J. E. H. Sawyer.

DELIRIUM occurs in an overwhelmingly large proportion of cases in the course of some well-known disease, commonly pyrexial, and beyond the fact that the condition itself in such diseases is a symptom of somewhat serious import, nothing need be said as regards the diagnosis. There are, however, a few prognostic points worthy of mention in connection with such cases. Thus, in typhoid during the height of the fever, in measles, and in scarlet fever, the delirium is commonly in proportion to the pyrexia in its violence, and can usually be controlled by controlling the pyrexia, if necessary; in the later stages of typhoid, a low muttering delirium is of very serious import. In pneumonia, on the other hand, some degree of delirium is an almost constant factor, no matter what the temperature may be, and its significance depends upon the previous (alcoholic) history of the patient, upon the violence or severity, and duration or persistence of the mental phenomena. In rheumatic fever (unless due to salicylates, *vide infra*) delirium is of extremely grave significance, being commonly associated either with hyperpyrexia or with definite intracranial inflammation, neither of which is at all common. In influenza, too, it is a symptom causing great anxiety.

The difficulties of diagnosis arise chiefly when we are called to a case of delirium of which we have no previous knowledge, where in fact our services are sought primarily because the patient has ‘gone off his head and is talking nonsense,’ and we must consider to what different factors this may be due. The following table embraces causes ordinarily met with :—

Delirium due to intrinsic brain-cell changes or to pure nerve influences	Mania or lunacy in all its forms Pain, occasionally so severe as to produce it Shock, ditto
Delirium due to poison arising in the body (autogenetic)	Uræmia Diabetes. Anæmia. Polycythæmia ? Impending death from any cause.
Delirium due to microbic activities	Fevers of any kind, known and unknown as to their specific microbic origin
Delirium due to chemical agents introduced from without	Belladonna, hyoscyamus, and their allies; alcohol and other less common intoxicants, anæsthetizing substances, and hypnotics. Lead, and other metals occasionally. Exceptionally, it arises from almost any poison or drug, most typically, perhaps, from artificial salicylate of sodium.

As with all other tables of diagnostic problems, the difficulties are much greater on paper than in practice, for in almost every case there is some one overwhelming and outstanding fact in the history which settles the matter off-hand. It is well, however, to have some fixed order of procedure laid down, which may take the following lines :—

1. Make strict enquiries as to anything unusual having been taken or applied lately.

Medicines containing belladonna, drops put into the eyes, or some strange or unusual vegetable eaten, are the most likely things.

2. Enquire as to the recent health of the individual bearing on the urinary secretion, anæmia, etc.; also enquire about incidents that might have caused shock, and incidents suggestive of a simple idiopathic mental disorder.

3. Take the temperature; if materially raised it suggests some form of microbic influence, although in some, such as rabies, the pyrexia may not be great.

4. Note the pupils; if dilated and fixed, they suggest belladonna or perhaps alcohol —delirium tremens can hardly occur without a definite history of 'soaking,' or an accident; contracted and immovable pupils suggest uræmia; unequal pupils, general paralysis of the insane.

5. Test the urine; this will go far in clearing up urinary causes. Further details must be sought under the appropriate headings.

6. Note the skin, whether dry or sweating, whether flushed or pale; in poisoning by belladonna, etc., it is often dry and flushed; if connected with other dangerous chemical poisons, it is commonly pale and sweaty. *Fred. J. Smith.*

DIACETURIA—or the passage of diacetic acid in the urine—occurs under precisely similar circumstances to ACETONURIA (p. 3). The following is the usual clinical test for diacetic acid:—To one inch of urine in a test-tube add liquor ferri perchloridi (B.P.) drop by drop. For a moment a white precipitate of iron phosphate forms, and then, if aceto-acetic acid be present, the liquid becomes deep purple-red, this colour being discharged on warming. If carbolic acid, salol, or salicylates are being taken, the urine contains phenyl compounds which give a similar reaction with ferric chloride, but the colour due to these does not disappear on warming. *Herbert French.*

DIARRHŒA.—It is important to remember that diarrhœa is a symptom and not a disease in itself, and in every case one must try to discover what the underlying cause of the looseness of the bowels is. In order to do this it may be necessary, in addition to routine physical examination in the ordinary way, to employ one or all of the following special methods: (1) Digital examination of the rectum; (2) Inspection of the lower colon by the sigmoidoscope; (3) Investigation of gastric digestion by test meals (see p. 319); (4) Examination of the stools by the naked eye and by the microscope. Most of these methods require no special description, or have been dealt with in other articles, but some account must be given of the examination of the stools.

Various 'test-diets' for the investigation of the intestinal functions have been proposed, but it is sufficient to let the patient include the following articles in the dietary for about forty-eight hours before the stool is examined, viz: (1) Milk; (2) Eggs; (3) Meat in some form; (4) Farinaceous foods, e.g., bread, potatoes, rice; (5) Green vegetables and stewed fruit; (6) Fats, e.g., butter, bacon, fat, ham, etc. The choice and amount of the individual articles may be left to the patient's taste.

In order *to examine the stool*, a portion the size of a walnut should be rubbed up with normal saline solution to a fluid consistency, and examined with the naked eye against a dark background. Normally one sees a homogeneous fluid made up of very small dark-grey particles. In pathological conditions one may recognize mucus, pus, blood, parasites, the remains of connective tissue in the form of yellowish-white shreds, brown muscle fibres, and the residue of potatoes in the form of glossy granules.

For *microscopical examination* one prepares three specimens. The first is examined as it is; to the second one adds a few drops of 30 per cent acetic acid, and heats a little to dissolve fat; to the third is added a little iodine solution.

A normal stool shows in the first preparation a few muscle fibres, some yellow lumps of lime salts, and a few empty potato cells. In the second preparation, a few fatty crystals; in the third a very few violet-tinted starch grains. In pathological conditions one may find in the first preparation many well-preserved muscle fibres, numerous fat droplets and fatty crystals, and abundance of potato cells; in the acetic acid preparation, numerous masses of crystals of fatty acids; in the iodine preparation, an excess of starch.

In order *to test for Bile*, mix some of the stool with concentrated corrosive sublimate solution and allow to stand for twenty-four hours. Normally it turns red from the presence of urobilin; greenish particles show the presence of unaltered bilirubin; absence of green or red colouring shows that bile is not present at all.

Reaction of the Stool.—A drop of the stool prepared as above by rubbing up with water is applied with a glass rod to a piece of moistened litmus paper. The reaction can easily be seen on the other side of the paper. A normal stool is nearly neutral; marked alkalinity indicates putrefaction; acidity shows carbohydrate fermentation.

Test for 'Occult' Blood.—The patient must have eaten no red meat for two or three days. A portion of the stool the size of a hazel-nut is rubbed up with 2 c.c. of distilled water in a mortar and placed in a test tube. Add half its volume of glacial acetic acid, and shake. Then nearly fill the tube with ether, and reverse several times. To about one inch of the resulting yellow, translucent, ethereal solution, add: (*a*) a few drops of glacial acetic acid, (*b*) one inch of *freshly prepared* saturated solution of benzidin in rectified spirit, (*c*) one inch of liq. hydrog. perox. Shake, and pour a few drops on to a porcelain slab. If blood be present, a blue colour appears.

DIARRHŒA IN INFANCY AND EARLY CHILDHOOD.

1. **Acute.**—The acute diarrhœas of infancy are either dyspeptic or infective in origin. The infective diarrhœas are usually spoken of as 'summer' or 'epidemic' diarrhœa. It is often impossible to distinguish sharply between the simple dyspeptic and the infective variety, but it may be said that the greater the signs of toxæmia (collapse, sinking in of the fontanelle, inelasticity of the skin, etc.) the more likely is it that the case is one of infection. High body temperature and epidemic prevalence of the disease are also in favour of such a diagnosis. Dyspeptic diarrhœa may be due to mal-digestion of any of the constituents of milk. Examination of the stools may enable one to distinguish which constituent is at fault, thus :—

Stools containing white tough particles, insoluble in alcohol and ether mixture = casein indigestion.

Green slimy stools containing small granular masses soluble in alcohol and ether mixture = fat indigestion.

Frothy sour stools = sugar indigestion.

Green stools are of no special diagnostic value, as they merely indicate that the contents have been hurried unduly through the intestine.

If the stools contain visible blood and mucus, and are passed with much pain and straining, *acute colitis* may be diagnosed, but not until *intussusception* has been excluded (see Blood per Anum, p. 75).

2. **Chronic.**—Chronic diarrhœa in infancy may follow upon an acute infective diarrhœa or be dyspeptic from the outset. The history and a consideration of the points mentioned above will determine the diagnosis in most cases, but it must be remembered (1) That an intestinal catarrh set up by an infection may lead to mal-digestion and persistent chronic diarrhœa in consequence; and (2) That a dyspeptic diarrhœa predisposes to the development of intestinal infections. The two classes may therefore pass into each other and an exact differential diagnosis be impossible.

There is a special form of chronic diarrhœa in early life which follows a very prolonged course, and to which the term 'cœliac disease' or 'the cœliac affection' is applied. It usually starts in the second or third year of life, and is characterized by the passage of stools which are not very frequent but are bulky, pale, and extremely offensive, containing much undigested fat and free fatty acids. The abdomen is tumid and tympanitic, and the child wasted and stunted in growth and development. This form of diarrhœa is very apt to simulate abdominal tuberculosis, and indeed is usually diagnosed as such; but in abdominal tuberculosis enlarged glands or a rolled-up and thickened omentum can usually be felt, or there is ascites or evidence of tuberculosis elsewhere. Sometimes, however, a diagnosis is only possible after watching the progress of the case. If the stools in a case of chronic diarrhœa contain visible mucus and blood, and are passed with much straining, special involvement of the large bowel may be diagnosed (chronic colitis). The history will usually point to the preceding occurrence of an attack of acute colitis.

DIARRHŒA IN ADULTS.

1. **Acute.**—The history is of great importance. It may elicit some *indiscretion of diet* (the eating of unripe fruit, etc.), or the consumption of some toxic article of food (*ptomaine poisoning*) or irritant drug (e.g., arsenic). In such cases vomiting is often present as well. In toxic cases there is great depression, and a feeble and, perhaps, irregular pulse. If there be fever, one should think of an infective cause, such as typhoid fever, or dysentery. In the case of *typhoid*, enlargement of the spleen is an early confirmatory sign, but is sometimes absent; spots should also be looked for. The presence of leucopenia may be of help, and the pulse-rate is low in proportion to the temperature. The agglutination reaction is not usually obtainable until the end of the first week. In *dysentery* there will

be tenesmus, with blood and mucus in the motions. In the amœbic form, the *Amœba coli* may be found in the stools (see *Fig.* 25, p. 77). In the specific form, the blood serum agglutinates Shiga's bacillus. Similar symptoms to those of dysentery are produced by *acute colitis*, especially of the ulcerative form.

Appendicitis may begin with acute diarrhœa, and the possibility of this should be borne in mind.

In *pernicious anæmia*, *exophthalmic goitre*, and *Addison's disease*, periodic attacks of acute diarrhœa are apt to occur. The other characteristic signs and symptoms of these affections will be present. (See ANÆMIA, p. 24; and PIGMENTATION OF THE SKIN, p. 528).

Finally, it should be remembered that even although diarrhœa is due to a new growth in the bowel, it may begin acutely, and a rectal examination should never be omitted.

2. **Chronic.**—Chronic diarrhœa in the adult may be the result of several causes, of which the following are the chief :—

Impaired Gastric Digestion (gastrogenic diarrhœa).—The looseness tends to occur in bouts, with intervals of freedom. The stools contain fragments of connective tissue and show under the microscope an excess of unaltered muscle fibres. A test meal reveals absence or great diminution of gastric juice (achylia).

Impaired Pancreatic Digestion (pancreatic diarrhœa).—The stools are pale or white in colour, very offensive, and show, on cooling, solidified fat masses; microscopically they exhibit excess of fat globules and fatty acid crystals along with undigested muscle fibres and starch granules.

Local Conditions in the Colon :—

(*a*). *Fœcal Impaction* (paradoxical diarrhœa).—This variety is commonest in elderly persons. Rectal examination reveals retained fæces, and fæcal masses may perhaps be felt through the abdominal wall. A thorough evacuation arrests the discharges.

(*b*). *New Growth.*—There is nothing absolutely characteristic about this form of diarrhœa, but the motions are often explosive and tend to occur in the early morning. Blood may be present in the stools, but not always. Digital examination of the rectum or the use of the sigmoidoscope will reveal a growth. It should be noted specially that neither a sudden beginning of the symptoms nor the youth of the patient excludes the possibility of growth.

(*c*). *Chronic Catarrh of the Colon or Rectum.*—The diarrhœa in this variety tends to be in the early part of the day ('morning diarrhœa'), the stools are well-digested and may or may not show visible mucus. Examination with the sigmoidoscope will show a catarrhal condition of the mucous membrane if the disease affects the pelvic colon. In cases in which the chief seat of the affection is higher up, it may only be possible to arrive at a diagnosis by the method of exclusion.

(*d*). *Ulcerative Colitis.*—The stools are frequent, usually small, often passed with some straining, and contain visible mucus, blood, and shreds. The sigmoidoscope reveals ulceration of the mucous membrane. The ulceration may be dysenteric or non-dysenteric in nature, but the history will usually enable one to make the distinction.

Catarrh of the Small Intestine.—The stools are usually copious, fluid, free from visible mucus or blood, unless the colon be involved as well, and show under the microscope impaired digestion of all the food constituents and the presence of bile-stained particles of mucus. Sometimes the diagnosis can be arrived at only by exclusion.

If catarrh of the small intestine be diagnosed, one has to determine its cause. The chief things to think of are : cardiac disease or cirrhosis of the liver producing chronic venous stasis in the bowel; phthisis or other forms of tuberculosis; chronic nephritis; alcoholism and the ingestion of irritants (e.g., arsenic, antimony).

Lardaceous Disease is a rare cause of chronic diarrhœa nowadays, and is not likely to occur unless there be signs of waxy disease elsewhere, e.g., in the spleen, liver, or kidneys. There may be a history of prolonged suppuration or tertiary syphilis.

Tropical Diseases.—The two chief tropical diseases causing chronic diarrhœa are, besides chronic dysentery already mentioned, *sprue* and *hill diarrhœa.*

In sprue the pale, frothy, and copious stools are characteristic, besides the presence of a painful stomatitis involving the tongue and lining membrane of the mouth. It should always be thought of as a possibility in the case of a patient who has lived in the East.

Hill diarrhœa, which is closely allied to sprue, is met with chiefly in Europeans on their going to the hills after living in the tropical lowlands. The diarrhœa tends to occur chiefly in the early morning, the stools being copious, pale, and frothy. The diarrhœa is accompanied by much flatulence and distention.

Nervous Causes.—If all the above causes of a chronic or recurring diarrhœa can be excluded, one may be dealing with a case of *nervous diarrhœa*, which is characterized by a tendency for the bowels to act directly after a meal (lientery) or on excitement or under emotional influences. A good many cases of so-called 'morning diarrhœa' are of this type, though in many there is a catarrhal basis as well. The history, the presence of other evidences of nervous irritability, and the fact that the general health and nutrition are well maintained, all yield confirmatory evidence. Frequent action of the bowels may accompany *tabes dorsalis*, either in a late stage when sphincter trouble has arisen, or earlier in the form of *rectal crises* analogous to the more familiar gastric crises of this disease.

Robert Hutchison.

DIAZO-REACTION.—The diazo-reaction of Ehrlich is obtained in certain urines on testing them with the following solutions :—

(1) Sodium Nitrite	0·5 gram	(2) Sulphanilic Acid	0·5 gram
Distilled Water	100 c.c.	Hydrochloric Acid	0·5 c.c.
		Distilled Water	100 c.c.

A strong solution of ammonia is also required, and all should be freshly prepared. To a drachm of sulphanilic acid solution add a drop of sodium nitrite solution, mix with a drachm of the urine, and add ammonia to excess. A normal urine turns brownish-yellow ; when the reaction is positive the mixture turns deep red, and, most characteristic of all, the froth produced on shaking the test tube is rosy red.

It is often regarded merely as an obsolete test for typhoid fever ; but it occurs in many other conditions—it is an indication of abnormal protein metabolism, leading to the elimination of certain aromatic substances which react in this way to diazo compounds. The following are some of the conditions under which the diazo-reaction has proved positive :—Many fevers, such as diphtheria, erysipelas, measles, pneumonia, scarlet fever, typhoid, typhus ; cachectic states, such as advanced phthisis, cancer, cirrhosis, syphilis, malaria, grave anæmias ; and as the result of poisoning by certain drugs, such as chrysarobin, guaiacol, carbolic acid, or opium.

Clearly a reaction which occurs under so many different circumstances can have but a limited value. There are some who say that it has no value at all ; others, however, find it of clinical use in the following respects : (1) It is never normal ; (2) It is more constantly present in cases of typhoid than in any other fever, so that, other things being equal, the presence of the diazo-reaction may help in diagnosing typhoid fever, though the converse is not true ; (3) In cases of phthisis a positive diazo-reaction is a sign of ill-omen, whilst should the diazo-reaction disappear after it has been present, this is evidence of material improvement, even though the physical signs remain the same.

Herbert French.

DILATATION OF THE HEART.—(See ENLARGEMENT OF THE HEART p. 206.)

DILATATION OF THE STOMACH presents itself clinically under two totally different aspects : (1) *Acute* ; (2) *Chronic*.

Acute Dilatation of the Stomach is generally a serious complication, or often rather a fatal catastrophe, arising in the course of some other condition, especially :—

After operations, notably laparotomy, performed for whatever cause
After abdominal injury
In the course of acute fevers, especially lobar pneumonia
In the course of chronic heart failure, especially in bad cases of mitral stenosis.

The diagnosis is generally easy ; it is the relief of the acute dilatation that is so difficult. The blown-up, drummy abdomen, the constant effort to bring up wind, sometimes in vain, sometimes with copious and recurrent eructations, often with ominous hiccough, are familiar and much to be dreaded. Sometimes shortly before, sometimes just after, death, immense quantities of blackish brown or dull greenish brown fluid flow from the mouth and nostrils, and the wonder is how it can all be coming from one stomach. The dilatation itself is of the nature of acute paralysis of the gastric walls, and the final outflow of fluid

—which gushes out rather than is vomited—is caused by the pressure of the gas associated with it, and not by active contractions of the stomach musculature.

Chronic Dilatation of the Stomach is due to totally different causes, which may be divided into two main groups, namely :—

1. *Those associated with stenosis at or on either side of the pylorus due to* :—

Cicatricial fibrosis of an old simple gastric ulcer
Cicatricial fibrosis of an old duodenal ulcer
Adhesions around or near the pylorus, the result of former local peritonitis due to such causes as : Former gastric ulcer ; Former duodenal ulcer ; Gall-stones. In many cases adhesions are found without any ascertainable cause.

Carcinoma of the pylorus
Carcinoma of the duodenum
Carcinoma of the gall-bladder
Carcinoma of the head of the pancreas
Rarities, such as calcified retroperitoneal cyst ; hydatid cyst at the portal fissure ; huge renal tumour.

2. *Dilatation without obstruction* :—

Atony. Over-distention by gas or excess of food or drink.

In the consulting room the two most suggestive signs of dilatation of the stomach are : (1) A gastric succussion splash, audible or palpable over a much wider area than normal. The mere presence of succussion is not an indication of dilatation, for a normal stomach containing fluid and gas gives marked succussion. The point to determine is the area over which the succussion is heard ; and if it extends right across the epigastrium and down to the umbilicus, or below it, when the patient is lying down, dilatation is almost certain. (2) Visible gastric peristalsis over an unduly large area (p. 521). The most important symptom when there is pyloric stenosis is the vomiting at relatively long intervals of larger quantities of material than were consumed at the last meal, especially if remains of a meal taken the day before can be recognized in the vomit. A very important point to remember, however, is that even a marked degree of pyloric stenosis, with extensive dilatation, may be present in a patient who never vomits at all. This has been proved again and again by bismuth and *x*-ray examinations followed by operation. It is by the *x*-rays that the diagnosis is made best, especially by a series of examinations after the original bismuth meal. If the bismuth is taken at 11 a.m., none should be seen in the stomach at 6 p.m. Very often in these cases, however, the black shadow is still obvious in the stomach, even at 11 a.m. on the following day—after twenty-four hours, and in some instances for longer still. It is generally easy to see the active peristaltic waves of the stomach at the same time, and thus distinguish between the dilatations due to obstruction, and atonic dilatation in which the stomach wall has sagged down and remains motionless when seen with the *x*-rays after bismuth. Skiagraphy is infinitely superior to any other method of diagnosis in these cases, and is replacing diaphany, lavage, inflation, and gastric juice analyses wherever available. At the same time it is often possible to detect such difficulties as hourglass stomach, or to distinguish ulcer from carcinoma. Once dilatation from stenosis has been demonstrated in this way, operative measures are indicated, for medicinal treatment cannot cure the mechanical stasis. The further details of the diagnosis are arrived at by the surgeon ; even when the abdomen has been opened, however, it is often exceedingly difficult to decide whether a given hard mass at the pylorus is malignant, or due to inflammatory matting round an old simple ulcer, and it may remain in much doubt which of the two is present until one finds that the patient survives for years after his gastroenterostomy, and thus demonstrates that what was thought at the time to be a carcinoma must after all have been not malignant, but the result of inflammatory matting round a simple chronic ulcer. *Herbert French.*

DIPLOPIA, or double vision, may be either monocular or binocular ; that is to say, an object may be seen double with one eye, or single with each eye separately, and only double when both eyes are open. To distinguish between the two conditions it is necessary that each eye should be closed in turn. If with either eye the object is still seen double, the diplopia is monocular and due to that eye alone ; if, on the other hand, the object is seen double only when both eyes are open, the diplopia is binocular, and due to some disturbance of the balance of the two eyes.

Monocular Diplopia may be due to : (1) Dislocation of the lens ; (2) Incipient cataract ; (3) Double pupillary apertures ; (4) Low degrees of astigmatism.

In a case of monocular diplopia it is necessary to examine the eye by light reflected upon the pupil from an ophthalmoscope mirror in a dark room. Diplopia from a *dislocated* or *displaced lens* will only occur when the edge of the lens is in the pupil, some rays passing outside the lens direct to the macula, and other rays, passing through the edge of the lens, being deflected to a different part of the retina. In these circumstances the edge of the lens will be seen in the pupil as a dark crescentic opacity of unmistakable form and appearance. Other symptoms which may serve to confirm the diagnosis are increased or irregular depth of the anterior chamber (the space between the iris and the cornea), and tremor of the iris during movements of the eye.

Early *cataract* usually leads rather to the appearance of multiple images, than of two only, a candle or light being seen as five or six. This polyopia is due to the fact that the lens is broken up by cortical cracks and opacities into sectors of varying refractive power, very often set in slightly different planes. These cracks and sectors of the lens will be seen easily as black radial opacities on illumination by an ophthalmoscope mirror, or as opaque white striæ when the eye is illuminated from the front by a lens.

The presence of *two pupillary apertures* will be at once apparent on a careful examination of the eye. They may be congenital, or due to accident or operation. In cases of diplopia due to multiple pupillary apertures, the double vision is most evident when the object looked at is not in accurate focus.

Should none of the three conditions mentioned above be found, it is most likely that the diplopia is due to a *low error of refraction*. In this condition letters and test types are often seen accompanied by faint ‘ ghosts ’ placed either above or to the side of the real letters, and in some cases overlapping them. This cause of monocular diplopia can only be determined by a careful examination of the refraction of the eye. The diplopia is cured by the wearing of suitable glasses.

Binocular Diplopia may be either (1) Physiological or, (2) Pathological.

Physiological diplopia occurs unnoticed in all normal binocular vision. It is evident that as the two eyes view any given object from different standpoints, the retinal images must differ as do the two views taken by a stereoscopic camera. The diplopia is not apparent, however, as the two dissimilar images are combined by the higher visual centres of the brain to form a single solid conception of the object viewed. The amount of dis-similarity of the retinal images gives the impression of space and distance, near objects causing images more unlike than those formed by things remotely placed. The dis-similarity of the two retinal images in normal binocular vision, giving the idea of space, is termed in psychology ‘ disparateness ’ or ‘ disparation.’

When, however, owing to some failure in the centre which controls the mental fusion of the two ocular images, they are not combined, or when some disturbance of the accurately balanced muscular mechanism upsets the automatic fixation of both eyes upon the same object, pathological or obvious diplopia results.

Pathological Diplopia.—Before discussing the various forms and causes of this condition it is necessary to have a clear idea of the visual process of localizing objects in space —projection, or orientation.

In normal binocular vision, looking at an object means that both eyes are so turned that the image of the object looked at falls upon the central most acute area of the retina, the macula or yellow spot, in each eye, and objects other than that directly looked at form images upon the retina which are more or less peripheral. From our experience of such sensations and their locality on the retina we are able accurately to determine the relative positions of objects in space. The image of any object will always fall upon corresponding areas of the retinæ of the two eyes. These areas, though always corresponding, are not in the true sense of the word symmetrical. The image of an object to the right of the eyes falls upon the nasal side of the right and the temporal side of the left retina ; but the corresponding areas are in normal circumstances always stimulated simultaneously, and from these retinal images is derived the idea of the position of the object in space.

If the normal relative position of the two eyes is upset in any way the image of an object no longer falls upon two usually corresponding areas, erroneous ideas of projection are formed, with consequent diplopia, and it is from an examination of this diplopia that we can ascertain the displacement of the eye and its probable cause.

For example, ***Fig.*** 81 represents diagrammatically a condition in which the left eye is looking at or fixing the object O, while the right eye is pointing abnormally inwards—a convergent strabismus. In consequence of the abnormal position of the right eye, the image of the object O does not fall upon the yellow spot on the macula, *f*, but upon a point internal to it, ***a***. In ordinary circumstances, with proper fixation of the two eyes, any object whose image fell upon *a* would be to the right of the object O, hence under the existing abnormal conditions the right eye erroneously projects the object O to the position O^1, and a diplopia results in which the right of the two images seen belongs to the right eye, and the left to the left eye. This is termed a homonymous diplopia. ***Fig.*** 82 shows in a similar manner the formation of a crossed diplopia in a divergent squint or strabismus. These two figures illustrate the formation of a diplopia in lateral deviations of the eyes. A moment's consideration will show that deviation in a vertical or oblique plane will equally cause diplopia, owing to the disturbance of the normal corresponding areas of the two retinæ.

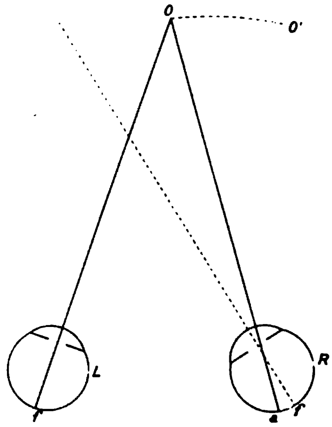


Fig. 81.—Homonymous double images.

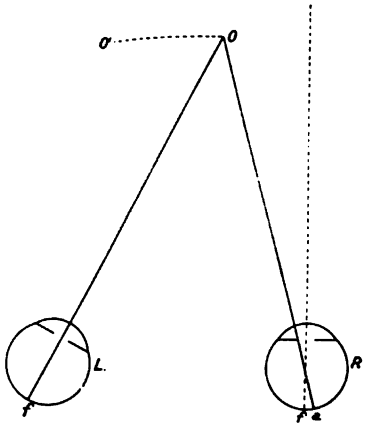


Fig. 82.—Crossed double images.

It will be seen from the figures that, in lateral deviations, a convergent squint causes homonymous, and a divergent squint crossed, diplopia. In ocular paralyses the diplopia will increase if the two eyes are carried in the direction of the usual action of the paralyzed muscle. As an example, ***Fig.*** 81 may be chosen as a diagrammatic representation of a paralysis of the right external rectus muscle. The more the eyes are turned to the right the greater will be the convergence, owing to the inability of the right eye to turn to the right to the same extent as the left ; the greater therefore will be the diplopia as the image of the object O falls farther and farther round on the nasal side of the right retina, the object being projected farther and farther to the right. It will also be seen from this consideration that in a case of diplopia from a muscular paralysis when the eyes are carried as far as possible in the direction of the usual action of the paralyzed muscle, the farthest displaced image always belongs to the paralyzed eye.

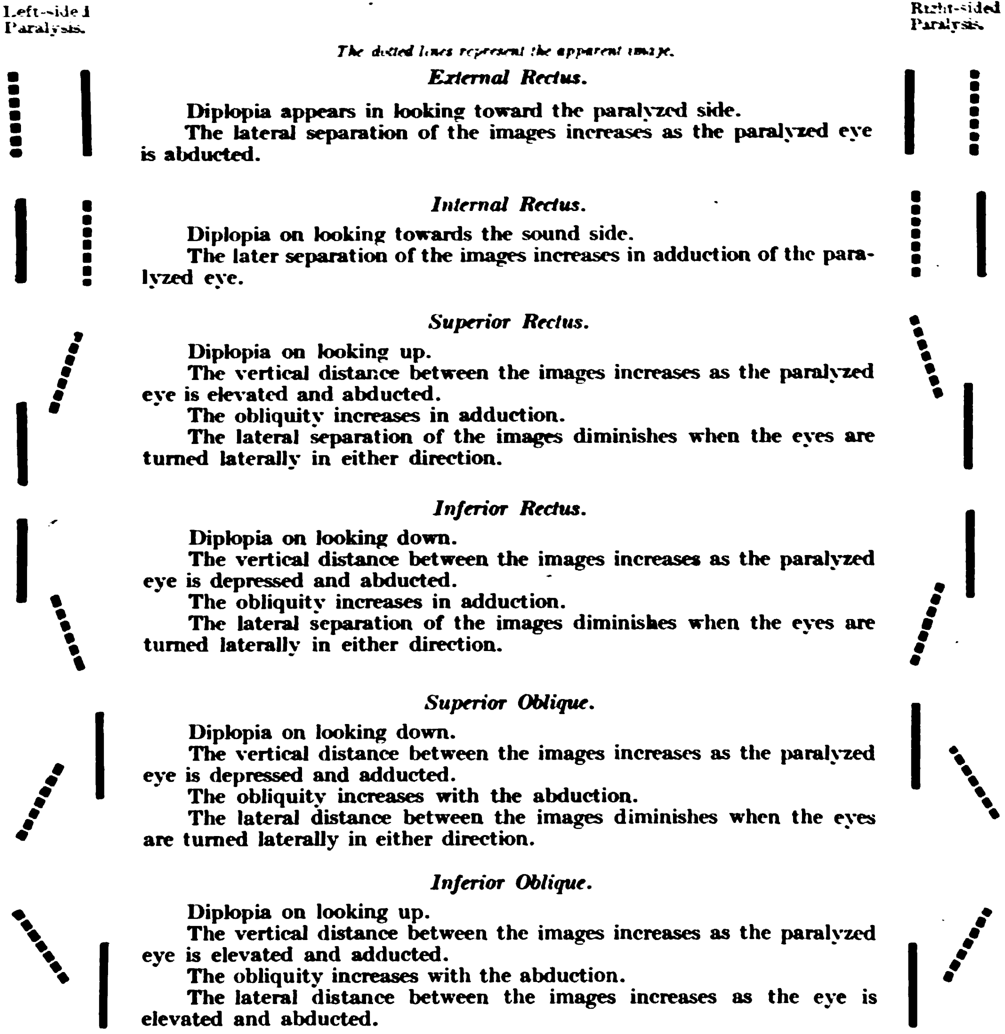
The two images are not equally distinct. That in the unaffected eye falls upon the macula and is seen most distinctly ; this is called the real image. That falling upon the retina of the affected eye is more peripheral, and therefore not so definite ; it is termed the false or apparent image.

With the above considerations in view, and with a knowledge of the individual actions of the ocular muscles, it is easy to elucidate cases of simple paralysis of one or more ocular muscles, but for convenience of reference the chart giving the position of the images in paralysis of the various ocular muscles is reproduced on the following page.

Binocular diplopia may be caused, as suggested above, by paralysis of ocular muscles, but it may also arise from the bodily displacement of one eye from *orbital growth, abscess,* or *hæmorrhage.* It may also occur after some operations for *tenotomy.*

Cases of displacement of the eye from local causes can usually be distinguished from those of ocular paralysis by the indeterminate character of the diplopia, which is accompanied by more or less fixation of the eyeball, and by proptosis.

Fig. 83.—To Illustrate the Behaviour of the Double Images in Paralysis of the Ocular Muscles.



The dotted lines represent the apparent image.

External Rectus.

Diplopia appears in looking toward the paralyzed side.

The lateral separation of the images increases as the paralyzed eye is abducted.

Internal Rectus.

Diplopia on looking towards the sound side.

The later separation of the images increases in adduction of the paralyzed eye.

Superior Rectus.

Diplopia on looking up.

The vertical distance between the images increases as the paralyzed eye is elevated and abducted.

The obliquity increases in adduction.

The lateral separation of the images diminishes when the eyes are turned laterally in either direction.

Inferior Rectus.

Diplopia on looking down.

The vertical distance between the images increases as the paralyzed eye is depressed and abducted.

The obliquity increases in adduction.

The lateral separation of the images diminishes when the eyes are turned laterally in either direction.

Superior Oblique.

Diplopia on looking down.

The vertical distance between the images increases as the paralyzed eye is depressed and adducted.

The obliquity increases with the abduction.

The lateral distance between the images diminishes when the eyes are turned laterally in either direction.

Inferior Oblique.

Diplopia on looking up.

The vertical distance between the images increases as the paralyzed eye is elevated and adducted.

The obliquity increases with the abduction.

The lateral distance between the images increases as the eye is elevated and abducted.

Isolated paralyses of individual ocular muscles or groups of muscles are nearly always nuclear in origin; basal growths rarely cause ocular paralyses of any extent on one side only, the affection sooner or later becoming bilateral.

In some rare cases of convergent or divergent squint with absence of binocular vision and good vision in each eye, there may be the power of alternate fixation with more or less evident diplopia. As a rule, however, the individual has the power of suppressing the image of the squinting eye, obtaining monocular vision. *Herbert L. Eason.*

DISCHARGE FROM THE EAR.—(See Otorrhœa, p. 421.)

DISCHARGE, NASAL.—A discharge from the nose may be acute, subacute, or chronic, and it may consist of clear fluid almost like water, of mucus, muco-pus, pus, food regurgitated through the nose, or blood. For the differential diagnosis of the causes of hæmorrhage from the nose, see EPISTAXIS, p. 220.

Regurgitation of Food through the Nose may be due to a congenital condition, especially *cleft palate* ; to acquired *perforation* of the palate, especially *syphilitic* ; to *post-diphtheritic paralysis* ; or to much rarer neuro-muscular lesions, such as *bulbar paralysis*, *pseudobulbar paralysis*, or *myasthenia gravis*, all of which are discussed elsewhere.

Serous, Mucous, and **Muco-Purulent Discharges** differ from each other chiefly in degree, for that which may begin as serous may later become muco-purulent and then purulent, as is seen during the course of a common cold. A watery discharge is sometimes spoken of as coryza, though for the latter to be typical there should at the same time be watering of the eyes ; it is generally acute in onset, and the diagnosis of its cause is not difficult as a rule. It may be due to the following different conditions :—

Common cold, early stage (*Micrococcus catarrhalis*)
Lachrymation
Hay fever (*coryza e feno*)
Measles
Iodism or bromism
Arsenic
Local irritants such as snuff, ammonia vapour, sulphur dioxide, chlorine, and other irritating gases
Fog
Some cases of spasmodic asthma
Some cases of trigeminal neuralgia
Neurosis

The differential diagnosis of these conditions needs little discussion, a careful inquiry into the circumstances of the case generally pointing to its nature at once. *Measles* probably presents the greatest difficulty, for the coryza precedes the macular eruption, and the patient, generally a child, may seem to be suffering merely from a severe cold, when in reality it is in the most infectious stage of measles. Examination of the buccal mucous membrane for Koplik's spots (*Plate VIII*) may sometimes serve to distinguish this malady as long as two days before the eruption appears. These spots are individually small, with a whitish centre the size of a pin's head, surrounded by a purplish red blush ; in many cases they are not single, but collected into groups of from two or three to thirty or more ; a common place to find them is on the inner aspect of the cheeks, in much the same position as that in which one expects to find brown pigmentation in Addison's disease ; but they should be looked for also on the gums, the inner aspects of the lips, and on the hard and soft palate.

The coryza resulting from *iodide* or *bromide of potassium* or from *arsenic* may be very severe, and the patient generally complains of constantly catching cold, when in reality the symptoms are due to the drug.

The term *influenza* is sometimes applied to severe febrile colds associated with running of the eyes and dripping at the nose, but it is often inaccurate to apply the term influenza here, for the symptoms are more often due to the *Micrococcus catarrhalis*. Bacteriological detection of the *Bacillus influenzæ* in the discharge is essential if influenza is to be diagnosed with accuracy.

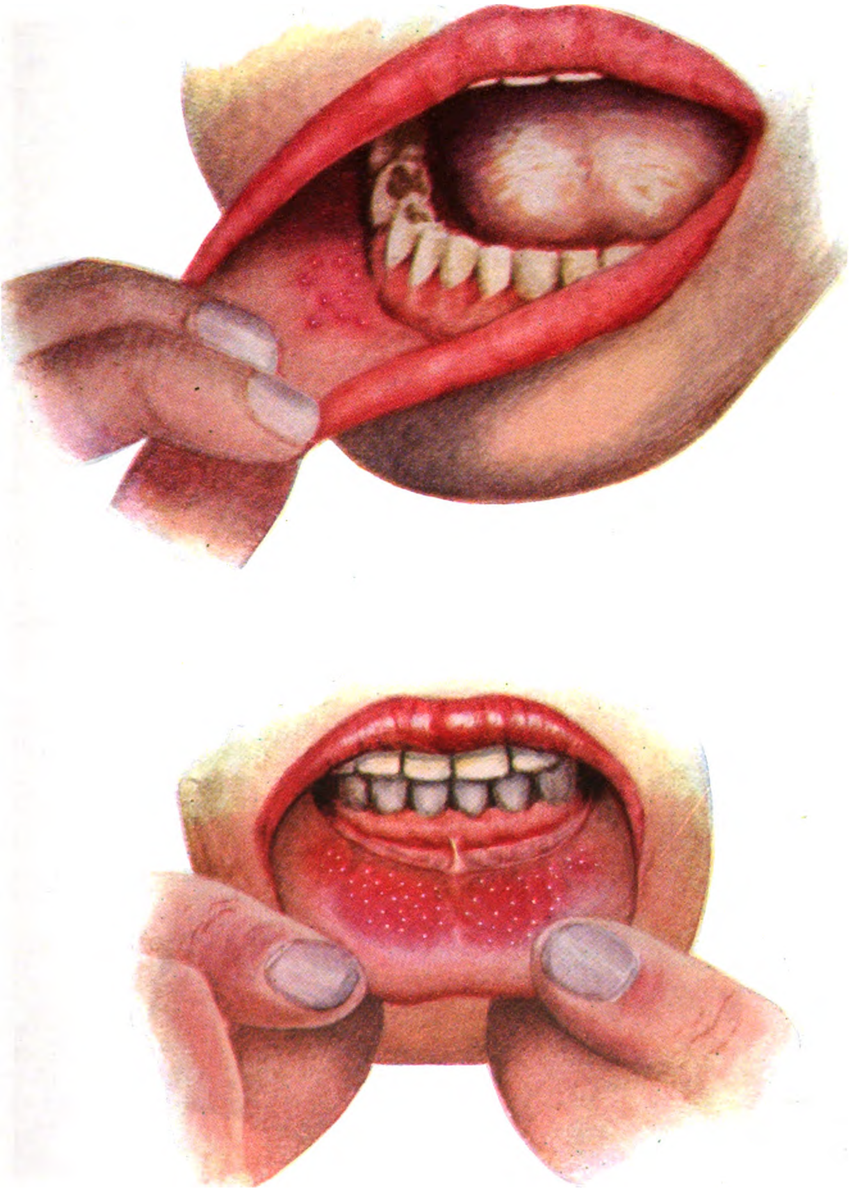
Excessive secretion by the lachrymal glands apart from emotion may, in some instances, lead to constant dripping of water from the nose as the result of *neurosis*.

One rare form of watery discharge from the nose is the escape of *cerebrospinal fluid* ; this fluid is perfectly transparent, like water, and it may be difficult to recognize its true nature unless there is a clear history of the commonest cause for the symptom, namely, an injury to the head leading to fracture through the base of the skull, involving one of the anterior fossæ. The fluid may drip steadily, at the rate of a certain number of drops per minute, and if it is collected in a test-tube it may be found to reduce Fehling's solution.

A **Purulent Discharge** from the nose may result from that which has been in the first place serous, mucoid, or muco-purulent ; or it may have been purulent from the beginning. If it is acute and bilateral, it is probably due to a local infection by some pyogenic micro-organism, and even when it may seem to be due to nothing more than a common cold, not a few different organisms may be discovered bacteriologically. Staphylococci, streptococci, and pneumococci (see *Plate XXVIII*, p. 614) are associated not at all infrequently with the *Micrococcus catarrhalis*. Influenza bacilli may be found. In rare cases, especially when the purulent discharge persists longer than it ought if it were the result merely of a cold, and especially in cases in which it is so acrid as to produce superficial excoriation and

PLATE VIII

KOPLIK'S SPOTS



soreness of the edges of the nostrils and the upper lip, diphtheria bacilli will be found more often than might be expected. *Nasal diphtheria*, indeed, is not altogether uncommon, but it is difficult to recognize except by bacteriological examination of the nasal discharge. The same applies to two very much rarer purulent lesions of the nose, namely those due to *gonococci* and to *glanders*. There may be a urethral infection or a vaginal discharge to point to the diagnosis in the former case, the patient having transferred gonococci directly from the genital source to the nose by means of the fingers or a towel. Purulent rhinitis due to *glanders* is fortunately rare, though when it does occur it may escape recognition entirely in its curable stage, unless the patient's occupation as a groom or horse-dealer suggests the source of the infection, or unless bacteriological methods are resorted to in all cases of nasal discharge that are not perfectly straightforward.

Chronic purulent nasal discharges are for the most part due either to lesions of the mucous membrane or to the emptying into the nose of purulent collections from the antrum of Highmore, frontal, ethmoidal, or sphenoidal sinus, or from necrosis of the nasal bones. The diagnosis may be obvious enough, but very often it is by no means easy. It is essential that both nasal cavities should be inspected directly in a good light by means of a speculum and mirror; the various kinds of chronic rhinitis may be recognized in this way; in chronic *atrophic rhinitis* the amount of discharge is usually small, the cavities of the nose are relatively spacious, the smell offensive (ozæna), and there are generally crust-like deposits upon the mucous membrane. Chronic *hypertrophic rhinitis* may also produce a very offensive smell, a considerable purulent discharge, and difficulty or even inability to breathe through the nose owing to the bulging of the inflamed mucous membrane. There may or may not be *polypi* at the same time, and perhaps *adenoids* and *enlarged tonsils* owing to the necessity for breathing through the mouth. *Membranous rhinitis* is not a distinctive variety, it being more or less an accident whether the inflamed mucous membrane produces a membranous exudate or not; the discovery of membrane would suggest diphtheria, but bacteriological examination alone can determine whether the lesion is diphtheritic or not. *Syphilis* is responsible for a large number of the cases of ozæna and chronic rhinitis, especially of the atrophic form, but it is not responsible for all, and the diagnosis as to whether the lesion is syphilitic or not will rest upon concomitant signs elsewhere, upon the history, and upon the result of Wassermann's reaction. Necrosis of the nasal bones, if it occurs spontaneously, is often syphilitic, but it may also result from an injury, such as a blow; the deformity which follows the falling in of the bridge of the nose is characteristic.

Tuberculous rhinitis is rare. There is a variety of nose affection called *rhinitis caseosa*, but this is acute and not tuberculous; the appearances might at first suggest that the nose was filled with a yellowish diphtheritic membrane, but on cultivation no diphtheria bacilli are to be found; what micro-organism is the cause of the cheesy exudate in these cases is not known; if left, the underlying mucosa is apt to ulcerate, but under simple antiseptic treatment cure results in a week or a little more. *Rhinoliths*, although they may cause persistence of a nasal discharge, are not in themselves a primary condition, but rather the result of preceding rhinitis. *Endothelioma*, *carcinoma*, or *sarcoma* affecting the nose are not common except as the result of direct spread to its interior from the lip, jaw, cheek, or forehead. Sometimes, however, considerable nasal discharge may result from the growth of a semi-malignant tumour known as *recurrent fibroma* or *fibro-sarcoma* arising from the external periosteum of the basi-sphenoid bone, thus obstructing the back of the nose, and detected by a digital examination viâ the mouth.

A *foreign body* inserted into the nose by a child or by an insane person may produce damage associated with a purulent discharge, which may persist even after the foreign body has been detected and removed.

Lupus of the nose is hardly ever primary, and although it may destroy the margins and lead to a purulent discharge from the nostrils, the diagnosis is generally clear from the apple-jelly deposits in the adjacent skin of the cheeks. *Rodent ulcer*, on the other hand, though starting in the skin, may spread deeply into the nose, causing destruction of cartilage and bone, with pain and purulent discharge. Whereas lupus starts in early adult life, rodent ulcer begins at or after middle age. Histological examination may be required to distinguish it from *epithelioma*, though the latter is likely to fungate more and to have advanced more rapidly than rodent ulcer does; the latter may have existed for years without any rapid advance. Radium treatment, efficiently applied, will cure most rodent ulcers of the skin, but this therapeutic test is no longer applicable when the cartilages and bones of the nose have become involved, for radium is then not able to cure the rodent ulcer any better than it can cure lupus or epithelioma.

Empyema of one antrum of Highmore may cause most troublesome purulent discharge from the nose, but it is not difficult to diagnose when the symptoms are definite. The patient generally complains that the pus invariably comes down one nostril; that it is associated with an odour which is offensive to himself in a way not common with ozæna generally; that he can often produce the discharge by tilting his head sideways in the opposite direction to that from which the discharge comes, and that he experiences dull aching pain in one side of the face, often spoken of as neuralgia, but upon investigation proving to be associated with tenderness located mainly in the corresponding superior maxilla. There may be a carious tooth, particularly a canine, from which infection of the antrum has taken place, though in a small number of cases a more serious cause exists, namely, carcinoma or endothelioma of the antrum, which can seldom be diagnosed until either an operation is undertaken or the growth itself begins to cause a protuberance either into the nose or through the face; the nature of these growths is determined histologically.

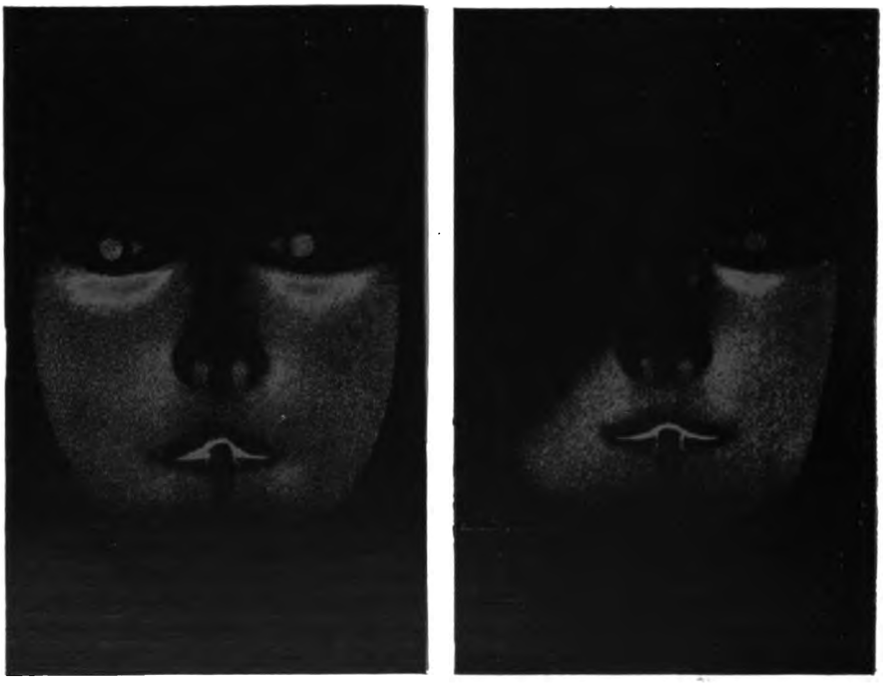


Fig. 84.—Transilluminations of the antrum. A shows the normal appearance. B shows no illumination of the right side, owing to purulent contents. (From *Medical Annual*, 1906.)

Examining the patient in a dark room by the introduction of an electric lamp into the mouth or posterior nares, may reveal empyema of the antrum by the transillumination of the superior maxilla of the normal side and the opacity of the other in which the antrum is full of pus (*Fig.* 84).

Empyema of a frontal sinus has generally been preceded by acute nasal catarrh, which has led subsequently to severe aching above one or other eye, with tenderness on percussion over the affected frontal sinus, and so much pain in this region that the patient may be compelled to hold his head before he is able to cough or blow his nose, because of the increased pressure within this sinus due to either of these acts. The condition nearly always starts acutely, though if untreated it may become chronic and come under observation only when the infection has tracked its way through into the subcutaneous tissue so as to point above the eye or in the angle between the latter and the nose.

Suppuration in connection with the ethmoidal or *sphenoidal sinuses* can be little more than guessed at unless special skill has been acquired in the direct examination of these air-cells. If, however, there is a purulent discharge from the nose coming apparently from high up, in a patient who has neither antral disease nor infection of the frontal sinus, and in whom local conditions of the mucous membrane of the nose itself can be excluded, infection of the sphenoidal or of the ethmoidal cells is to be suspected. *Herbert French.*

DISCHARGE FROM THE NIPPLE.—Discharges from the nipple may be divided into three classes:—(1) *Normal discharges*; (2) *Normal discharges at abnormal times*; (3) *Abnormal discharges.*

Normal Discharges.—It is quite natural for a woman during the period of pregnancy and lactation to have a discharge of milk from the breast. It is usually of small amount, except when the child is put to the breast, but occasionally the flow at other times may be sufficient to be distressing.

Normal Discharges at Abnormal Times.—Milk may come from the breast at other times than during pregnancy and lactation. In infants it may be found as the result of undue stimulation on the part of the nurse, and it has been noted in the breasts of both sexes at the time of puberty. No great importance attaches to it.

Abnormal Discharges.—*Blood or Blood-stained Discharge.* This is a very significant sign and should not be neglected, for it almost always indicates the presence of some abnormal condition in the breast which requires careful investigation. The commonest is some growth involving the larger ducts in the neighbourhood of the nipple. This may be either innocent—a *duct papilloma*; or malignant—*duct carcinoma, scirrhous carcinoma,* or *sarcoma.* It behoves one therefore never to neglect such a significant sign. When a well-marked lump is felt the diagnosis can usually be made without difficulty, and for this the reader is referred to the article on SWELLING, MAMMARY (p. 685). Difficulty arises when there is no obvious swelling. In these cases the breast must be palpated carefully with the flat of the hand and also with the tips of the fingers, special attention being given to the part immediately subjacent to the nipple. If no swelling can be made out, and the bleeding remains a persistent sign, it may become necessary to make an incision into the breast for diagnostic purposes, recognizing the fact that a papilloma may be so delicate as to escape detection with the finger. Probably the commonest cause of bleeding is a duct carcinoma (columnar-celled carcinoma); after that duct papilloma and scirrhous carcinoma, and last of all sarcoma.

A *purulent discharge,* or pus mixed with milk, generally indicates acute suppurative mastitis; the other signs of inflammation or abscess are well marked as a rule, so that there is no difficulty at arriving at a diagnosis. Chronic mastitis seldom causes a discharge of pus from the nipple, but the symptom is met with sometimes when the lesion is *tuberculous*; the discovery of tubercle bacilli in the discharge will distinguish this from carcinoma, with which it is often confused.

A *discharge of serum* will suggest chronic interstitial mastitis with cyst formation, but the symptom is rare.

Hydatid fluid has been recorded as escaping through the nipple from a *hydatid cyst* of the breast, but it is so rare as to be a pathological curiosity. The nature of the fluid would be recognized by the finding of hooklets in it (*Fig.* 18. p. 49). *George E. Gask.*

DISCHARGE, URETHRAL.—Any inflammatory process in the urethra causes a discharge. Although most commonly the result of infection by the gonococcus, by no means every urethritis is of this nature, and bacteriological examinations show that other organisms besides the gonococcus may produce a urethral discharge and the same symptoms as an acute gonorrhœa. Further than this, a purulent discharge may occur in which no micro-organisms can be found; for instance, when the urethra has been injured or subjected to irritation by the injection of strong solutions, or when it contains a foreign body, such as a calculus or a retained catheter. It is stated that a urethral discharge may be associated with *gout* and *rheumatism*; but although a few cases of the former have come under my care, I have been unable to prove that the small amount of discharge was not the remains of a former uncured urethral infection, or that it was directly due to the same source as the arthritic symptoms.

There is no doubt that an acute urethritis may be caused by other organisms than the gonococcus, and sometimes there is considerable trouble in completely curing it. These cases may cause complications in the genito-urinary organs similar to those due to the gonococcus, such as prostatitis, epididymitis, or cystitis. They may arise by the infection of the urethra by septic instrumentation, or after connection with a woman subject to leucorrhœa. A careful bacteriological examination should always be made; more than once the reputation of a wife has been at stake until it was proved that the husband's urethritis was of staphylococcal and not gonorrhœal origin. An acute urethritis may accompany a hæmatogenous urinary infection; for instance, an acute pyelitis due to bacillus coli may be followed by acute cystitis, prostatitis, and urethritis, in which no other organism but *Bacillus coli* can be found.

Gonorrhœal Urethritis is due to the infection of the urethra by the gonococcus of Neisser (*Plate XXVIII, Fig. R*, p. 614). In form it is a diplococcus with flattened surfaces approximating each other; it stains readily with basic aniline dyes, but differs from other diplococci in being decolorized by Gram's stain. The gonococcus is seen in a stained specimen to be *intracellular*, penetrating not only the leucocytes but also the epithelial cells found in a smear preparation, and, though the cocci may be found also between the cells, their appearance in the cells is strong evidence of their specific nature.

In any case presenting a purulent discharge from the urethra, it is necessary, in order that appropriate treatment may be carried out, to ascertain the extent of the infection, not only in the urethra itself, but also in the other organs of the genito-urinary apparatus. For the purposes of clinical investigation, the urethra is divided into anterior and posterior portions, separated by the membranous urethra, the anterior comprising the bulbous and penile urethra, and the posterior the prostatic portion. A urethritis is also, according to its clinical aspect, acute or chronic, the acute form being characterized by a thick, creamy, purulent discharge, with pain, and the chronic by a thin, greyish, mucopurulent discharge. Acute gonorrhœa affects not only the superficial layers of the urethral mucous membrane, but also the sub-epithelial tissues and the glandular elements, causing a leucocytic infiltration. The tendency of the inflammation is to spread backwards along the canal, so that the prostatic urethra may become infected, even in the acute stage, though most frequently this occurs at a later period; the prostatic and the ejaculatory ducts may become infected, and the inflammation may spread to the seminal vesicles, epididymes, or testes. In the acute stages of the disease, the infection of the anterior urethra is accompanied, as a rule, by redness of the external meatus, scalding pain during micturition, and painful erections; occasionally all pain is absent, especially in patients previously infected with gonorrhœa. If the anterior urethra be irrigated with sterile water or saline solution, the urine passed immediately afterwards will be quite clear; or without irrigating, if the urine be passed into two glasses, the first portion will be turbid from admixture with the urethral discharge, whilst the second portion remains clear.

When the posterior urethra becomes infected in the acute stages, the symptoms are much more severe. Micturition is more painful and greatly increased in frequency, both day and night, the patient often being obliged to pass urine every half-hour. Even after irrigating the anterior urethra the urine passed will be turbid with pus that has accumulated in the prostatic portion or passed backwards into the bladder, and the terminal urine may be tinged with blood. In these circumstances it may be necessary to eliminate *acute prostatitis* or *prostatic abscess*, either of which may complicate an acute posterior urethritis. In either condition, micturition may be very painful, or there may be acute retention; the temperature will be raised, and in cases of abscess there is often a rigor; upon rectal examination, the prostate is found much swollen, hot to the touch, and extremely tender, whilst with an abscess a soft fluctuating area may be felt. An acute posterior gonorrhœa is practically always accompanied by infection of the bladder, and the diagnosis between it and cystitis is practically impossible.

Under suitable treatment an acute urethritis may remain confined to the anterior urethra and clear up, but in less favourable cases a slight discharge remains. If this continues for longer than six weeks after the initial onset, it is spoken of as *chronic gonorrhœa* or *gleet*. The discharge is small in amount, thin and watery, or may be so slight as only to be present in the morning after a long period of freedom from urination, or as filaments in the urine. There is no pain or increased frequency of micturition, and there is no difference in the subjective symptoms between an anterior and a posterior infection, although in most cases of chronic gonorrhœa both are present.

In any case of chronic urethral discharge, examination should be conducted to ascertain not only the seat of infection, but also the nature of the lesion promoting the discharge. Thus, the patient should be directed to hold urine for at least three hours before he presents himself for examination, when the anterior urethra may be irrigated thoroughly by a fairly forcible stream of sterile water, the urinary meatus being alternately occluded and opened during the process, so that the whole length of the anterior urethra is distended by the fluid. The washing is then examined for any threads, which, if present, must proceed from the anterior urethra. The patient is then directed to pass urine into two separate glasses; if there is turbidity due to excess of phosphates, this is cleared by the addition of acetic acid, when, if any threads or plugs of muco-pus are present in the first specimen, they probably arise from the posterior urethra, whereas pus and turbidity of the second show that cystitis is present in addition. If there be any threads in the posterior urethra, or if only a small amount of discharge is present, it is advisable first to fill up the bladder with sterile fluid by direct Janet irrigation, after which the prostate is massaged by a finger in the rectum, and the patient is again directed to pass the fluid from the bladder. Plugs of muco-pus will be found if chronic prostatitis is present. In any case the threads from either the anterior or posterior urethra should be spread as a film, stained, and examined under a microscope for pus and micro-organisms.

If the remaining infection is found to be limited to the anterior urethra, the latter should be examined under direct vision by the endoscope. A few minims of a 3 per cent solution of cocaine are injected into the urethra and, with aseptic precautions, the largest sized endoscope tube that the meatus will admit comfortably is passed for about an inch. The canal is then illuminated, and at the same time distended with air by means of the inflating bellows attached to the instrument; each part of the anterior urethra can then be examined successively as the endoscope tube is passed gradually on until the membranous portion of the canal is reached. It will be found much better to examine the urethra in this manner than by first passing the instrument to the full extent and examining the canal as it is withdrawn, for any infection of the urethral glands, infiltration of the walls, or granular areas are observed under aero-distention before the instrument has passed over them. When the whole length has been examined under distention, the air is allowed to escape by opening the window of the instrument, and the canal again examined from behind forward by gradually withdrawing the tube, normal urethral walls falling together in a characteristic striated manner, which is altered into a slight rigidity by infiltration, whilst at the same time glandular infection or ulceration is again seen. Similarly, a definite stricture or a small polypus which may keep up a slight urethral discharge can be diagnosed with certainty, and any local treatment for the various lesions applied. By careful examination conducted on these lines we are able to determine, not only which part of the urethra is producing the discharge, but also the nature of the lesion, so that appropriate treatment can be carried out. In most cases in which a gleet remains in spite of treatment with various kinds of injections, it will be found that there is an infection of the posterior urethra or prostatic ducts, which no urethral injection except a complete irrigation into the bladder will reach. There is often no abnormality to be detected on digital examination of the prostate per rectum; but after urethral irrigation the secretion squeezed out from the prostate by massage will usually show pus corpuscles in addition to the refractile globules and epithelium which are contained in the normal prostatic secretion. In other cases of obstinate chronic urethritis, a distinct infiltrative process will be found in the anterior urethra, a process which results in rigidity of the urethral wall, and in severe cases leads on to stricture. The urethral glands are implicated, and their secretion gives rise to the filaments in the urine. This infiltration is seen readily by urethroscopy, but it may be imperceptible on the passage of a sound. The urethral meatus is the narrowest part of the canal, and a sound which will completely fill the meatus may still pass steadily through an infiltrated portion of the urethra, even when its normal calibre is diminished considerably.

In spite of all forms of treatment, a slight urethral discharge occasionally persists, and the physician may be asked if any infection remains, or whether a patient may be allowed to marry. A chronic urethral discharge may contain gonococci or may be entirely free from any organisms. Obviously, if any gonococci are found, the discharge is still infectious, but there is often difficulty in detecting the organism in these chronic cases, whilst in some they may be found if any slight exacerbation of inflammation occurs. Other cases again show a chronic urethral discharge which resists all treatment, but which contains a few pus and epithelial cells, though no organisms can be found. That pus cells are present in this small urethral discharge is no detriment to marriage, provided that no gonococci can be found, and in practice, if no cocci are found after irritation of the urethra by irritant injections, instrumentation, or the free use of alcohol, on several successive examinations, marriage may be permitted.

A urethral discharge may in rare cases be present in *other conditions than that produced by gonorrhœa or septic urethritis*, and as difficulty may arise if one of these cases be met with, it is necessary to mention them.

Herpetic Urethritis.—The mucous lining of the urethra is undoubtedly affected by herpes in the same manner as other mucous membranes, frequently as a tertiary lesion of syphilis. There is irritation of the urethra during micturition, and a slight muco-purulent discharge from the meatus. The small vesicles may be seen by the endoscope, and may be associated with herpes of the prepuce.

Soft Sores in the Urethra are distinctly uncommon. They occur in the terminal portion of the urethra, and cause painful micturition and a profuse, thin, purulent discharge, which contains no gonococci. There may be other sores on the glans penis, and an ulcerated surface will be seen on endoscopic examination. They occur within a few days of infection, and, if extensive, may produce narrowing of the urethra on healing.

Syphilis may affect the urethra either as a hard chancre or as a gumma.

The *Chancre* occurs in the anterior end of the urethra, forming a firm indurated mass which can be felt readily on external palpation. The meatus is œdematous and swollen, so that the introduction of an endoscopic tube is impossible ; there is a thin, purulent, and often blood-stained discharge from the meatus. A urethral chancre must be diagnosed carefully from peri-urethral infiltration due to urethritis ; the period of incubation from the time of infection, the presence of small, hard inguinal glands, the occurrence of secondary lesions of syphilis, and Wassermann's serum test will point to the diagnosis.

Gummata of the urethra give rise to a watery urethral discharge when they break down and cause ulceration. They may ulcerate through the canal and form fistulæ, but may usually be recognized on careful examination.

Papillomata of the Urethra may occur either in the anterior or posterior portion, as small, pedunculated tumours in the canal, and frequently as a sequel to a chronic gonorrhœa. They may arise, however, in the urethra of a patient who has never had urethritis. They cause a thin, scanty discharge, which does not yield to injections ; they are seen readily through the endoscope.

Carcinoma of the urethra is very rare as a primary disease, and in the few cases recorded has been in association with stricture. It forms a tumour in the urethra palpable from the exterior, and causes painful micturition with a blood-stained discharge, and enlargement of the inguinal glands. Suspicion of carcinoma should arise if a hard, irregular tumour be felt in the course of the urethra, without gonorrhœal infection, in an elderly patient, but the final diagnosis depends on histological examination of a portion of the growth.

Tuberculosis of the Urethra is always secondary to disease elsewhere in the genito-urinary tract, usually of the prostate or seminal vesicles.

Foreign Bodies in the Urethra may cause a purulent urethral discharge if they remain in the canal for any length of time. They may be introduced through the meatus by intent—matches, pins, etc. ; or a piece may be detached from a damaged catheter ; or a small calculus may come down from the bladder and be arrested. In the latter case the history is usually clear—sudden stoppage of the stream of urine during micturition, with penile pain ; a calculus may be felt from the exterior or seen through the endoscope.

R. H. Jocelyn Swan.

DISCHARGE, VAGINAL.—In order to recognize the varieties of pathological vaginal discharges, it is first important to realize what the normal secretions found in the vagina consist of. The secretion normally present must be a mixture of those from the uterine body, cervix, and vaginal wall. That from the uterine body is watery and small in amount, whilst that from the cervix is thick and mucoid, but clear and transparent, like unboiled white of egg. The vaginal secretion is merely a transudation of plasma from the vessels, mixed with desquamated vaginal epithelium, and in virgins looks like unboiled starch mixed with water. Naturally it is very small in amount. The bulk of the secretion found in the vagina comes from the cervix, because there are far more glands there than in any other part of the genital tract.

The secretion from Bartholin's gland, which is thin and mucoid, may be copious under sexual excitement, but under normal conditions is absent, and so does not contribute to the secretions in the vagina. The vaginal mixed secretions are acid in reaction, owing to the presence of lactic acid produced by a long bacillus which is found normally in the vagina. On the other hand, the unmixed uterine secretions are alkaline. Normally, the amount of mixed vaginal secretion should do no more than just moisten the vaginal orifice. When the amount is so great as to moisten the vulva and consequently stain garments, the secretion is pathological.

The composition of an abnormal secretion varies considerably according to the source from which most of it comes. The commonest type is the thick white or yellow discharge associated with inflammatory changes in the cervix. It contains a large proportion of mucus, many leucocytes, masses of shed epithelium from the vagina ('squames'), and bacteria of various kinds. This is quite typical, and is produced by *endocervicitis* and *cervical erosions* of the various kinds. When, however, there is a *corporeal endometritis* present as well, the discharge becomes thinned, white, or yellow, on account of the admixture of much watery secretion from the body of the uterus. The yellow colour is due to the admixture of red blood corpuscles, and in some cases the fluid may become actually blood-stained. Menorrhagia accompanies these discharges and serves to distinguish a mixed corporeal and cervical endometritis from a simple cervical catarrh. Microscopically the films made from the mixed cases show proportionately less mucus, but otherwise the constituents are the same.

Vaginitis rarely exists alone, but when it does occur the discharge is thick and pasty if it is a simple catarrhal condition: pasty on account of the large admixture of desquamated vaginal squamous epithelium. On the other hand, in granular catarrhal vaginitis the discharge is much more purulent and copious owing to the exudation of more fluid from the exposed blood capillaries. This is the kind of discharge associated with traumatism of the vagina, especially from the irritation of badly-fitting pessaries, and actual ulceration as in decubitus ulcers on prolapsed portions. Practically no mucus is found in such discharges unless the cervix shares in the inflammatory process.

There is nothing characteristic of *gonorrhœal discharges* to the naked eye or on simple microscopical examination. The detection of the gonococcus alone can decide the question. This is often a matter of great difficulty, because it is only in the few days immediately after infection that the gonococcus can be found free in the vaginal discharge. In chronic cases the gonococcus must be looked for in two places, either the interior of the cervix or in the urethra and Skene's tubes, which open by the sides of the meatus urinarius. The best plan is to take some discharge from within the cervix, after carefully wiping away discharges from the os uteri with sterile wool, using a Fergusson's speculum. This discharge should be spread on a glass slide and put by to dry. A second film on another slide should then be made, by squeezing the urethra from behind forwards and mopping up any secretion thus made to appear on the meatus. After drying in the air the films should be fixed by passing through a flame, and then stained by Gram's method, followed by neutral red as a counter-stain. In films prepared in this way gonococci are stained red whilst organisms which retain Gram's stain appear deep violet or black. The gonococci are usually found in the cytoplasm of the polymorphonuclear leucocytes (*Plate XXVIII*, p. 614).

Offensive smelling vaginal discharge is associated with decomposition, and it may be that the discharge itself is decomposing because it cannot escape fast enough from the passage, or that the source of the discharge is a decomposing substance like a *sloughing fibroid* or necrotic *carcinoma of the cervix*. In the two latter cases the discharge is copious, watery, and blood-stained, with a horribly fœtid smell. When the discharge itself is decomposing, it is usually thicker and purulent, and is commonly retained by pessaries or by redundant folds of vaginal mucous membrane. In old women a foul discharge may come from the interior of the uterus, a pyometra ; in which case pus can be made to flow from the os uteri by squeezing the uterus or passing a sound. It is due to *senile endometritis*, the result of infection, and is often associated with cancer of the body of the uterus.

Watery blood-stained discharge, not offensive, occurs in *cancer of the body of the uterus*, in early *cancer of the cervix*, with *mucous polypi*, *placental polypi*, and *hydatidiform mole*. The differential diagnosis of these conditions cannot be made from the discharge alone, but must rest upon physical examination combined with the use of the microscope upon materials removed from the uterus.

Vaginal casts may be composed of coagulated surface epithelium, the result of astringent injections or applications, and are easily recognized with the microscope. Membranous flakes may be passed with discharge in cases of membranous vaginitis. They consist of vaginal epithelium entangled in coagulated blood plasma, and present quite a different appearance to casts of coagulated epithelial layers. These membranous masses may be seen lining the whole vagina, and are generally due to special organisms. The diphtheria bacillus (*Plate XXVIII*, p. 614) has been found to be the causal agent in such cases, and in one investigated by the writer, the *Bacillus coli communis* was the offending organism.

T. G. Stevens.

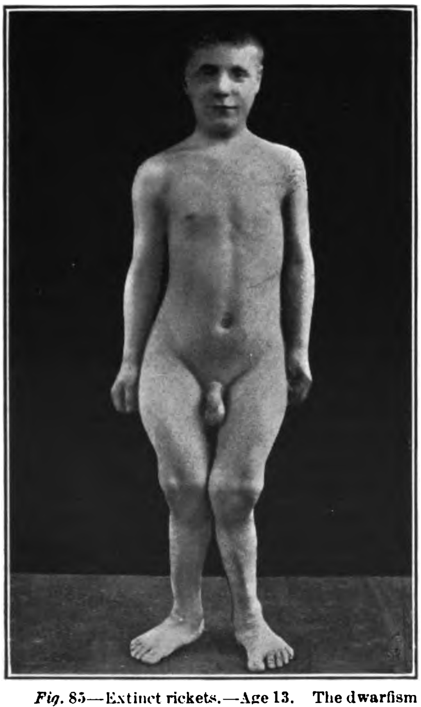


Fig. 85—Extinct rickets.—Age 13. The dwarfism is moderate, and is due to bending and shortening of the thigh and leg bones. There is knock-knee, the tibiæ are sabre-shaped, the feet flat. The wrists and ankles are large ; the muscles are not affected.

DIZZINESS.—(See Vertigo, p. 751.)

DOUBLE VISION.—(See Diplopia, p. 174.)

DROP-FOOT.—(See Paraplegia, p. 510 ; and Paralysis of One Extremity, Lower, p. 496.)

DROP-WRIST.—(See Atrophy, Muscular, p. 65).

DROPSY.—(See Œdema. p. 411.)

DWARFISM (Microsomia, Nanosomia).—For purposes of diagnosis, dwarfism may be divided into two classes, namely, *dwarfism the result of deformity*, and *dwarfism without deformity*. Generally speaking, well-proportioned dwarfs owe their defective stature to a generalized delay or arrest of development, and are therefore in a state of *infantilism*, whereas deformed dwarfs are stunted in growth only, though the reduction in height may be due rather to the warping or collapse of the bony frame-work than to actual curtailment of height.

I. DWARFISM THE RESULT OF DEFORMITY.

This kind of dwarfism is due mainly or solely to shortness of the legs. In most cases the primary fault lies in the skeleton, but occasionally the dwarfism has its source in deficiency of the brain, and still more rarely is brought about by a local defect of development implicating the lower extremities.

A. **Skeletal Dwarfism** is occasioned by :—

(1) Rickets ; (2) Achondroplasia ; (3) Osteogenesis imperfecta ; (4) Anosteoplasia ; (5) Osteomalacia.

Rickety Dwarfism (*Fig.* 85) is usually moderate in degree, and is due partly to actual shortening of the bones of the lower limbs and partly to bending (bow-legs or knock-knees).

It may also be the outcome of antero-posterior or of lateral curvature of the spine. The skull looks big and is of the square or hot-cross-bun type, with bulging forehead. The shape of the nose is not affected. There is often a pigeon breast or a transverse groove round the lower part of the chest (rickety girdle, Harrison's sulcus), and an hour-glass shaped or, at times, beaked (rostrate) pelvis. The muscles are well developed, and the body is squat and thickset.

In **Achondroplasia** (*Fig.* 86) the limbs are shorter than in rickets, and the stature less. The proportions are of the dachshund pattern. The shortening of the limbs is chiefly of the proximal segment, and the body, though actually short, is relatively long. The legs are often bowed, and there may be bending of the upper limb bones. The joints are usually prominent. The forehead is bulging, the bridge of the nose depressed. There is conspicuous lordosis, and the pelvis is small and contracted. The muscles are often disproportionately big, giving the achondroplasic a sturdy appearance and a surprising degree of strength. The fingers are broad, the three middle fingers being of equal length and divergently curved.

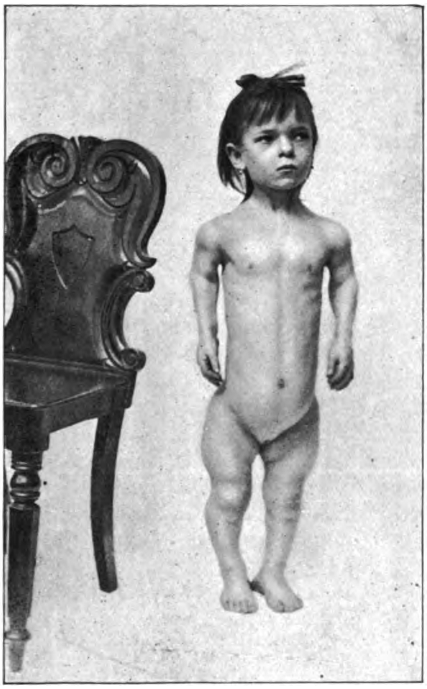


Fig. 86.—Achondroplasia.—Age 15. The trunk is almost of normal length, and the limbs very short, the proximal being shorter than the distal segments. The epiphyses are enlarged, and the forearms and legs are curved. The nose is deficient at the bridge. In this case there is infantilism as well as dwarfism.

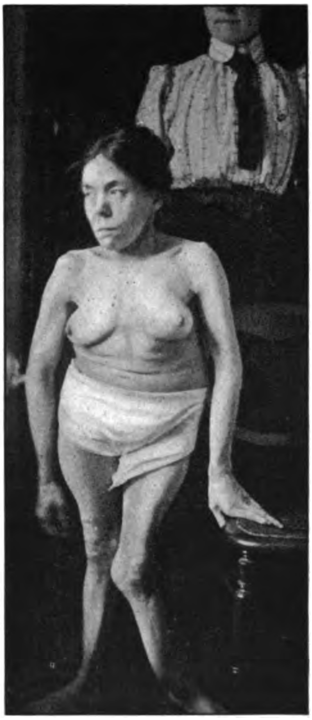


Fig. 87.—Osteogenesis imperfecta.—Age 32. Bending of the tibiæ, femora, spine, etc., began at five years, and continued for nearly ten years before it ceased. There was no epiphyseal enlargement.

Osteogenesis Imperfecta (osteopsathyrosis, fragilitas ossium) (*Fig.* 87) is characterized by brittleness with softening. There is not much dwarfing, except as the result of the yielding of the bones, and the muscles are usually weak. The disease probably sometimes runs on into osteomalacia.

Anosteoplasia, or cleido-cranial dysostosis. With general impairment of bone growth, causing moderate dwarfism, there is pronounced defect in the formation of the membrane bones. The skull is rounded and broad, the face small, the dentition delayed ; the clavicles are rudimentary or absent. The disease is often hereditary.

In **Osteomalacia** the dwarfism is due almost solely to the crumpling of the decalcified bones ; but when the disease occurs in childhood there is also some diminution of stature from arrest of bone growth. The muscles are conspicuously weak.

Diagnosis of Skeletal Dwarfism.—Though osteogenesis imperfecta, rickets, and achondroplasia can, as a rule, be distinguished readily one from another, cases occur in which one of these diseases seems to blend with another, or at any rate to partake of its characters. In distinguishing rickets from achondroplasia it must be remembered that the most characteristic features of rickety dwarfism are the bending and the post-natal origin, and of achondroplasia the shortness of the limbs (micromelia) and the pre-natal origin. The enlargement of the ends of the bones which is so distinctive of rickets disappears as the disease settles down and the bones continue to grow, whereas in the hyperplastic form of achondroplasia it remains throughout life. Extreme softening must cause us to suspect osteogenesis imperfecta or osteomalacia, even if there are rickety enlargements as well, especially if the bending continues to increase after the age of six years.

Dwarfism may be due solely to *spinal curvature.* If a *kyphosis* it is usually the result of tuberculous disease (caries) of the spine, but is occasionally a local manifestation of rickets, or possibly of osteomalacia. When of rickety origin there is not only kyphosis of the dorsal region, but a compensatory lordosis of the dorso-lumbar. If it begins in middle or old age it is usually osteo-arthritic, but as a rare event it may be due to osteomalacia (o. senilis). In the latter event the softening is usually confined to the spine and pelvis, and may take place with extraordinary rapidity, and be followed by gradual hardening and fixation in the deformed position.

Scoliosis is usually of mixed origin, the main factor being an inherent laxity of tissue showing itself in weakness of the back muscles and of the spinal ligaments. This laxity is supplemented by faulty positions of standing, sitting, etc., or by the injudicious use of stays. But it is probable that spinal curvature of sufficient severity to produce dwarfism is invariably the result either of rickets or, in rare cases, of a mild and local form of osteomalacia.

B. **Cerebral Dwarfism.**—This form of microsomia is most pronounced in *microcephaly,* but hydrocephaly, porencephaly, imbecility, or any degenerative cerebral affection of early progressive development may be associated with puny growth. The microcephalic dwarf is characterized not only by the relative smallness of his head (circumference never exceeding 17 ins.), but also by his sloping forehead, projecting nose, and receding chin, giving him a ferret- or rat-like physiognomy. He is usually quick of movement, and restless, and is either imbecile or idiotic, according to the degree of his microcephaly.

C. **Dwarfism from Pre-natal Deficiency of the Lower Limbs.**—This is of two kinds : phocomelus and ectromelus. In *phocomelus* the defect is in one or both of the proximal segments, leaving the hands and feet unaffected, so that the individual affected resembles a penguin or a seal (phoca). In *ectromelus* there is absence of part or whole of the limbs from the feet up.

II. DWARFISM THE RESULT OF DEFECTIVE GENERAL DEVELOPMENT. INFANTILISM.

Well-proportioned dwarfs are not invariably of backward development, for we meet with men of excellent development, who, if not actual dwarfs, are so dwarfish in stature that we have to admit the possibility of the existence of a true dwarfism in which there is no infantilism. Nevertheless, generally speaking, the dwarf of correct proportions is affected with infantilism.

To Distinguish Infantilism from simple Dwarfism.—Dwarfism is a defect of growth, whereas infantilism is a defect of development. In determining whether development is implicated, stature, ossification, and sex development are of great but not decisive importance. Thus infantilism may co-exist with gigantism ; and the ossification in some cases of symptomatic infantilism is not only not delayed, but may be actually premature. It is also premature in progeria. Moreover, a sexually mature child of five or six does not cease to be a child because its ossification and sexual condition resemble those of an adult. Evidently therefore neither height, nor sex, nor ossification is a cardinal feature of infantilism. Indeed, in some cases of sexual ateleiosis the presence of infantilism is determined by the child-like stature, proportions, and physiognomy alone, the individual being in all other respects a well-developed human being.

The Forms of Infantilism.—Infantilism may be widespread among whole races or nations (racial infantilism), or may select certain individuals or families, and occur epidemically or sporadically as morbid infantilism among people of ordinary development. Morbid infantilism is of two sorts, namely, *symptomatic*, the result of causes ; and *essential*, or cryptogenetic.

A. Symptomatic Infantilism.

This is seldom or never of extreme degree, has no uniform type of physiognomy, and, being an acquired condition, is never transmitted. It is best classified according to the nature of the cause by which it is produced.

It may be the result of *intoxication* with the poison of syphilis, wine, tobacco, or with that of rheumatic, scarlet or other fever, or with lead. Herter claims that the intoxication may arise from over-abundance of the normal flora of the intestine (intestinal infantilism).

It may be the result of *correlation*, as when it is associated with *kyphosis* or with *splenomegaly*, or with *hypertrophic cirrhosis* of the liver. Perhaps the best example of this form of infantilism is that which is associated with *microcephaly*. In some microcephalic dwarfs there is not only an impairment of growth, constituting dwarfism, but the development of the whole body is stayed, apparently because it is the custom for a certain development of the body to go with a certain size of the brain, and such customs are liable to be maintained even under abnormal conditions. Dwarfs with diminutive heads may be of just proportions and of fairly good intelligence, provided the growth of the body is so retarded that it remains in keeping with the growth of the brain. In *thymic* infantilism there is fatness with anæmia, and liability to syncopal attacks, which often end in death.

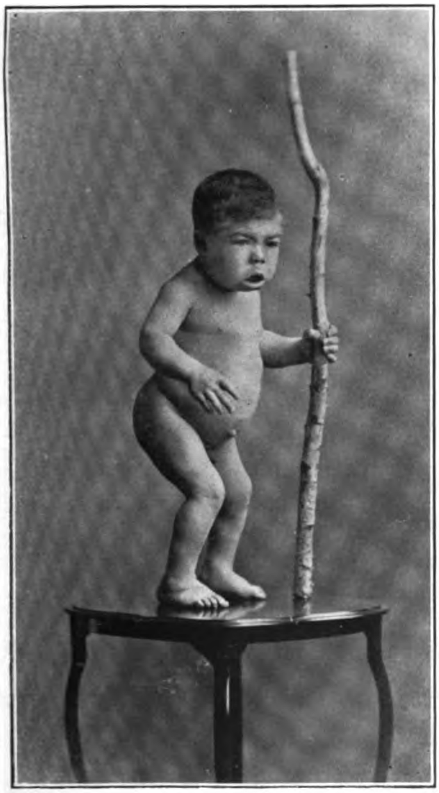


Fig. 88.—Cretinism.—Age 20. The infantilism is uniform and extreme. The intelligence, proportions attitude, manner, correspond with those of a child of 18 months. The features are puffed and disfigured with the characteristic pseudo-œdema.

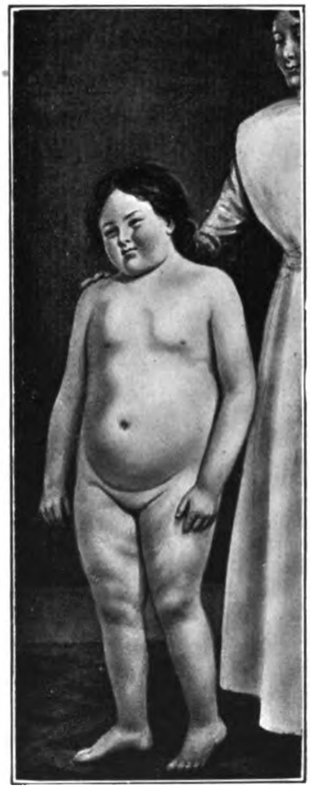


Fig. 89.—Pituitary infantilism.

It may be due to the *deficiency of a hormone* which ordinarily stimulates development. There are two forms : (*a*) Thyroid, and (*b*) Pituitary.

Thyroid infantilism in its most characteristic form—***cretinism*** (***Fig.*** 88)—is unmistakable; but cases of infantilism occur in which the physiognomy, stunting of growth, and backward sex development suggest mere thyroid inadequacy. Some reserve the name of thyroid infantilism for these cases of 'myxœdème fruste,' but the term should only be applied when the intelligence is defective and uniform improvement sets in as the result of giving thyroid extract. The thyroid inadequacy may however not be primary, but a mere incident in some other form of infantilism, ***e.g.***, ateleiosis.

In ***Pituitary*** infantilism (Froelich's syndrome, ***Fig.*** 89) there is fatness with conspicuous genital backwardness as well as a general defect of development. Polyuria or glycosuria is often present, and there may be drowsiness or nutritional changes in the skin and its appendages. Sickness, headache, or other symptoms of a cerebral tumour are occasionally present.

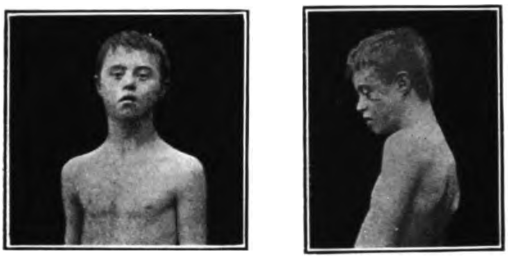


Fig. 90.—Mongolism.—Age 14. The general development is delayed. The physiognomy is undeveloped, is without facial hair, and is slightly Mongoloid. The occipital region is flat. The boy is an amiable imbecile.

Mongolism (***Fig.*** 90) is distinguished from cretinism or myxœdème fruste by the predominance of the imbecility as compared with the slightness of other cretinoid symptoms. In reality the physiognomy is only cretinoid because it remains of the infantile type. It is not disfigured by the thick lips and general pseudo-œdema of cretinism, and the tongue, though sometimes protruding, is not large.

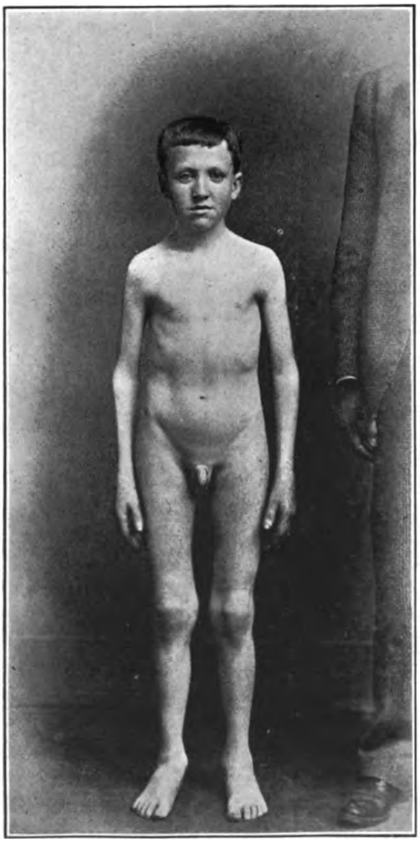


Fig. 91.—Anangioplastic infantilism.—Age 16. There is general delay of development, but not to an extreme degree. The physiognomy and proportions are of the adult pattern, but sex development is more immature.

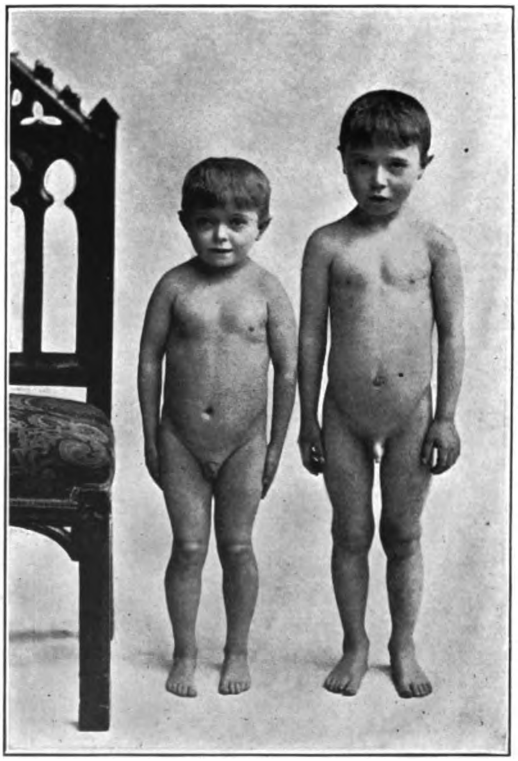


Fig. 92.—Essential Infantilism (Ateleiosis).—An ateleiotic boy, age 12, standing beside his normal and taller brother, age 6 years.

The eyes remind one of the obliquely-set eyes of the Chinaman, but it is sometimes difficult to make out the resemblance. The ligaments are lax, and mongols are liable to become knock-kneed and to have 'double-jointed' thumbs. There is often some valvular affection of the heart.

In a given case of infantilism it may be impossible to say how much is due to correlation, how much to intoxication, how much to hormonic deficiency, and how much to mere lack of nutrition. This may be said, *e.g.* of cardiac, or arterial, of renal, and of pancreatic infantilism.

Cardiac infantilism exists when there is some dominating incapacity of the cardiac valves. Sometimes there seems to be a deficient development of the whole arterial system, constituting *anangioplastic* infantilism (*Fig.* 91).

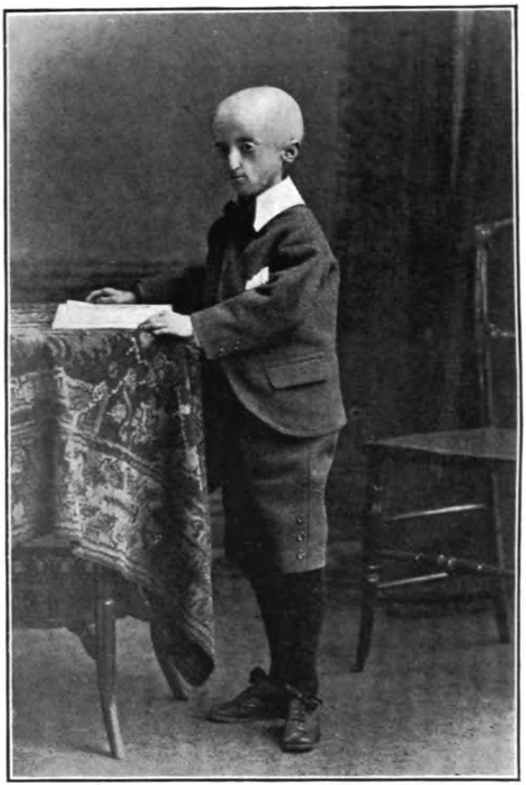


Fig. 93.—Progeria.—Age 15. The stature and proportions are childish, but the physiognomy, leanness, and baldness are elderly. The scalp was in reality sparsely covered with grey hair. The ear lobule is absent, the nasal cartilages are conspicuous, and the fingers nodose owing to the prominence of the epiphyses.

In *pancreatic* infantilism there are indications of pancreatic incompetence. The stools are fatty, copious or frequent, pale and offensive. Capsules of iodoform enclosed in a glutoid envelope (Sahli's capsules) are soluble only in the pancreatic secretion, and are therefore not dissolved in this form of infantilism. The infantilism is improved by pancreatic extract.

Renal infantilism is consecutive to chronic Bright's disease, and is suggested when there are polyuria, albuminuria, or other symptoms of Bright's disease, and no indication of a prior syphilitic or other intoxication.

B. **Essential or Cryptogenetic Infantilism.**

This is distinguished from symptomatic infantilism by its pronounced degree, by its seemingly spontaneous appearance, and occasionally by its heredity. There are two forms: *ateleiosis* and *progeria.*

Ateleiosis (*Fig.* 92) is primary, spontaneous infantilism. It may begin at any age of progressive development, and its characters are for the most part those normal to the age of its first appearance. It usually begins in infancy or early childhood, and the size, proportions, and physiognomy of this time of life are perpetuated. It is prone to be associated with cryptorchism, or with some corresponding ill-development of the ovaries, causing divergence into two varieties, sexual and asexual. In *asexual ateleiosis* all the physical features of infantile life are stereotyped; but in *sexual ateleiosis*, though the physiognomy and proportions remain infantile or childish, the onset of puberty (often greatly delayed) brings with it some accession of growth and the addition of the primary and secondary sex characters of the adult.

Progeria (*Fig.* 93) is primary, spontaneous infantilism mingled with premature senility (senilism). Hence, with shortness of stature and other indications of infantilism, there are baldness, emaciation, arterial sclerosis, and general decrepitude. Death from angina pectoris or other senile disease may ensue at 18 or even earlier. *Hastings Gilford.*

DYSARTHRIA.—(See SPEECH, ABNORMALITIES OF, p. 626.)

DYSCHEZIA.—(See CONSTIPATION, p. 121.)

DYSIDROSIS.—(See Sweating, Abnormalities of, p. 654.)

DYSMENORRHŒA owes its origin to a variety of causes, which must be differentiated carefully in order that treatment may be successful. The following table presents the causes of the three common varieties :—

1. Spasmodic.	2. Congestive.	3. Membranous.
Congenital malformations	Endometritis	
Deficient uterine muscle	Uterine congestion	
Long conical cervix	Retroversion and flexion	
Stenosed external or internal os	Uterine fibroids	
Neurasthenia	Salpingo-oöphoritis	
	Pelvic peritonitis	
	Small cystic ovary	
	Neurasthenia	

The distribution of the cases into these three classes is often easy ; in the first place, because spasmodic cases are practically always *primary*, that is, they commence with the onset of menstruation ; whilst congestive and membranous cases are *secondary*, that is, acquired as a result of some definite lesion. Further, the nature of the pain is often characteristic of the type of case, for in spasmodic cases the pain is intermittent, griping, and 'colicky,' commencing at the same time as the blood-flow, or only just before it. In the congestive cases, on the other hand, the pain is continuous and aching, and begins some hours or days before the flow. In typical cases also this pain is relieved by the flow. In the membranous cases the nature of the pain partakes of the characters of both the former types, being aching and continuous first ; then becoming colicky and spasmodic when the uterus is attempting to expel the characteristic membrane or cast, and being finally relieved when this comes away. Many cases are met with in which the pain partakes of the nature of both the congestive and spasmodic types. This usually means that a woman who originally had spasmodic dysmenorrhœa acquires some lesion which in its turn gives rise also to the congestive type of pain.

Having settled that a case belongs to one of the three main types, it is not very difficult to work out the actual causation. This is more difficult in the spasmodic cases than in the congestive, because the latter depend upon well-defined lesions, and the former do not.

Spasmodic Cases.—The causation of this type of case is often obscure ; but a bimanual examination, or a recto-abdominal examination in virgins, will usually reveal a condition of the uterus which can only be described as a *congenital malformation*. It may be small, but of the adult type ; it often has an exaggerated anterior bend, the 'cochleate' uterus of Pozzi ; and, in addition, the vaginal portion of the cervix is often too long, with a conical shape, and a very small pin-hole external os. Into such uteri the sound may pass with difficulty, owing to stenosis and rigidity of the internal os. The underlying true cause of the pain, however, is commonly admitted now to be imperfect development of the uterine muscle, in itself again a congenital malformation of texture occurring in an organ whose external form also is malformed The muscle being imperfect it is also possible that the endometrium is abnormal in these cases, unduly fibrous perhaps, and resistant : a point which our present knowledge does not prove or disprove. One proof, however, of the truth of these views is the effect of pregnancy and labour on such cases. They are nearly always cured, owing to the great muscular development during pregnancy, and the extreme stretching of the lower segment during labour. Neurasthenia also colours and increases the pain in these cases ; but, by itself, will not start a spasmodic any more than a congestive dysmenorrhœa.

Congestive Cases.—It is unnecessary to differentiate the congestive cases as tubal, ovarian, or uterine, because the underlying cause in all is uterine congestion accompanying such lesions as are shown in the table. The differential diagnosis of these lesions is to be made by a careful consideration of the history, combined with bimanual examination of the pelvic organs and, if necessary, curettage of the uterus, which also serves to cure the cases of pure endometritis. Cases due to *endometritis* are to be recognized by the cardinal symptoms of this lesion, namely, menorrhagia, leucorrhœa, often blood-stained, and chronic backache. These symptoms accompany slight enlargement of the uterus without any irregularity in shape such as would occur if fibroids were present. Simple *retroversion and flexion* can be recognized on bimanual examination ; the fundus will be felt posteriorly, the cervix looking directly down the vagina in a forward direction. *Salpingo-oöphoritis* in its typical chronic form gives rise to irregular very tender swellings on either side and behind the uterus, sometimes forming definitely retort-shaped swellings, especially if pus is present in the tubes. Fixation of these swellings and of the uterus is a very definite sign of the disease; whilst the history of one or more attacks of acute illness, with pelvic pain, will assist to make the diagnosis certain. The *small cystic ovary* may exist without obvious salpingo-oöphoritis, and without widespread fixation. The ovary is found to be permanently enlarged and irregular in shape from the projection of cysts from its surface. *Neurasthenia* is included under this heading because any menstrual pain is made worse by it, and only a very slight lesion need be present for this nerve weakness to accentuate any pain arising from it.

Membranous Cases.—The membrane, or cast, is of two types, and is easily recognized and distinguished from other uterine casts, such as those formed by the decidua of pregnancy. The classical cast of membranous dysmenorrhœa is hollow, triangular, not more than one-eighth of an inch thick, and possesses three openings. This, however, is not the common form; for in most cases the cast is solid, and formed by the mucosa being rolled upon itself. These casts contain connective-tissue cells and uterine glands in a stroma which is crowded with leucocytes. The solid cast may be nearly half an inch thick, and looks microscopically as if it were composed of endometrium into which hæmorrhage and leucocytic infiltration had occurred. The glands in it are broken up, and often lie on the outside. These casts never contain any compact masses of large cells of the decidual type, but an occasional hypertrophied connective-tissue cell may be found. Decidual casts, on the other hand, are the result of pregnancy, and consist of compact masses of large polygonal cells without any fibrillated connective tissue. They contain glands with hypertrophied epithelial linings, and often show large hæmorrhagic foci. The occasional presence in them of chorionic villi absolutely settles the diagnosis.

It must not be forgotten that cases of dysmenorrhœa may be mistaken for those of abdominal pain due to other lesions unconnected with menstruation; and the differentiation of such cases may be a matter of considerable importance. It is conceivable that dysmenorrhœa may be mistaken for:—

Appendicitis
Colic, intestinal, renal, or hepatic
Perforated gastric ulcer
Ruptured tubal gestation
Torsion of an ovarian cyst pedicle
Hæmorrhage from or into a Graafian follicle
Rupture of an ovarian cyst or pyosalpinx
Dyspepsia with flatulent distention.

Obviously, some of these lesions are dangerous to life, and therefore it is essential that they be not overlooked. The danger of this occurring is increased if any of these lesions start at or near the expected time of a menstrual period, and would hardly arise at all if a menstrual period had taken place recently, or was not expected for some days. It will be noted that all these lesions are accompanied by sudden abdominal pain, which might perhaps lead to a suspicion of spasmodic dysmenorrhœa, but hardly of congestive, owing to the character of the pain. *T. G. Stevens*

DYSPAREUNIA, or painful coitus, may depend on a variety of local lesions which require careful differentiation for their appropriate treatment, or it may exist when no local lesion can be found at all. It is associated closely with vaginismus, or painful spasm of the levator ani muscle on attempts at coitus, and the same lesions which cause simple dyspareunia may also give rise to vaginismus. It is remarkable that in some women a small local lesion will produce no pain upon attempts at coitus which in another will cause pain accompanied by violent spasm of the levator ani. In some cases pain arises because there is a difficulty of penetration of the vaginal orifice, whilst in others there is no difficulty, but pain is caused. The lesions which commonly give rise to dyspareunia are the following:—

Congenital absence of the lower part of the vagina
Unruptured hymen
Inflamed hymeneal orifice
Vulvitis
Bartholinitis
Leukoplakic vulvitis
Kraurosis vulvæ
Neuritis of the pudic nerve
Healed perineal lacerations
Urethral caruncle
Urethritis
Cystitis
Prolapsed tender ovaries with retroverted uterus
Chronic metritis
Salpingo-oöphoritis with adhesions
Anal fissure
Thrombosed and inflamed piles

It will be noted from a perusal of the above that the lesions fall into natural groups, according as the situation of the lesion is at the vulva, the uterus and ovaries, the urinary passages, or at the anus and rectum. Consequently it is necessary to carry out a detailed examination of any case of dyspareunia in order to find out whether any of these well-defined lesions are present.

The commonest lesion is certainly *inflamed hymeneal remains*, very often gonorrhœal in origin and accompanied by redness and swelling of the orifice of the duct of Bartholin's gland. The lesion is self-evident on inspection, and the parts are acutely sensitive to the least touch. *Leukoplakic vulvitis* is a lesion that is obvious from the white, sodden appearance of the labia minora, and causes pain on account of the sensitive cracks and fissures which accompany it. *Kraurosis vulvæ* causes actual contraction of the vaginal orifice, and consequently penetration is difficult and causes pain. The red projecting growth from the meatus urinarius, *caruncle*, is self-evident and acutely tender, whilst *urethritis* is diagnosed by the issue of pus on squeezing the urethra. *Cystitis* is diagnosed by the presence of pus and mucus in the urine, accompanied by frequency of micturition, and it causes pain because the bladder is painful in such cases and intolerant of the disturbance caused by coitus. *Pudic neuritis* is not a well-defined condition, but can be recognized by tenderness along the pudic nerve just inside the vaginal orifice, where the nerve passes along the inner side of the ischial ramus. In *prolapsed tender ovaries* and *backward displacements* there is no pain on penetration and no difficulty, but coitus gives acute pain. The condition is recognized by a bimanual examination, the same remarks applying to *salpingo-oöphoritis*, bearing in mind that there is usually a history of some acute attack of pelvic peritonitis in such cases. In *chronic metritis* the tubes and ovaries may be normal, but the uterus though normal in position is tender to the touch, and consequently coitus causes pain. *Anal fissure, thrombosed and inflamed piles*, can only be recognized by a careful examination of the anus and rectum by the finger and speculum.

In the cases which occur without local lesions the vaginal entrance will be found to be hyperæsthetic as a rule, and penetration is impossible. Such cases are almost always accompanied by spasmodic vaginismus. The most careful examination fails to demonstrate a lesion in such cases, and they are usually termed 'neurotic' for the want of a better term. Such cases do not necessarily mean absence of sexual desire ; on the contrary, many such patients are desirous of the consummation of marriage. Enlarging the orifice, or even child-bearing, does not cure a true case of this nature ; it must be in some way a disorder of function of the nerve centres. These cases must be distinguished from those in which the underlying factor is absence of sexual desire and actual dislike of the sexual act. Unhappy and unsuitable marriages conduce to this state of affairs, and the patient is liable to complain of pain when dislike is really what is meant. There is no difficulty in penetration in such cases. *T. G. Stevens.*

DYSPEPSIA.—(See INDIGESTION, p. 315 ; and FLATULENCE, p. 240.)

DYSPHAGIA literally means difficulty in swallowing, but the term itself does not indicate whether the difficulty is mechanical, nervous, or due to pain ; there are consequently several entirely different groups of cases, to each of which the term dysphagia has been applied :—

1. **Dysphagia due to Mechanical Obstruction to the Œsophagus.**—The usual history of progressive mechanical obstruction to the œsophagus is as follows : there is little or no pain, but the patient notices that whereas formerly he could swallow anything with ease, he is beginning to experience difficulty with the more solid kinds of food, such as meat, dry bread, and vegetables, so that he is obliged to live mainly upon pulpy foods : milk puddings, gruel, and the like. Later he can swallow only liquids ; ultimately he finds that even these are apt to be regurgitated soon after they have been swallowed, and there is often a sense of obstruction at some point between the level of the cricoid cartilage and the lower end of the gladiolus, which latter corresponds, as regards sensation, with the cardiac end of the œsophagus. When with the above history the patient gives a definite account of having swallowed some strong irritant or corrosive substance, such as an alkali or a mineral acid, the diagnosis of *fibrous stricture* from *corrosive* injury is easy. When similar obstruction succeeds the swallowing of a *foreign body*, such as a tooth-plate (*Fig.* 94),

a large piece of bone, or a coin, the diagnosis is also easy as a rule, though in some cases there may be doubt as to the existence of a foreign body in the œsophagus unless the œsophagoscope is used, or the x-rays employed with or without bismuth (*Fig*, 95). Where the symptoms are not directly attributable to anything of this nature, however, but come on insidiously, the diagnosis generally lies between *squamous-celled carcinoma* of the œsophagus, *carcinoma of the stomach* directly invading the lower end of the œsophagus, and *aortic aneurysm* stenosing the œsophagus from outside. The actual fact of obstruction has first to be determined, and there is danger in passing a bougie unless aortic aneurysm can be excluded; this exclusion is by no means easy, however, for that variety of aneurysm which is most liable to stenose the œsophagus is one affecting the descending thoracic aorta, so that it does not give rise to any tumour, or pulsation, or bruit, and it is placed too far along the aorta to cause inequality of the pulses, inequality of the pupils (from interference with the cervical sympathetic), paralysis of a vocal cord (from interference with the left recurrent laryngeal nerve), tracheal tugging, or pain down either arm. The only other effects besides œsophageal obstruction likely to be due to aneurysm in this position are: pain in the dorsal region of the spine, possibly radiating along the course of one or more of the mid-dorsal intercostal nerves towards the left, and perhaps obstruction to the lower part of the root of the left lung, causing impairment of note, of air-entry, or of voice sounds, with or without some crackling râles over the left lower lobe behind. If a bougie is passed, it should be a soft one, and extreme care should be taken; but the danger may be avoided in towns where x-ray installations exist, for the obstruction may often be demonstrated by making the patient swallow a capsule or gruel containing bismuth or barium chloride and watching its course (*Figs*. 96, 97); while an aneurysm would cast a characteristic shadow in the posterior mediastinum. The older the patient, the more likely is it to be carcinoma of the œsophagus and not aneurysm. The differential diagnosis between primary growth of the œsophagus and infiltration of the œsophagus by a growth starting at the cardiac end of the stomach, is often one of great difficulty, unless there

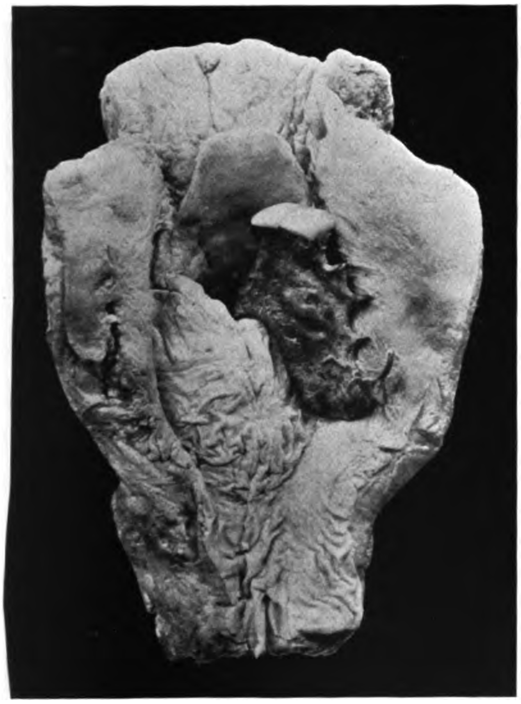


Fig. 94.—Sudden death from acute dyspnœa and asphyxia: tooth-plate impacted in the larynx. (*From a case of Dr. T. Warner Lacey, of Woolwich.*)

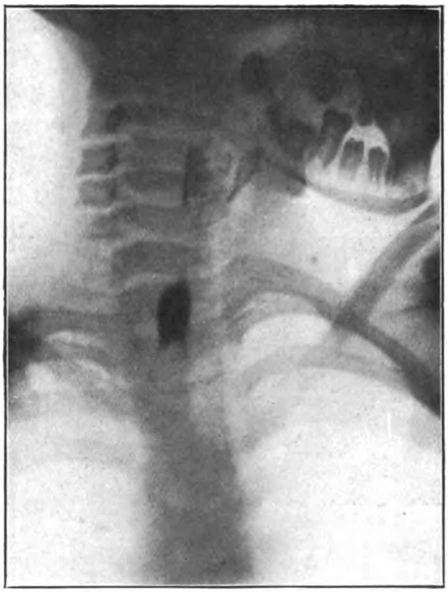


Fig. 95.—Œsophagus blocked by a bean, which does not show, but suspends the bismuth food, the lower border of which arches over the bean. As a result of the x-ray examination the œsophagus was further explored and the foreign body removed.
(*Skiagram by Dr. C. Thurstan Holland.*)

have been definite gastric symptoms before dysphagia set in. Secondary nodules would naturally be looked for, especially in the lymphatic glands in the lower part of the neck and in the liver. A history of syphilis and evidence of syphilitic aortic regurgitation, especially in a man between the ages of forty and fifty who had been a hard manual worker and not teetotal, would render aneurysm probable.

When aortic aneurysm can be excluded, much information as to the nature of an œsophageal obstruction may sometimes be obtained from the use of an œsophagoscope, and the latter can be used at the same time in facilitating the removal of such things as a foreign body.

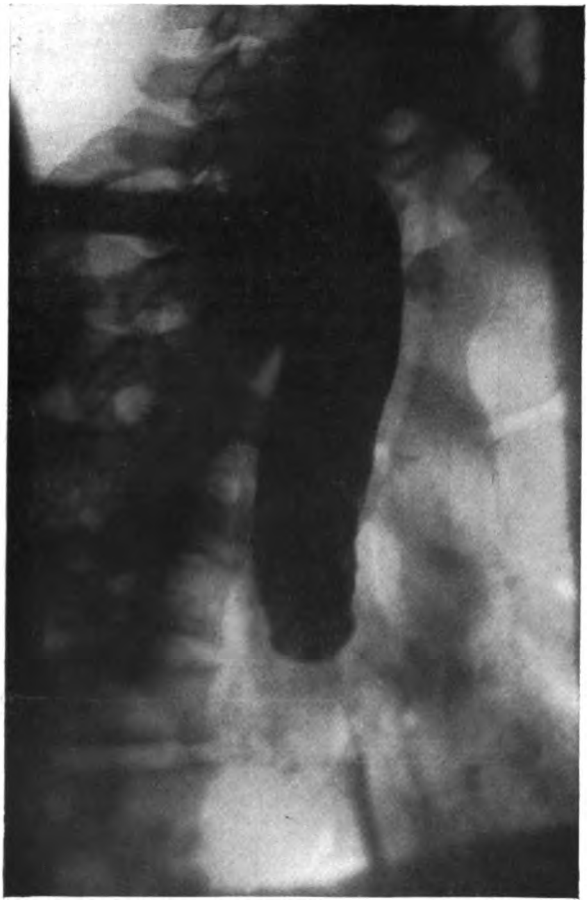


Fig. 96.—Skiagram, after a bismuth meal, showing the bismuth held up by a malignant stricture of the œsophagus at about the level of the bifurcation of the trachea.

Dysphagia lusoria is a very rare condition due to compression of the œsophagus by the right subclavian artery when it arises from the aorta beyond the left subclavian and passes to the right side either in front of or behind the œsophagus; the diagnosis in such cases will be almost impossible, though it might be guessed at if there were other congenital deformities, such as club-foot or transposition of the viscera.

Œsophageal pouches cause symptoms which can seldom be interpreted with certainty unless the case is watched for some time. Generally the patient can swallow with ease on some days, but with considerable difficulty on others; aneurysm, new growth, and traumatic or corrosive obstruction to the œsophagus will be excluded partly by the results of *x*-ray examination and partly by the age—pouch cases are relatively young. The point which suggests the diagnosis of a pouch is that the patient who has been able to swallow perfectly well for a few days, and then begins to have difficulty in getting the food down, finds relief presently on the regurgitation—clearly not from the stomach but from some situation higher up—of a larger quantity of food material than had been swallowed immediately before, including perhaps articles which were taken one or more days previously. The reason for these symptoms is that the pouch does not obstruct the œsophagus until it becomes very much distended by the gradual accumulation in it of portions of the food swallowed, relief coming when the greatly distended sac empties itself back into the œsophagus.

2. **Dysphagia due to Nervous Causes without Obstruction.**—The two commonest varieties of dysphagia due to purely nervous causes are probably *post-diphtheritic* and *hysterical*. The former is characterized by regurgitation of the food through the nose, due to paralysis of the soft palate; inspection may demonstrate the flaccid condition of the latter; there may have been a history of sore throat, of other cases of diphtheria in the patient's neighbourhood, or Klebs-Löffler bacilli may have been found, or may still be found in the patient's throat. When regurgitation of the food through the nose develops

in a person who is not known to have had diphtheria, the symptom will usually arouse grave suspicion that diphtheria of a mild type has occurred but has been overlooked. There may or may not be other signs of peripheral neuritis, or there may be paralysis of the ciliary muscles of the eyes.

Hysteria as a cause for dysphagia is familiar enough under the heading of globus hystericus, the diagnosis of which is not as a rule difficult, especially if the patient be a young woman who has suffered from other functional nervous affections, for instance hysterical aphonia.

Less common varieties of dysphagia of nervous origin are :—

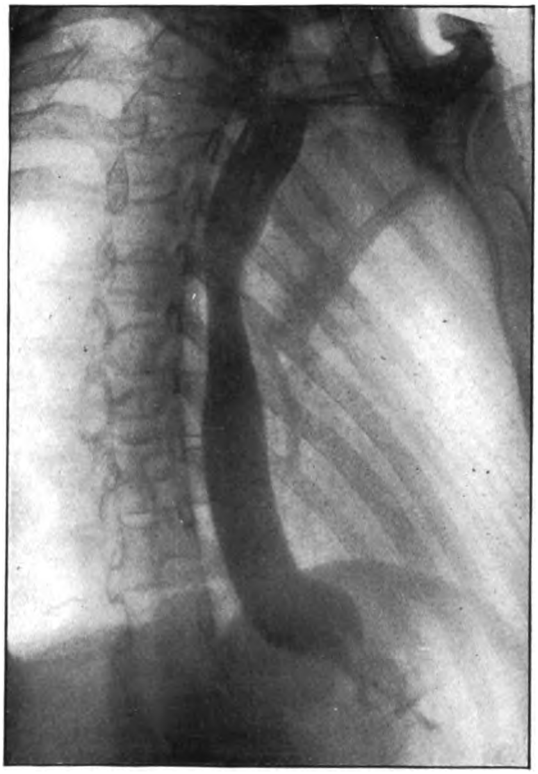


Fig. 97.—Skiagram taken in the semi-lateral position after bismuth administration in a case of stenosis of the œsophagus by a carcinoma at the cardiac orifice. The diagnosis was verified at subsequent operation. The bismuth had been taken 20 minutes previous to the x-ray examination and none had yet entered the stomach. (*Skiagram by Dr. C. Thurstan Holland.*)

Bulbar paralysis, in which the characteristic and progressive difficulty in the use of the lips, tongue, pharynx, and larynx points at once to the diagnosis, the only difficulty that may arise being perhaps in distinguishing true bulbar paralysis, in which the lesion is in the motor nuclei of the medulla oblongata, from pseudo-bulbar paralysis, where the lesion is due to bilateral cortical softening : in the true form there is atrophy of the tongue, in the pseudo variety the tongue does not atrophy, and chiefly upon this point is the differential diagnosis made.

Syphilitic degeneration of the medullary centres may produce symptoms not unlike those of ordinary bulbar paralysis, but it is generally differentiated by the fact that other cranial nerves, particularly those of the eyeball, are probably affected at the same time, and there may also be evidence or a clear history of syphilis, with or without a positive Wassermann's reaction.

Lead poisoning and *alcoholism* may also be responsible for degenerative lesions affecting the nerves concerned in the process of swallowing.

General paralysis of the insane ultimately results in inability to swallow ; the swallowing reflex is amongst the very last to disappear, and the diagnosis has long since been established upon other grounds.

Spasmodic dysphagia, due to spasm of the muscular coats of the œsophagus and pharynx, is probably the cause of globus hystericus, but similar spasticity may prevent swallowing in much more serious diseases, and constitutes a prominent symptom in *hydrophobia,* in which any effort to swallow liquids produces the symptom in extreme degree. The history of a dog-bite as a source of contagion is the chief point in arriving at the diagnosis.

Myasthenia gravis is a very characteristic disease, in which the muscles that are affected are perfectly able to do their work when they first begin to contract, but become fatigued with great rapidity, so that after the first few contractions, those which succeed become less and less effectual, until they cease, and the affected muscles will only be able to work again when they have been given a long rest. The neck muscles, and those of the eye, larynx, and mouth, become involved early (*Fig.* 111, p. 235), and difficulty in swallowing after the first few mouthfuls is sometimes a characteristic feature of the case. The myasthenic electrical reaction (see REACTION OF DEGENERATION, p. 584) serves to distinguish these cases from those due to bulbar paralysis.

Finally, there are very rare cases in which, without any known pathology, the œsophagus becomes enormously hypertrophied and dilated, and the patient cannot swallow, though a bougie passes perfectly well. This so-called *idiopathic dilatation and hypertrophy* of the œsophagus is fortunately very rare ; it has generally been regarded as due to cardiospasm—an erroneous spasmodic contraction of the cardiac orifice, which refuses to relax for the ingress of food into the stomach ; but a more recent view is that it is due, not to any extra spasm, but to defective relaxation of the normal tonicity—a condition to which the term *achalazia* has been applied. It leads to dilatation of the œsophagus behind it, with much hypertrophy, the latter, great though it is, eventually failing to overcome the neuro-muscular constriction of the œsophageal sphincter, though bougies pass without difficulty. It may be recognized by the use of bismuth or barium and the x-rays (*Fig.* 98).

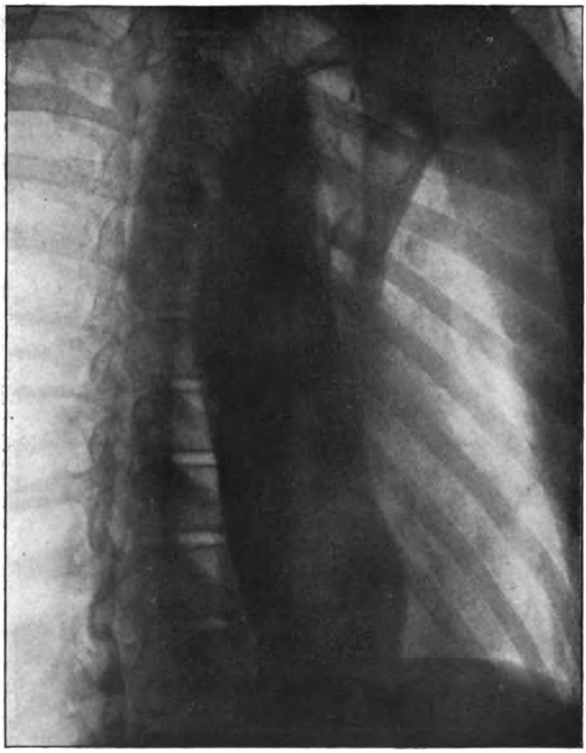


Fig. 98.—Skiagram in the semi-lateral position after administration of bismuth in a man, age 46, suffering from 'cardiospasm' or idiopathic dilatation of the œsophagus. History of 28 years' vomiting, indigestion, pain after food. Had been to many physicians : had taken "all the medicines in the pharmacopœia," but was never diagnosed correctly until bismuth and x-ray examination was resorted to previous to a proposed gastro-enterostomy. (*Skiagram by Dr. C. Thurstan Holland.*)

3. **Dysphagia due to Mechanical Defects of the Mouth or Pharynx, the Œsophagus being Normal.**—This group of cases includes patients suffering from such conditions as widely cleft palate, syphilitic stenosis of the pharynx, inability to use the tongue, either because it is acutely swollen from glossitis, bee-sting, or angina Ludovici, or because it is fixed from carcinomatous infiltration, and so forth. There is little need to enter into the differential diagnosis of this variety of dysphagia, for it can generally be determined by direct examination of the buccal cavity. Mumps, quinsy, and post-pharyngeal abscess belong to the same group, the last-named causing more dyspnœa than dysphagia, and being confined to quite early childhood.

4. **Dysphagia in which there is no Mechanical Obstruction, but in which the Act of Swallowing causes the Patient so much Pain that he hesitates to Swallow.**—The chief causes of dysphagia which come under this heading are : Inflammatory affections of the mouth or tongue, including the different varieties of *stomatitis* (p. 542) ; *pemphigus* or *erythema bullosum* of the buccal cavity, evidenced by similar eruption upon the skin (see BULLÆ, p. 96) ; *ulcers* of the tongue, whether malignant, gummatous, tuberculous, or due merely to erosion by a carious tooth or an ill-fitting tooth-plate ; *sore throats* of various kinds (see SORE THROAT, p. 613) ; pain in the mouth, larynx, or œsophagus after swallowing acute irritants or fluids that are either exceedingly cold or burning hot ; and *inflammatory affections of the larynx* and its immediate neighbourhood.

The nature of the buccal lesions will generally be indicated by inspection.

The different varieties of sore throat may be distinguished to some extent by inspection, though bacteriological confirmation is usually advisable.

The chief difficulties arise when the cause of the dysphagia is a painful affection of the larynx. Rarities such as variolous, lupoid, leprous, typhoidal, decubital, and traumatic ulcers of the larynx will seldom be diagnosed unless there is obvious collateral evidence, such as the eruption of small-pox upon the skin, residence in leprous countries, prolonged confinement to bed, and so forth, to indicate the nature of the case. The commoner varieties of laryngeal trouble which produce dysphagia are *acute laryngitis*, *tuberculous laryngitis* with or without ulcers, *carcinomatous ulceration* of the larynx, and *syphilis*. Laryngoscopic examination is essential, local anæsthesia by the use of the cocaine generally being necessary first. If tubercle bacilli can be found in the sputum, or if there are abnormal signs at the apices of the lungs, the diagnosis of tuberculous laryngitis is probable, and the pallid swelling of the aryteno-epiglottidean folds, and, still more so, multiple small ulcers of the edge or posterior surface of the epiglottis or of the free edges of the true or false vocal cords, or similar ulcers in other parts of the larynx, bilaterally situated, would indicate the diagnosis with certainty. The chief difficulty arises in the more chronic cases in which, after the larynx has become involved, the lung condition has improved, and tubercle bacilli may not be found in the sputum. Epitheliomatous ulceration of the larynx may be very extensive, and yet for a long time remain confined to one side ; this unilateral distribution of the infiltration is often important in distinguishing epithelioma from syphilis of the larynx, whilst the latter may also be distinguished by the repair which may ensue even after extensive destruction of the tissues has led to much deformity of the parts. The influence of salvarsan or potassium iodide and mercury upon the lesions may assist the diagnosis, and Wassermann's serum test may be employed. Doubt may remain, however, and sometimes, where it is very important to arrive at a certain diagnosis as soon as possible, a small portion of the affected tissue may be excised and examined microscopically. When tuberculosis, syphilis, and new growth are excluded, and yet laryngitis is present, the probability is that it is due to some infecting organism. Probably the symptoms will have started more or less acutely, even though they persist and become chronic ; laryngeal inspection may show acute hyperæmia and injection of the parts with extensive œdema without ulceration, and the nature of the micro-organism concerned—the diphtheria bacillus, streptococcus, pneumococcus, etc.—may be determined bacteriologically by preparing cultures from local swabbings. It is possible, of course, for two or more maladies to occur simultaneously, and it is particularly difficult to distinguish syphilitic laryngitis from tuberculous in a syphilitic patient who has undoubted phthisis ; similarly, it may be difficult to distinguish catarrhal laryngitis from tuberculous in phthisical patients, and so on ; indeed, in many instances the diagnosis may be one of opinion only. *Measles* is very apt to be accompanied by laryngitis, which may often be merely catarrhal, but which not infrequently is due to diphtheria developing synchronously with the measles. In order to exclude diphtheria, it is always advisable to take swabbings for bacteriological investigation even where it seems almost obvious that the laryngeal catarrh is merely part of the general coryza of measles. In all these cases dysphagia will be accompanied by hoarseness or other alteration in the voice pointing to an affection of the larynx.

Herbert French.

DYSPNŒA, or marked difficulty or distress in breathing, may or may not be associated with orthopnœa : in the milder cases a patient when at rest has no dyspnœa, the difficulty with breathing being brought out only by exertion ; nearly all conditions which may produce dyspnœa, however, are capable in later stages of producing orthopnœa, so that the causes of dyspnœa and of orthopnœa are similar in kind though they differ in degree. There is no need, therefore, to repeat what will be found under the heading ORTHOPNŒA (p. 418) whilst the article on BREATH, SHORTNESS OF (p. 87) should also be consulted.

Herbert French.

DYSTOCIA signifies difficult birth or labour. The difficulties of delivery show themselves by prolongation or delay in the completion of the stages into which labour is usually divided. Difficult labour is accompanied by progressive symptoms, objective and subjective, which are to be explained by physiological exhaustion, especially in its effect upon the central nervous system of the patient. The results of difficult labour are thus of such importance, affecting, as they do, the life of the mother and child, that anticipation of it, and therefore early and appropriate treatment, are of paramount importance in scientific midwifery.

The causes may be tabulated according as they occur in the first or second stage, the first series delaying the dilatation of the cervix, the second the expulsion of the child. It is not out of place in this connection to add also the causes of difficulties in the separation and expulsion of the placenta, for delivery cannot be said to be complete until the placenta is expelled.

CAUSES OF DELAY IN COMPLETION OF THE THREE STAGES OF LABOUR.

1st Stage.	*2nd Stage.*	*3rd Stage.*
Weak uterine contractions	Weak uterine contractions	Weak uterine contractions
Rigidity of cervix: relative, spasmodic, cicatricial, new growths	Secondary uterine inertia	Morbid adhesion of placenta
Pendulous belly, causing anteversion	Absence of accessory muscular effort	Uterine spasm
Early rupture of membranes, due to malpresentations, morbid adhesions to the lower uterine segment, undue friability	Rigidity of vagina and perineum	'Hour glass' contraction
Malpresentations in general	Loaded rectum	Adhesion of membranes
Anything which prevents the head entering the lower uterine segment	Distended bladder—cystocele	
Hydramnios	Contracted pelvis	
Deficiency of liquor amnii	Pelvic tumours: Fibromyoma, ovarian tumours, growths of the pelvic bones, hæmatoma, varicose veins, vaginal growths	
Twins.	Malpresentations; Occipito-posterior, breech, face, brow, transverse	
	Any abnormal enlargement of the child: Hydrocephalus, meningocele, ascites, tumours, double monsters, very large child	
	Excessive ossification of the head	
	Short cord: absolute, relative	
	Locked twins	

From the above it will be seen that the causes of delay are very numerous and important; and the successful delivery of the child under many of these conditions depends very much on their *anticipation*, rather than their recognition when delivery is already dangerously obstructed. Consequently, accurate diagnosis at the beginning of labour will often save much trouble to the practitioner, and danger to the mother and child. Indeed, some of the dangers of obstructed labour can only be avoided satisfactorily by careful examination of the patient during pregnancy, say at the thirtieth week. This applies specially to the recognition of contracted pelves, of pelvic tumours, and sometimes of malpresentations, and constitutes an important reason why every patient should be urged to undergo an examination during the later weeks of pregnancy.

The routine method of examination of the pregnant woman, whether in labour or not, is the same; and the deductions to be made from it are identical. The examination is made as follows: first, by abdominal palpation; secondly, by vaginal examination.

Abdominal Palpation.—First feel for the fœtal head in the pelvis by the 'pelvic grip,' or Pawlik's grip. In a primipara the head should be well down in the pelvis; not necessarily so in a multipara. Failing to find the head in the pelvis, palpate for it at the fundus; failing to find it here, it will be found in one or the other lateral situations. If the head is in the pelvis, and fixed, there can be no pelvic contraction of importance, and tumours of the uterus or ovaries *below the brim* are quite unlikely. If, however the head is above the brim and movable in a primipara, pelvic contraction must be suspected, whilst a tumour preventing entrance into the pelvis is a possibility. Pelvic contraction may be verified by pelvimetry, for which see below. Abnormal presentations are recognized by abdominal palpation; breech and transverse by the actual position of the head; occipito-posterior by the presence of the 'small parts,' arms and legs, in front, and the absence of the back of the fœtus; a face cannot be diagnosed absolutely except in mento-posterior cases, when the groove between the extended occiput and back will be felt in front whilst the head remains above the brim. *Hydramnios* may be recognized if there be fluctuation, and the fœtal parts can only be felt by deep dipping through the fluid. *Twins* may possibly be recognized by feeling two heads, and hearing two fœtal hearts beating with different rhythms.

Vaginal Examination.—It is important to remember that very little can be made out with one or two fingers. As a rule, all that can be noted is the *condition of the canal*, whether narrow or rigid, with a powerfully acting levator ani muscle, and the *condition of the os* ; note especially its consistence, and the integrity of the membranes. It may not even be possible to recognize the presentation if this has not been made out by abdominal palpation. If contracted pelvis is suspected, the important diameter, namely, the diagonal conjugate, should be measured with the fingers, and the true conjugate estimated by subtracting half an inch from this measurement. The only accurate instrument for taking this measurement is *Skutsch's pelvimeter* ; but its use requires considerable experience, and, in general, the simpler method with the fingers is sufficiently accurate for most purposes. External measurements may be made to supplement the important internal one ; but they are not of the same practical importance. When a difficulty arises in labour, accurate diagnosis is indispensable, and the whole hand should be inserted into the vagina under anæsthesia. The presenting part may then be grasped, and its absolute character determined. In this way occipito-posterior presentations (the commonest cause of difficult labour) can be diagnosed with certainty, and rectified. Hydrocephalus may be recognized by this manœuvre ; the hand may be pushed on above the head without danger in most cases, and the neck felt for coils of cord, the body of the child palpated for the presence of tumours or enlargement by ascites. Tumours obstructing delivery are best felt from the vagina ; they are usually wedged between the presenting part and the sacral promontory, part below and part above this prominence. If fluctuating and soft they are usually ovarian cysts ; if hard and unyielding they may be fibromyomata of the uterus ; but these also are apt to soften during pregnancy, and to feel like fluid tumours. Tumours of the pelvic bones are usually bony, or cartilaginous ; growths of the cervix may be fibroid, but more commonly are friable carcinomata, bleeding freely on examination.

Little more than the method of examination can be indicated in a short article on the diagnosis of a case of difficult labour ; but too much stress cannot be laid on the value of abdominal examination and palpation as the most important means of gaining information in any labour.

Delay in the Delivery of the Placenta, though not strictly a part of difficult labour, presents difficulties in the completion of delivery, and must not be overlooked. The placenta may be simply retained in utero ; may be adherent to the uterus, totally or partially ; or may be retained in the vagina. In the first case, if there is no hæmorrhage, the placenta is likely to lie in the lower uterine segment and vagina, and is not expelled owing to weakness of the accessory muscles. If partially adherent, bleeding is certain to occur, whilst total adhesion does not permit of any bleeding. In any case of this kind if, after a sufficient time has elapsed, the placenta cannot be expressed, the hand must be introduced into the uterus in order to diagnose the condition. It must not be forgotten that the placenta may be retained above a spasmodic stricture of some part of the uterus, the so-called *hour-glass contraction*. Hæmorrhage always accompanies this condition if the placenta is partly separated.

Finally, the *symptoms of exhaustion* consequent upon obstructed labour may be mentioned. The first are rise of temperature and increase in frequency of the pulse-rate. These afford very important indications of obstructed labour, and assist us to distinguish this from simple delay from weak uterine contractions, in which the pulse and temperature remain normal. The later symptoms of obstruction, if not relieved, are local and general. Locally, the vaginal secretions fail, the parts become hot, dry, and swollen. The uterus contracts powerfully, and may go into a tetanic condition, usually known as tonic contraction, in which case the uterus is hard, never relaxing, and is tender to the touch. The exact opposite occurs in uterine inertia, when the uterus remains flaccid, along with a normal pulse and temperature. Later still, vomiting may occur, signs of septic infection may appear, and rupture of the uterus may take place owing to the dangerous thinning of the lower segment when tonic contraction supervenes. This series of symptoms should never occur in properly conducted midwifery ; their possible occurrence should always be anticipated by correct diagnosis early in labour, followed by immediate appropriate treatment. *T. G. Stevens.*

EAR, DISCHARGE FROM.—(See OTORRHŒA, p. 421.)

EARACHE is the term usually applied to the pain experienced in acute inflammation of the middle ear. It is most acute when suppuration ensues. There are, however, a number of other conditions--many of them of great importance—which also give rise to otalgia or to pain roughly localized by the patient to the ear.

In *acute otitis media* the pain is usually dull, continuous, and throbbing, with sharp exacerbations in which the pain shoots to the occiput, to the top of the head, or forwards to the temporal region. It is usually worse at night—indeed it may disappear in the day—and it is increased by pressure over the tragus and on opening the mouth. Not infrequently there is some tenderness over the mastoid process. There is always some impairment of hearing. In adults there will probably be a slight rise of temperature; in children the temperature may rise to 103° F., or more, the pain is often very acute, and constitutional symptoms may be very marked, with convulsions, vomiting, and delirium. Such cases may be mistaken for meningitis, especially in children too young to talk; but in these little patients attention may be directed to the trouble by the extreme tenderness of the affected ear, the least manipulation of which may cause the child to scream. In young children the presence of cerebral symptoms with pyrexia should always lead to a careful examination of the ears. The presence of optic neuritis favours a diagnosis of extension of the inflammation to the interior of the cranial cavity, but this is not a universal rule, for cases are recorded in which otitis media by itself has caused optic neuritis. Attacks of earache in childhood are frequently caused by *adenoids*, and indeed, acute otitis media is practically always caused by an extension of inflammation from the nasopharynx along the Eustachian tubes. When suppuration occurs, the membrane becomes perforated, pus escapes, and the pain usually ceases. When it persists, the perforation is probably too small to allow of satisfactory drainage of the pus. Examination of the tympanic membrane by means of a speculum will show redness, loss of lustre, and probably bulging of the membrane, with blurring of the handle of the malleus.

Chronic middle-ear suppuration is usually painless. When necrosis occurs pain is often present and may be very acute, but this is by no means invariable, and some cases of extensive caries are remarkably free from pain.

Pain and tenderness over the mastoid process are also present in acute *mastoid abscess* and *periostitis*.

Pain in the ear may also be caused by the following lesions of the external auditory meatus, which may be diagnosed on examination through a speculum:—

A foreign body, especially if an *insect* finds its way into the meatus.

Furuncles; intense pain, often throbbing in nature, is followed by a discharge of pus, after which the pain diminishes; the meatus is so tender that it may be impossible for the patient to endure the presence of the speculum.

Cerumen is usually painless, though sometimes a dull pain may be present.

Eczema of the meatus may be the cause of a burning or smarting pain.

Sometimes a careful examination of the ear will fail to reveal any lesion. Under these circumstances the possibility of one of the following causes of referred pain must be considered:—

A carious molar tooth, a very common cause of pain referred to the ear.

Epithelioma of the tongue or *ulceration of the pharynx or larynx*; pain in the ear may be a very troublesome symptom of any of these.

Acute or subacute *tonsillitis* often causes acute pain in the ear without any inflammatory lesion of the middle ear. Less frequently, *suppuration in the accessory sinuses* of the nose has a similar result.

Otalgia may sometimes be neuralgic, and it is then usually associated with *trigeminal neuralgia*. It may also occur in nervous anæmic patients, and sometimes must be regarded as a neurosis. It must also be remembered that the glenoid lobe of the parotid gland extends into the non-articular portion of the glenoid fossa, and thus *parotitis* may cause pain referred to the ear. Similarly otalgia may occur with *osteo-arthritis* or *inflammatory trouble in the temporo-mandibular joint*.

Certain diseases of the auricle may cause pain more or less severe in character. Perichondritis is by no means uncommon. It may be traumatic or spontaneous, and in the latter syphilis may be the cause. Herpes, acute eczema, erysipelas, Raynaud's disease, and chilblains are all accompanied by pain and may affect the auricle. Sebaceous cysts and dermoids also occur here, and when inflamed will cause more or less severe pain. A condition known as 'telephone ear' has been described in persons who constantly use the telephone. More or less severe pain is present, and this may be accompanied by tinnitus and associated with the presence of boils.

Lastly, it must be remembered that there is a lymphatic gland situated over the mastoid process which drains lymph from the side of the scalp; when inflamed, this gland may be the cause of pain and tenderness, which may lead to a suspicion of suppuration in the mastoid process. *Philip Turner.*

ECCHYMOSIS.—(See PURPURA, p. 552.)

ECTHYMA.—(See SCABS, p, 599.)

EFFUSION, PLEURAL.—(See CHEST, BLOODY EFFUSION IN, p. 102; CHEST, SEROUS EFFUSION IN, p. 104; and CHEST, PUS IN, p. 103.)

EGG-SHELL CRACKLING.—(See CRACKLING, EGG-SHELL, p. 150.)

ELECTRICAL REACTIONS.—(See REACTION OF DEGENERATION, p. 582.)

EMACIATION.—(See MARASMUS, p. 384; and WEIGHT, LOSS OF, p. 768.)

EMPHYSEMA, SURGICAL.—Surgical or subcutaneous emphysema is due to distention of the subcutaneous areolar tissues with air or gas. The diagnosis of the condition and its cause is not as a rule difficult. Its commonest starting-place is in connection with the thorax, particularly when there has been *injury to the lung* tissue by a broken rib, a stab with a knife, a bullet wound, the rupture of alveoli due to excessive coughing, as in whooping-cough and bronchitis, or during great strain, as in difficult labour; or by operative injury to the lung, as in exploratory needling of the chest. The gas spreads rapidly, and may extend over the greater part of the trunk in a short time, disappearing again in the course of a few days. It may do so similarly after the operation of *tracheotomy.*

The face may sometimes be almost suddenly involved unilaterally by the escape of air into the subcutaneous tissues from the upper part of the nose, after violent *sneezing* or energetic *blowing of the nose.*

Rarer causes for the escape of actual air into the subcutaneous tissues are *ulcerative* or *traumatic lesions* of the *œsophagus*, *stomach*, *duodenum*, *cæcum*, *bladder*, or *rectum.* Air escaping in the areolar tissues around any of these parts may sometimes extend and become palpable as crepitus under the skin.

Quite another type is that in which the gases in the tissues are not air, but the results of *infection by gas-producing bacteria.* Fortunately cases of this kind are now rare; they were less uncommon in the days of *hospital gangrene* and *putrefaction.* The *Bacillus coli communis*, however, not infrequently liberates gas in an abscess to which it may give rise—for instance in the region of the vermiform appendix—and sometimes subcutaneous emphysema results. Another gas-producing organism that attacks man, though less often as a primary affection than intercurrently during some other malady, is the *Bacillus aërogenes capsulatus*; this, however, more often produces gas-containing loculi in the liver and other internal organs than in the tissues beneath the skin. *Herbert French.*

EMPYEMA.—(See CHEST, PUS IN, p. 103.)

ENLARGEMENT OF A BONE.—(See SWELLING ON A BONE, p. 667.)

ENLARGEMENT OF THE FOREHEAD.—Many individuals who have passed middle age—males more so than females—tend to develop an increasing prominence of that part of the forehead which corresponds with the outer casing of the frontal air sinuses; with the result that their eyebrows seem to overhang the eyes more and more, and the countenance looks different from what it did ten or fifteen years before. This is due to slow enlargement of the air cells of the frontal sinuses, and it is not pathological. This normal enlargement of the forehead has to be distinguished from two diseases which, though rare,

are generally recognizable with ease if the patient is watched over a period of months or years, namely, *leontiasis ossea* and *acromegaly*.

The commonest symptom that a patient suffering from leontiasis ossea complains of is that in former years he always took a certain size of hat, and was able to order hats without having to go and try them on ; of recent years, however, he has found that he has had to get progressively increasing sizes, so that whereas formerly a number 7 may have fitted him, he may now require even so large a size as a number 8 ; in a few exceptional cases special hats have had to be made for the patient because the enlargement of the head, especially of the forehead, has become tremendous, whilst at the same time it may very likely not be quite symmetrical. The general health remains good, and if the patient does not mind his personal appearance and the size of his hats, he lives for years without suffering any other inconvenience. On the other hand, the bony changes may not be confined to the skull, but may affect the bones of the limbs as well, especially the tibiæ ; there is probably a relationship between leontiasis ossea and osteitis deformans or Paget's disease of the bones (see p. 155). If the cranium is examined after death, it is found that there is no longer any distinction between the hard ivory bone upon the surface and the cancellous bone in the centre ; both have assumed an intermediate character, so that the whole bone is more or less of the same texture, very thick and heavy, and in a condition which used to be spoken of as *osteoporosis*. In some cases the change is syphilitic.

In acromegaly it happens very rarely that the frontal bone is affected alone ; much more often the affection of the forehead is much slighter than the increase in size of the lower jaw and of the phalanges of the hands and feet (p. 237). If, however, the changes were more marked in the frontal bone or in the bones of the skull generally than in those elsewhere, it is probable that a case of acromegaly would be diagnosed as one of leontiasis ossea, and one does not really know what essential difference there is between these two. Whereas, however, in acromegaly the bigness of the lower jaw makes the characteristic facies, in leontiasis ossea the prominence of the forehead gives the face that leonine character from which the name of the disease is taken.

No other maladies in adults are likely to cause uniform increase in the size of the forehead, but occasionally one meets with tumours of the frontal bone which cause a symmetrical enlargement of the forehead, the most important of these being the *ivory exostosis*—a non-malignant tumour which may arise from any of the flat bones of the skull ; it grows very slowly but enlarges progressively, and in so doing is apt to displace anything which comes in its way, and in the course of many years great deformity of the eye or nose may thus result. The slowness of the growth, and its very hard character generally, point to the diagnosis at once, and an x-ray examination may help to confirm it.

Other asymmetrical enlargements of the forehead may result from *syphilitic nodes* caused by gummatous periostitis terminating in bony organization ; *sarcoma of the periosteum*, a very rare primary growth in this region, but when met with suggested by the relative softness of the mass and its rapid increase in size ; *secondary malignant disease*, likely to be mistaken for primary sarcoma if no primary growth elsewhere is known, but readily diagnosed correctly if the existence, now or formerly, of a carcinoma of the breast, thyroid gland, or other part is known.

Any other tumours in connection with the frontal bone are exceedingly rare. The very extensive disease of the frontal, as of any other cranial bone, which used to be met with in syphilitic subjects, is now practically unknown on account of the greater adequacy of the treatment of syphilis in its earlier stages.

Leprosy may be mentioned as a cause of enlargement of the forehead (*Fig.* 173, p. 404), for in the nodular form any part may be affected ; but it must be very rare for leprosy to affect the forehead region only, and the diagnosis will be suggested by the lesions elsewhere and by the history of the case.

The above remarks apply to enlargement of the forehead in adults ; in children quite different causes will suggest themselves, the three most important being : (1) *Hydrocephalus*, (2) *Rickets*, (3) *Congenital syphilis*.

It happens not infrequently that a child's forehead enlarges very considerably, and bulges with much convexity to such an extent as to make both the parents and the physician fear hydrocephalus when the child is suffering from nothing more serious than rickets. The diagnosis may be quite difficult if there are not at the same time the other familiar signs of rickets mentioned on page 167; and there are not a few instances in which it is only when the case has been watched for months or years that one can be sure that there is not hydrocephalus. The same applies to the swelling of the frontal bone that may result from congenital syphilis. In the case of both rickets and congenital syphilis, one will examine the whole of the head carefully, to try and make up one's mind whether the enlargement, which usually affects not only the forehead but also other parts of the skull, is a more or less uniform stretching such as hydrocephalus gives rise to, or whether there are not some parts which are enlarged and other parts which are more or less normal. Both congenital syphilis and rickets are apt to produce diffuse round prominences of the parietal regions as well as of the frontal regions, so that there are four main bulges with an antero-posterior and a transverse groove between them, constituting the hot-cross-bun-shaped type of head; but the difficulty of excluding hydrocephalus is made greater still when, as sometimes happens, there is such thinning of the bones in the occipital region from cranio-tabes that the bones can be dented inwards like stiff parchment; such cranio-tabes may result either from rickets or from congenital syphilis. One would then pay special attention to the regions of the sutures; if these are obviously stretched asunder the case is almost certainly hydrocephalus, and not rickets or congenital syphilis. One would also be able to draw some conclusion perhaps from the appearances of the eyes, for the eyeballs will be in normal position when the cause of the forehead enlargement is rickets or congenital syphilis, whilst with hydrocephalus the eyes will give the impression of being displaced; sometimes they look very much deeper set than normal; in other cases they look as though they are depressed as the result of the downward pressure exerted by the excess of fluid upon the roofs of the orbits. If the enlargement and prominence of the forehead dates from birth or soon afterwards, this will be an argument in favour of hydrocephalus; if the change develops later in the infant's or child's life, there will almost certainly be a history of a severe attack associated with symptoms of increased intracranial pressure, for probably the commonest cause of acquired hydrocephalus is a preceding attack of meningococcal meningitis, from which the child has recovered. The history, therefore, may help in deciding the diagnosis. The optic discs should also be examined, for in a certain number of cases of acquired hydrocephalus there is optic atrophy (*Plate XX*, *Fig. n*, p. 418), and this is practically never met with as the result of rickets and very seldom as the result of congenital syphilis. It is of course only when the degree of hydrocephalus is medium that it is difficult to distinguish it from the forehead enlargements due to rickets or congenital syphilis. Major degrees of hydrocephalus cause such extreme enlargement of the whole head, coupled with such thinning of the bones and stretching of the sutures, that the diagnosis is almost unmistakable.

Although either simple or malignant tumours may affect the frontal bones, even in an infant or child, they are very rare. They should be diagnosed in the same way as similar tumours in adults. *Chloroma* may perhaps be mentioned specially, rare though it is. The tumours in such a case are never single, but as they may develop upon bones, they sometimes attract notice first in connection with the cranial bones, and thus perhaps a local enlargement of the forehead may be the first symptom in the case. There is a tendency for the glands generally to become enlarged and sometimes the spleen also, and in some respects the malady simulates lymphatic leukæmia. Neoplasm of some kind will be an early suspicion, and the nature of the growth is indicated by the greenish colour of the tumour when it has been excised. The actual diagnosis, however, is made more often post mortem than during life.

The commonest local swelling of the forehead in a child is a *hæmatoma* resulting from injury, and as the blood clot is often quite deep-seated there is sometimes no discoloration of the skin, and some more serious tumour may be thought of until the disappearance of the mass in the course of a week or two proves its simple character. Such a hæmatoma after a day or two softens in its central part in a remarkable way, leaving very hard raised edges, and on palpation it feels almost as if there were a hard bony ring with an absence of any bone at all in the centre; the first time such a softening hæmatoma of the forehead is felt, one can hardly believe that it is only a hæmatoma and not an actual hole in the bone covered merely by scalp and skin. The feeling, however, on palpation is so characteristic that once felt the condition is readily recognizable in any subsequent case.

Herbert French.

ENLARGEMENT OF THE GALL-BLADDER.—(See GALL-BLADDER ENLARGEMENT, p. 252.)

ENLARGEMENT OF THE HEART may be due to hypertrophy of the walls of any of its cavities, but especially of the ventricles; to dilatation of the cavities; or to these two conditions combined.

The most important physical signs of enlargement of the heart are: (1) Displacement of the cardiac impulse; (2) An increased area of cardiac dullness. After puberty the normal cardiac impulse is usually situated in the fifth left intercostal space, about three-quarters of an inch internal to the left nipple line. Before puberty it is normally in the fourth left space in the nipple line. When the heart is enlarged, the impulse is displaced outwards and also downwards. Particular care must be taken to determine the exact position, as from this observation a good idea of the particular part of the heart which has enlarged may be obtained. When the left ventricle is much hypertrophied, the cardiac impulse is displaced more in a downward direction than outward, e.g., it may be found in the sixth or even the seventh left intercostal space in the nipple line or outside it. When the enlargement is due to hypertrophy of the right ventricle, the cardiac impulse is displaced more in an outward direction than downward, and frequently there is also considerable pulsation in the epigastrium.

Where the cardiac impulse is thus displaced, before cardiac enlargement is diagnosed the possibility of its mechanical displacement by fluid or air in the right pleural cavity pushing it, or a retracted left lung pulling it, over to the left, must be excluded by careful physical examination of the front and the back of the chest. In the case of pleuritic effusion the dullness on the right side of the chest, and the absent or deficient vesicular murmur would point to fluid; in the case of retraction of the left lung the left side of the chest would be smaller, there would be deficient movement, dullness and deficient voice sound and vesicular murmur, or possibly bronchial breathing, consonating râles, and pectoriloquy over the left lower lobe.

The character of the impulse must be noted carefully, for, when forcible and heaving, it denotes hypertrophy; when feeble and diffused, dilatation.

The cardiac impulse is invisible and impalpable in some cases of enlargement of the heart, on account of emphysema of the lungs. In these circumstances even the *x*-rays may be required before one can be sure of the diagnosis of cardiac enlargement.

Careful mapping out of the area of cardiac dullness may afford valuable information as to the part of the heart involved in the enlargement. If the area of deep dullness is increased downwards and outwards, an increase in the size of the left ventricle is indicated; if upwards and to the right, hypertrophy of the right ventricle; if in all directions, enlargement of both ventricles.

Enlargement of the heart in children may produce definite local bulging of the chest wall in the cardiac area.

Having determined the position and character of the impulse, mapped out carefully the area of cardiac dullness, and thus arrived at the conclusion that the heart is increased in size, the next step is to determine not only what particular part is enlarged, but also the actual cause of the enlargement.

ENLARGEMENT OF THE LEFT VENTRICLE.

The left ventricle may become enlarged in:—

1. **Aortic Disease**:—Stenosis and regurgitation; regurgitation; stenosis; aneurysm of the first part of the aorta involving the aortic ring.
2. **Mitral Regurgitation**:—Disease of the mitral valve; dilatation of the left ventricle involving the mitral ring.
3. **Arteriosclerosis and Granular Kidney.**
4. **Alcoholism.**
5. **Long-continued Over-exertion**:—Athletes; workers at laborious occupations, e.g., stokers, firemen, furnacemen, blacksmiths.
6. **Exophthalmic Goitre.**
7. **Congenital Heart Disease.**

1. Aortic Disease.

Aortic disease may cause very great enlargement of the heart—cor bovinum or bovine heart. In the Guy's Hospital Museum there is a heart of this kind which weighs 53 ounces, the normal weight being about 10 ounces.

Stenosis and regurgitation is the commonest form of aortic disease, then regurgitation, and pure stenosis is the rarest.

Aortic Stenosis and Regurgitation.—The cardiac impulse is displaced downwards and outwards, and the cardiac dullness much increased towards the left.

It may be in the fifth, sixth, seventh, or even eighth space in or outside the left nipple line, and may be as far out as the anterior axillary line. The further the impulse is down the larger the left ventricle, and the further it is out the more the dilatation. When the impulse is forcible, heaving, and limited, it indicates that hypertrophy predominates ; when, on the other hand, the impulse is diffused and feeble, dilatation preponderates. Young people may present well-marked bulging in the precordial area.

A systolic thrill may be felt over the base of the heart, especially over the second right intercostal space close to the right border of the sternum. More rarely a diastolic thrill may be felt also or independently, either to the right or to the left of the upper part of the sternum.

On auscultation, a systolic and early diastolic murmur are heard over the base of the heart. The former usually replaces the first sound, is loudest in the second right intercostal space close to the sternum, and is transmitted upwards towards the clavicle and into the carotids. It varies in character, being in some cases soft and faint, and in others harsh, rough, and loud. The diastolic might be described as post-systolic, for it replaces the second sound ; it is generally soft and blowing, though in rare instances it is harsh or even musical. It may be heard over the upper part of the sternum and on both sides of it. When the aortic incompetence is due to fibrosis resulting from endocarditis following acute rheumatism or chorea, it is usually best heard to the left of the sternum, loudest in the third intercostal space close to the sternum. When the incompetence is due to syphilitic atheroma or to aneurysm of the first part of the aorta, the bruit is generally loudest and best heard in the second space to the right of the sternum. The early diastolic bruit which denotes aortic regurgitation may also be heard at the cardiac impulse, and in some cases may even be traced outwards into the left axilla. It cannot be mistaken for a mitral stenotic bruit, because there is no interval between the second sound and it. If there is complete compensation, the first sound may be loud and clear at the apex, but if dilatation of the left ventricle has occurred, there may be a loud blowing systolic murmur replacing the first sound and traceable outwards into the left axilla. Another bruit, which is rumbling in character and pre-systolic in time, may be heard at the cardiac impulse when the ventricle is dilated, the so-called *Flint's bruit* (*Fig.* 40, p. 95).

Patients are usually anæmic, and the carotid, brachial, and other superficial arteries are seen pulsating forcibly. A feeling of faintness on rising from the supine to the erect posture, dizziness, headache, a sensation of throbbing in the extremities, palpitation, dyspnœa, and precordial pain on exertion are early manifestations of this disease. As compensation fails, the dyspnœa and palpitation increase, œdema of the legs supervenes, pain becomes worse, and is felt not only over the region of the heart, but tends to radiate into the left shoulder and arm, and it may be followed by attacks of true angina pectoris.

The curious splashing or 'water-hammer' pulse is pathognomonic ; it is appreciated best if the radial pulse is felt when the arm is raised, the pulse-wave striking the finger with a sudden sharp jerk, and then as suddenly collapsing. When compensation fails, the pulse-rate may become rapid and the beats irregular and intermittent, as in mitral disease, but earlier in the disease the rate and rhythm are normal.

Capillary pulsation, which may be detected in the lips, finger-nails, and skin, is a very characteristic sign. It can be demonstrated by drawing a finger nail two or three times across the skin of the forehead or abdomen, so as to produce a line of hyperæmia, which, if watched carefully, will be seen to blush and pale alternately, each blush being synchronous with the pulse.

Aortic Regurgitation.—The symptoms are practically the same as in aortic stenosis and regurgitation, but there is no systolic thrill and no well-marked systolic bruit in the aortic area. The pulse is of the typical water-hammer type. The presence of a soft systolic bruit in the second right intercostal space close to the sternum does not indicate aortic stenosis unless there be at the same time a thrill there.

Aortic Stenosis is the rarest form of aortic disease. In addition to the absence of a diastolic bruit at the base, there is a pulse very different from that of the water-hammer type. If there is full compensation the pulse is slow, frequently below 60, and it may be only 40, or less, to the minute. It is usually regular, long sustained, and of good tension. A sphygmographic tracing shows a slow rise, often with an anacrotic break in the upcurve, a broad summit, and a gradual decline. The mere presence of a systolic murmur in the aortic area. even if its point of maximum intensity be in this region, is not sufficient evidence on which to base a diagnosis of aortic stenosis. A little roughening of a segment of the aortic valves, slight sclerosis of a valve, atheroma or dilatation of the first part of the aorta, and even anæmia, may give rise to a well-marked systolic bruit in this region. Before diagnosing aortic stenosis of clinical degree, one should have a big heart, a harsh systolic bruit in the aortic area, and a corresponding well-marked systolic thrill.

Aneurysm of the First Part of the Aorta is another important cause of hypertrophy of the left ventricle if the dilatation of the aorta involves the aortic ring, increases its circumference, and thus renders the aortic valves incompetent, though the cusps may be individually healthy. In addition to the characteristic pulse and the usual signs and symptoms of aortic regurgitation, there may be several indications which point to an aneurysm of the first part of the aorta as the cause of the aortic incompetence :—

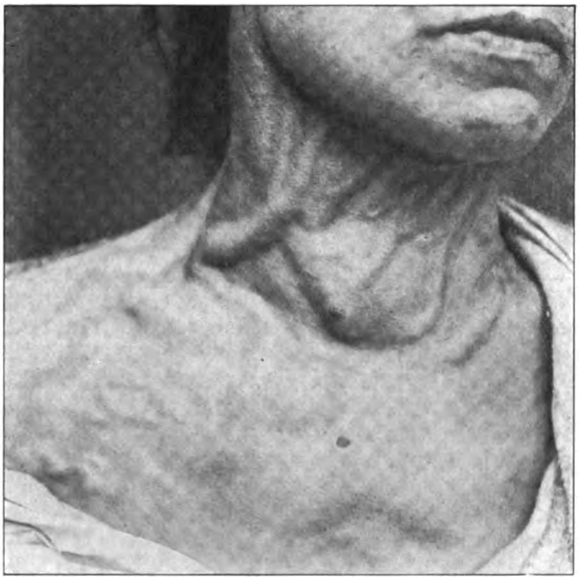


Fig. 99.—Obstruction to the superior vena cava by an aortic aneurysm ; collateral circulation through the distended superficial veins of the neck and thorax.

There may be a distinct bulging of the thoracic wall involving the first and second interchondral spaces close to the right border of the sternum.

There may be well-marked pulsation in the second right interchondral space and also in the adjacent spaces, according to the size of the aneurysm, close to the sternum ; when not obvious to the hand this may sometimes be detected by the ear laid flat on the chest.

In addition to an increase of the cardiac dullness downwards and to the left, there will be dullness in the second right space close to the sternum.

There may also be some signs of intrathoracic pressure :—

The right carotid pulse may be weaker than the left.

The face and neck may be deeply cyanosed if the aneurysm has extended outwards and has stenosed the superior vena cava, though this is a rare occurrence in this disease (*Fig.* 69, p. 158). There may be a loud systolo-diastolic bruit audible in the second right space over the superior vena cava, with maximum intensity an inch or more to the right of the sternum. The superficial veins over the upper part of the right side of the chest in front may be varicose (*Fig.* 99), and the direction of the blood-current in them may be from above downwards, instead of from below upwards.

The right bronchus may be stenosed if the aneurysm projects posteriorly, and this leads to impairment of percussion note and deficiency in the vesicular murmur over the upper lobe of the right lung. The *x*-rays might be used to determine the diagnosis (*Fig.* 100), though the aortic diastolic bruit should serve to distinguish aneurysm from new growth.

A diagnosis of aortic disease is incomplete until the actual cause of the lesion has been determined. It may be due to :—

1. *Lesions of the Valves* :—

Acute endocarditis
Fibrosis after former endocarditis
Infective endocarditis

Sclerosis due to : Strain (persistent), Syphilis, Alcohol
Rupture of a segment
Congenital malformation.

2. *Dilatation of the Aortic Ring from Aneurysm of the first portion of the Aorta.*

Lesions of the Valves.

Acute Endocarditis occurs most frequently as a complication of acute rheumatism, chorea, or scarlet fever. The indications of acute inflammation of the aortic valves will be a systolic murmur in the aortic area, and less commonly an early diastolic (post-systolic) murmur, which first becomes audible in the third left space close to the left border of the sternum. If the bruits are already present when the patient is first seen, it may be difficult to decide whether they are due to existing acute inflammation or to fibrosis after former inflammation. They may be noticed to arise whilst the patient is under treatment in bed for acute rheumatism, and then their acute nature will be obvious. In cases in which the bruits are due to acute aortic endocarditis and not to permanent fibrosis, the pulse will have little of the water-hammer type, the heart will not be much hypertrophied, though it may be dilated from acute rheumatic toxæmia, and the bruits will be found, as the days go by, either to diminish or increase in intensity, according as the inflammation of the valves resolves or passes on into permanent fibrosis.

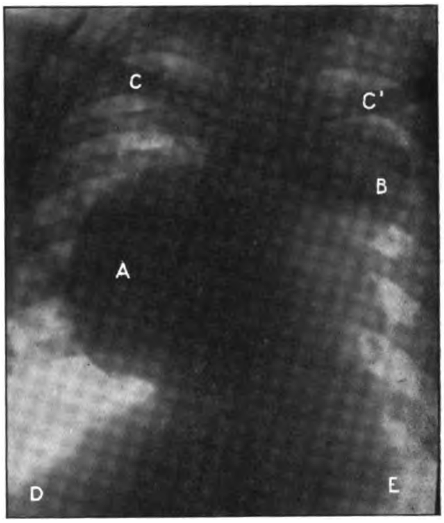


Fig. 100.—Skiagram of a large saccular aneurysm (A) of the ascending part of the arch of the aorta ; (B) transverse arch displaced to the left ; (C C′) clavicles ; (D) diaphragm ; (E) apex of left ventricle.—*By Dr. Alfred C. Jordan.*

Fibrosis from Previous Endocarditis.—When aortic disease is due to fibrosis from previous endocarditis, there will generally be a history of attacks of acute rheumatism, chorea, scarlet fever, or tonsillitis. The diastolic bruit which indicates the presence of aortic regurgitation is heard best along the left border of the sternum, the point of maximum intensity being in the third left intercostal space close to the left border of the sternum. There will generally be evidence of organic mitral disease at the same time, and if mitral stenosis be associated with aortic disease, whether there is a history of acute rheumatism or not, the valvular lesions may be considered without doubt to be due to the effects of former endocarditis. The patients are generally children or young adults, though a few survive into middle life.

Infective Endocarditis.—In this form of endocarditis, in addition to the signs and symptoms of aortic disease, there may be others, described on p. 34. In some cases bacteriological examination of the blood detects such organisms as the *Streptococcus pyogenes*, *Staphylococcus pyogenes aureus*, *Micrococcus rheumaticus*, *Pneumococcus*, or others.

Sclerosis not due to former Endocarditis :—

Strain.—Persistent strain is an important factor in the production of aortic disease. Occupations entailing long and continued manual labour, and excessive indulgence in athletics, may thus lead to incompetence. The tendency is not nearly so great, however, in those who have not had syphilis as in those who have ; so that sclerosis from strain alone must not be diagnosed unless there be neither a history nor evidence of rheumatism, chorea, syphilis, or alcoholism.

Syphilis.—A history of syphilis, and any manifestations of this disease in the form of pigmented scars on the legs, body, and face, ulceration of the tongue, patches of leukoplakia, ulceration, scarring, or perforation of the palate, necrosis of the nasal bones, etc., would point to this disease as the cause, and this conclusion would be strengthened if there were no previous history of rheumatism, scarlet fever, or chorea. The Wassermann reaction may be positive. The patients are nearly always males who have worked hard, and their first symptoms are often brought on by some undue muscular effort which strains the enlarged heart, or even bursts an atheromatous patch in the diseased valve. Uncommon before forty, the lesion is met with often enough between forty and fifty ; in many cases the heart has been passed as normal at forty, whilst at forty-five the aortic regurgitation is extreme. These patients often suffer from very severe attacks of angina pectoris, to which they are much more liable than are rheumatic aortic cases.

Alcohol.—The constant use of alcohol raises arterial tension and may be followed by sclerosis. The general appearance of the patient, and the signs described on p. 726, would suggest alcohol as the cause in the absence of any evidence of rheumatism or syphilis, but alcoholism without syphilis leads to definite aortic disease less often than it does to a generally hypertrophied heart, which sooner or later exhibits fibroid or fatty degeneration.

Rupture of a Segment of the Aortic Valve is a rare occurrence, usually brought about by some severe and sudden muscular exertion. The following is a good illustrative case of aortic regurgitation caused by rupture of a valve segment. A sailor, who had been examined just previously and passed as sound, was one day pulling on a rope, when suddenly the strain on it was unexpectedly and much increased. He made a tremendous effort to prevent the rope slipping through his hands, in doing so fainted, and was picked up in an unconscious condition ; on coming round he was very dyspnœic, and complained of pain in the precordial region. When the doctor examined him again he found a well-marked musical early diastolic murmur in the third and fourth left intercostal spaces close to the sternum and came to the conclusion that as his heart sounds were normal before the accident, he must have ruptured one of the segments of his aortic valve and thus caused the incompetence. There is always the probability of such a valve having been previously the site of syphilitic atheroma, without bruit, until the extra strain caused a weak spot to give way suddenly.

Congenital Malformations of the Aortic Valves are extremely rare, and they are to be diagnosed with great caution.

Dilatation of the Aortic Ring from Aneurysm of the first portion of the Aorta is nearly always due to syphilitic atheroma of the aortic walls, and in such a case it will be probable that there is syphilitic disease of the aortic valves themselves also. The dilatation of the aorta (' fusiform aneurysm ') will be indicated by definite impairment of note in the second right intercostal space near the sternum ; and the *x*-rays will confirm it. It will be next to impossible to assess with any degree of accuracy how much of the aortic regurgitation is due to the dilatation of the ring, and how much is due to the concomitant valve changes.

2. MITRAL REGURGITATION.

As a result of mitral regurgitation the left auricle becomes dilated and hypertrophied, the left ventricle dilated and hypertrophied, and later from backward pressure the right ventricle and auricle may be affected similarly. The chief symptoms are dyspnœa on exertion, palpitation, congestion of the face and lips, cough, possibly hæmoptysis, œdema of the feet and legs, and later albuminuria, ascites and enlargement of the liver. In the early stages the pulse may be regular, full, and of low tension. When compensation begins to fail, the pulse becomes rapid, irregular, and intermittent. The cardiac impulse is displaced downwards and outwards. It may be in the fifth intercostal space in the left nipple line, or outside it, or in the sixth space outside the nipple line. It is usually diffused, and there may be epigastric pulsation. There may be marked bulging of the precordial area in children. A systolic thrill is rare, but it may be felt at the cardiac impulse.

The cardiac dullness is increased outwards and downwards, but also upwards and to the right when the right side is involved.

At the impulse there is a systolic murmur, usually of a blowing character, which may either follow or replace the first sound. It is best heard at the cardiac impulse, but it can generally be traced outwards into the left axilla, can sometimes be heard behind at the inferior angle of the left scapula, and can also be traced inwards towards the left border of the sternum. The pulmonary second sound is accentuated or reduplicated in the second interspace close to the left border of the sternum.

When compensation fails, in addition to the above there may be :—

A systolic murmur, softer than and different in character from that at the impulse, over the lower part of the sternum and the fourth and fifth left interspaces, due to tricuspid regurgitation ; œdema of the feet, legs, and lower part of the body ; abdominal distention from ascites ; enlargement and pulsation of the liver ; signs of hydrothorax ; albuminuria.

A diagnosis of mitral regurgitation is incomplete by itself, for it may be due to different conditions. It is necessary to determine, if possible, the actual cause of the defect.

Causes of Mitral Regurgitation.

1. *Lesions of the Mitral Valve* :—

Acute endocarditis
Infective endocarditis
Fibrosis the result of former endocarditis.

2. *Dilatation, or Hypertrophy and Dilatation, of the Left Ventricle, without organic changes in the Mitral Valve itself* :—

Secondary to aortic disease
Secondary to increased systemic blood-pressure :—
    Chronic Bright's disease
    Arteriosclerosis.

3. *Diseases of the Myocardium and Pericardium* :—

Myocarditis
Fatty degeneration
Fibroid degeneration
Pericarditis
Adherent pericardium.

4. *Acute Dilatation of the Heart from* :—

Over-exertion
Acute febrile diseases
Acute nephritis.

Lesions of the Mitral Valve :—

Acute Endocarditis.—Simple acute endocarditis is not a disease *per se*, but occurs as a complication of some other disorder, especially acute rheumatism, chorea, and scarlet fever. It sometimes complicates tonsillitis, which is in many instances a manifestation of rheumatism occurring without any changes in the joints ; and in children acute endocarditis may be the only indication of an attack of rheumatism. There are no characteristic symptoms which point to acute endocarditis. If in the course of acute rheumatism the patient complains of a little palpitation, precordial pain, and distress, and it is found that the heart action has increased in rapidity without any increase in the joint affection, endocarditis should be suspected. The temperature chart seldom indicates the complication. At first the position of the cardiac impulse and the heart-sounds remain normal, but if watched from day to day, endocarditis having developed, the impulse will be found to have moved outwards, the first sound becomes prolonged and roughened, then doubled, and in a few days it is either followed or replaced by a localized soft blowing systolic murmur.

Fibrosis the result of Previous Endocarditis.—If acute endocarditis of the mitral valves does not resolve, the valve-flaps become sclerosed, and in the later stages even calcified. In many cases the circumference of the orifice is narrowed, so that the valve is not only incompetent but also stenosed. A diagnosis of fibrosis after endocarditis as the cause of mitral incompetence may be made if there is a previous history of acute rheumatism or chorea, and independently of such a history if there is evidence of stenosis as well as regurgitation. If actual mitral stenosis can be diagnosed with certainty, it must be due to fibrosis from endocarditis, though there may of course be recent endocarditis as well.

Infective Endocarditis of the mitral valve suggests itself if there is a mitral bruit, and if any of the symptoms and signs mentioned on page 34 are present at the same time.

Hypertrophy and Dilatation of the Left Ventricle.

Secondary to Aortic Disease.—Aortic disease leads to hypertrophy of the left ventricle, followed after a time by dilatation of that cavity and mitral regurgitation. Marked pulsation of the superficial arteries, a splashing pulse, capillary pulsation, and the systolic and early diastolic murmur at the base of the heart, the former best heard in the second right space close to the sternum, and the latter in the third left space close to the left border of the sternum, would indicate the presence of aortic disease. If the patient has suffered from either rheumatism or chorea, the mitral regurgitation might be due to primary endocarditis of the mitral valve, but if the aortic disease is the result of syphilis, hard work, or aneurysm of the first part of the aorta, then it may be assumed that the mitral regurgitation is the result of secondary dilatation of the left ventricle, and not of primary mitral disease.

Secondary to Increased Systemic Blood-pressure due to Chronic Bright's Disease.—Associated with the increased blood-pressure of chronic Bright's disease, the left ventricle hypertrophies first, and after a time, when compensation fails, dilates ; mitral regurgitation follows, and may be succeeded by all the signs of backward pressure, such as œdema of the feet and legs, ascites, enlargement of the liver, hydrothorax, hæmoptysis from congestion or infarction of the lungs, and so forth. A patient presenting such a group of symptoms may at a first glance be considered to be a case of primary disease of the heart, but a careful investigation will often enable one to determine that the primary changes have occurred in the kidneys. The radial artery may be thickened and tortuous, the tension of the pulse higher than in mitral regurgitation from primary heart disease ; there may be albuminuric retinitis and retinal hæmorrhages ; the urine is variable, for whereas it may formerly have been abundant, of low specific gravity (1008 to 1012), with only a trace of albumin, heart failure may lead to its being diminished in amount, of specific gravity 1020 or more, and albumin may be abundant ; microscopical examination, however, will generally reveal renal tube-casts.

Secondary to Increased Systemic Blood-pressure due to Primary Arteriosclerosis.—In this disease there may be signs of enlargement of the heart, mitral regurgitation, backward pressure, and a thickening of the arteries, but in contrast to chronic Bright's disease the urine will be of higher specific gravity, and there will be no albuminuric retinitis. It often becomes merely a matter of opinion, however, whether a given patient is suffering from arteriosclerosis or from granular kidney ; post-mortem examination may reveal both, or arteriosclerosis may predominate when granular kidney had been diagnosed, and vice versa.

Diseases of the Myocardium and Pericardium.

Myocarditis—Inflammation of the myocardium is associated most frequently with either pericarditis or endocarditis, but occasionally it may occur in acute rheumatism as a primary condition. In one form of the disease there is an infiltration of leucocytes between the muscular fibres—interstitial myocarditis ; in another form the actual muscle fibres are involved—parenchymatous myocarditis ; and there is a third variety which occurs in pyæmia, especially from bone disease, characterized by the formation of abscesses in the myocardium. The weakened condition of the heart muscle leads to dilatation of the ventricles, and thus to enlargement of the heart. When accompanied by pericarditis or endocarditis, the signs of myocarditis are overshadowed by the symptoms associated with these other conditions. The diagnosis of myocarditis is therefore a difficult matter. If in a case of acute rheumatism there is no evidence of either pericarditis or endocarditis, but there are signs of cardiac failure, a feeble irregular pulse, a good deal of precordial pain and distress, dyspnœa and palpitation, a tendency to sudden collapse, and signs of dilatation of the left ventricle, with a feeble cardiac impulse and a weak first sound, myocarditis may be suspected.

Fatty Heart.—The heart may be covered with fat (fatty superposition) ; fat may infiltrate between the muscular fibres (fatty infiltration) ; the muscle fibres may be degenerated, losing their striation, and containing fat granules (fatty degeneration) ; or all these conditions may be associated. Fatty degeneration may occur in patches or be general. When general, the heart becomes enlarged from dilatation as the muscle becomes flabby, has less contractile force, and is more yielding. It is a condition which may be associated with general obesity, severe anæmia, wasting diseases such as cancer, phthisis, phosphorus poisoning, and alcoholism. It may be a sequela of severe attacks of typhoid and other specific fevers. The symptoms and signs of the condition are due to the diminished contractile power of the ventricles which leads to dilatation. The pulse may be small, feeble, and slow—30 to 40 beats per minute—or it may be frequent and irregular. The cardiac impulse is very feeble or imperceptible. There may be an increased area of cardiac dullness from dilatation, and the first sound may be very faint. The patient is usually feeble and anæmic, and suffers from faintness or severe syncopal attacks which come on suddenly and are characterized by coma, convulsive twitching, and stertorous breathing. Œdema of the legs and venous congestion of the lips and face, which are common in valvular disease, are usually absent. There is dyspnœa on exertion, a feeling of coldness and depression, and a general impairment of the nutrition of the muscles which are soft, flabby, and diminished in power. In some cases attacks of cardiac 'asthma' in the early morning are complained of, and in the later stages of the disease there may be Cheyne-Stokes breathing. The chief diagnostic signs are the feeble cardiac impulse, the feeble pulse, and the weak first sound, associated with dyspnœa and attacks of syncope, and the absence of evidence of other causes for the heart symptoms.

Fibroid Heart.—Fibroid degeneration of the myocardium is usually associated with some obstructive lesion of the coronary arteries caused by syphilis. It may be general, or rarely localized to the apex of the left ventricle; in the latter case there may be thinning and weakening followed by aneurysm of the heart, and then by rupture. It is one of the causes of sudden death. The most important symptoms are: dyspnœa on slight exertion, palpitation, and precordial pain. The physical signs are those of dilatation of the left ventricle. The pulse is slow. and in late stages feeble and irregular. There may be severe attacks of angina pectoris. The diagnosis is more or less a matter of guesswork. Such signs and symptoms in a patient who has had syphilis, but neither acute rheumatism nor chorea. and who has neither aortic disease nor signs of granular kidney or arteriosclerosis, might be considered indications of this form of cardiac degeneration.

Pericarditis.—In pericarditis the cardiac impulse is usually displaced, and the area of cardiac dullness increased. These physical signs may be due to enlargement of the heart, or to effusion of serous fluid into the pericardial sac, and it is very difficult to differentiate between these two conditions. Enlargement of the heart due to dilatation is generally the result of the myocardium being affected as well as the pericardium, and the cardiac impulse is diffused and displaced outwards. If there is an effusion of serous fluid into the pericardial sac, it is said that the impulse is displaced *upwards* as well as outwards, so that it may be found on a level with, or above and external to, the left nipple, but this is a very unreliable sign. The dullness is increased laterally and upwards, and when carefully mapped out it is said to have a triangular shape, with the base on the diaphragm and a somewhat rounded apex pointing towards the left clavicle, and reaching to the second left intercostal space or higher. Percussion, however, is quite unable to distinguish between a pericardial effusion and a much enlarged heart without effusion. The intercostal spaces are filled out, and may be almost obliterated, so that the ribs feel much less prominent on this part of the chest. On auscultation, in addition to a systolic murmur at the impulse due to mitral incompetence from the accompanying dilatation of the left ventricle, a triple 'cantering' sound, and perhaps a definite rub, may be heard in some part of the precordial region, especially near the sternum, independently of respiration, and generally increased in intensity by firm pressure of the stethoscope. The rub is audible whether effusion is present or not.

Adherent Pericardium.—Adhesions between the visceral and parietal layers of the pericardium are found frequently post mortem when they had never been suspected during life. Sometimes, however, they are associated with chronic mediastinitis, or what should more correctly be termed mediastinal fibrosis, the outer surface of the pericardial sac becoming adherent to the thoracic wall and to adjacent structures. This condition usually leads to considerable hypertrophy and dilatation of the heart. There may be marked bulging of the precordial area to the left of the sternum. The cardiac impulse may be seen not only in the sixth space outside the left nipple line, but also in the fifth, fourth, and third left spaces, and the pulsation may extend in these spaces from the left border of the sternum to the left nipple line, or even outside that line. The impulse has a curious wavy character, and it may be noticed that coincident with the impulse in the sixth space there may be a systolic retraction of the spaces above, or of the lower ribs below and outside the cardiac area, best seen when the patient lies over to the other side with his left arm raised above his head. If the heart is adherent to the diaphragm, there may be a systolic retraction of the eleventh and twelfth ribs on the left side behind. Some cases of adherent pericardium of this type exhibit dilatation of the superficial veins in the precordial area. Diastolic collapse of the cervical veins is said to occur also. On rolling the patient from side to side it is found in many cases that the cardiac impulse remains nearly in the same position, not altering so much as it does in health under similar circumstances. The hand placed over the heart may feel a diastolic shock or rebound, which is regarded by some as a characteristic sign of the condition. On auscultation there may be a systolic murmur at the apex, indicative of mitral regurgitation, and frequently there is also a presystolic murmur due to a relative stenosis of the mitral orifice.

There is also a therapeutic sign which may help in doubtful cases. Mitral regurgitation in young people, if due simply to fibrosis of the valve after endocarditis, will usually improve under treatment by rest in bed and the administration of appropriate doses of digitalis. Where the mitral regurgitation, however, is associated with adherent pericardium, similar treatment has little effect, and very slight, if any, improvement follows. If, in a young person who is presumably rheumatic, the size of the heart and the symptoms are not easily accountable for by the extent of valvular disease suggested by the bruits, the patient probably has adherent pericardium with mediastinal fibrosis. The diagnosis, therefore, is guessed at rather than made.

Acute Dilatation of the Heart.

From Over-exertion.—Acute dilatation may result from over-exertion. For example, if a man who has been run down from excessive mental work, and in consequence is in poor condition or bad training, takes a holiday, and attempts the ascent of a mountain or engages in some violent form of exercise, his heart is very liable to give way under the strain. The chief indication of such an occurrence will be a feeling of pain, distress, and discomfort in the region of the heart, dyspnœa, and palpitation. The pulse will be rapid, weak, and irregular. The cardiac impulse will be displaced outwards, diffuse, weak and undulating in character, and although a maximum point of the impulse may be visible, it cannot be located clearly by palpation. There will be epigastric pulsation, the cardiac dullness will be increased outwards, and the first sound will be feeble, reduplicated, or replaced by a soft blowing systolic murmur.

From Acute Specific Fevers.—Similar signs and symptoms, especially weakness of the first sound, occurring in the course of diphtheria, typhoid fever, typhus, scarlet fever, erysipelas, and other fevers, would point to dilatation of the heart in consequence of the toxæmia producing loss of tone in the cardiac muscle from parenchymatous degeneration.

3. ARTERIOSCLEROSIS AND GRANULAR KIDNEY (see p. 14).

4. ALCOHOLISM.

Patients who have been addicted to alcoholism are liable to enlargement of the heart. It is a cause of which the importance is frequently overlooked. The usual signs of hypertrophy and dilatation may be present, with mitral and tricuspid incompetence and signs of backward pressure. The enlargement may be considerable. At a post-mortem examination it is by no means unusual to find the heart weighing as much as from 20 to 30 ounces. The valves are healthy, the aorta normal, and evidence of arteriosclerosis and granular kidney is absent. Alcoholism may be suspected as the cause of enlargement of the heart where there is no evidence of primary valvular disease, adherent pericardium, arteriosclerosis, or chronic Bright's disease. Other signs of alcoholism may also be present (p. 726).

5. LONG-CONTINUED OVER-EXERTION

produces hypertrophy of the ventricles ; for a considerable period there may be no symptoms, but after a time, when compensation fails owing to the hypertrophy being insufficient to continue the excessive work, dilatation is produced, and mitral incompetence and signs of backward pressure ensue. The subjects of this form of enlargement of the heart are usually either middle-aged men who are robust and healthy in appearance, but have had to follow for many years a laborious occupation entailing severe manual labour, or else young men of good physique who have indulged in excessive athletic exercises, such as rowing, football, boxing, and running, often with insufficient preliminary training. At first, palpitation, dyspnœa, and irregular cardiac action are noticed. Later the ventricles dilate and the mitral valves become incompetent, and all the signs of backward pressure may follow. Enlargement of the heart from this cause is much more liable to occur where the patient is accustomed to take a considerable amount of alcohol. As a cause of enlargement of the heart it should not be diagnosed until primary valvular disease, granular kidney, and arteriosclerosis can be excluded.

6. EXOPHTHALMIC GOITRE.

In this disease, moderate enlargement of the heart, as shown by the displacement outwards of the cardiac impulse and the increased area of cardiac dullness, is common, and is probably the result of the long-continued increased rapidity of cardiac action. It is rarely, however, the most prominent sign of the disease. It is distinguished from other forms of enlargement by the presence of tachycardia—the pulse-rate in a well-marked case varying between 120 and 160, or being even higher than this—the marked pulsation of the carotids and other superficial arteries, the exophthalmos, the enlargement and pulsation of the thyroid gland, the fine tremor of the extremities, the loss of weight, the excitability, and the pigmentation of the skin of the eyelids. There is very often a loud blowing systolic bruit in the pulmonary area, less often one at the impulse, but frequently one over the thyroid gland. Certain signs associated with the names of von Graefe, Stellwag, and Moebius are not of the least value in making the diagnosis.

7. CONGENITAL HEART DISEASE.

When there is a patent interventricular septum there may be considerable enlargement of the heart from hypertrophy and dilatation of both ventricles. It is frequently associated with some narrowing of the pulmonary orifice. In addition to the symptoms common to most forms of congenital heart disease, viz., cyanosis, clubbing of the fingers and toes, dyspnœa, and polycythæmia, the cardiac impulse will be displaced downwards and outwards, there will be epigastric pulsation, perhaps a prolonged systolic thrill, best felt over the third left intercostal space close to the sternum, an increased area of cardiac dullness in all directions, and a loud systolic murmur at the base of the heart, the point of maximum intensity being the third or fourth left intercostal space close to the left border of the sternum. It is often very difficult to say whether the lesion is pulmonary stenosis or patent interventricular septum. A well-marked thrill is associated more constantly with the former than with the latter, but the point of maximum intensity of the murmur produced by pulmonary stenosis is in the second left space, close to the left border of the sternum, whereas in patent interventricular septum the murmur is loudest lower down.

ENLARGEMENT OF THE RIGHT VENTRICLE.

When the enlargement of the heart is due to hypertrophy or dilatation of the right ventricle, the cardiac impulse is displaced outwards more than downwards, there is frequently well-marked epigastric pulsation, and the dullness is increased upwards and to the right rather than to the left. The causes of enlargement of the right ventricle are as follows :—

1. **Diseases of the Left Side of the Heart :—**
   - Mitral stenosis
   - All the conditions which cause enlargement of the left ventricle (p. 206).
2. **Diseases of the Lung :—**
   - Fibroid lung
   - Chronic bronchitis and emphysema.
3. **Diseases of the Right Side of the Heart :—**
   - Congenital pulmonary stenosis
   - Pulmonary incompetence : (i) Due to dilatation of the pulmonary artery ; (ii) Due to infective endocarditis of the pulmonary valve.

1. DISEASES OF THE LEFT SIDE OF THE HEART.

Mitral Stenosis.—This is a common and most important cause of enlargement of the right ventricle. The obstruction to the flow of blood from the left auricle into the left ventricle leads to hypertrophy and dilatation of the left auricle, passive congestion of the lungs, red and brown induration of these organs, thickening dilatation and atheroma of the branches of the pulmonary arteries in the lungs as a result of the increased tension in these vessels. All these changes increase the amount of work to be performed by the right side of the heart, and are responsible for the hypertrophy of the right ventricle, by which means compensation may be maintained for some time. When the right ventricle dilates, compensation fails. In the early stages the pulse shows little variation from the normal, and there may be no obvious symptoms pointing to the existence of mitral stenosis. In more advanced phases the pulse becomes rapid, small, and irregular. The cardiac impulse is displaced outwards, and pulsation occurs in the epigastrium and in the third, fourth, and fifth intercostal spaces close to the sternum. On placing the palm of the hand over the region of the cardiac impulse and the adjacent fourth and fifth intercostal spaces, a characteristic thrill may be felt. It usually has a curious rough grating quality. It is diastolic in rhythm, and may be felt to terminate suddenly in a sharp shock which is synchronous with the apex beat. The dullness is increased upwards from the third left rib to the second, or even higher ; it extends well to the right of the sternum, but it does not reach far to the left, though in a few cases it extends to the left nipple line, even when mitral stenosis is the only lesion present. The more the dullness extends to the left, however, the less likely is the diagnosis of mitral stenosis alone to be correct. At or just inside the cardiac impulse, a loud, rough, rumbling, vibrating bruit may be heard, which runs up to, and is continuous with, a loud, accentuated, slapping first sound, which may or may not be followed by a systolic murmur. This characteristic bruit may occupy the whole of the diastole, and may commence with a doubling of the second sound. It increases in intensity until it finally ends in the loud first sound. It may, however, be shorter, and commence in the middle or latter part of diastole. It is usually termed presystolic, as it runs up to and is continuous with the first sound. The other abnormal signs to which mitral stenosis may give rise are described on pages 93 and 94.

All the Conditions which cause Enlargement of the Left Ventricle.—Whenever compensation begins to fail in cases of mitral regurgitation from any cause, aortic disease, enlargement of the left ventricle from chronic Bright's disease, arteriosclerosis, alcoholism, or other causes discussed above, and there is backward pressure through the lungs, hypertrophy of the right ventricle serves to maintain compensation for a time. The increase in the size of the right ventricle would be indicated by the advent of epigastric pulsation and a further increase of the dullness to the right of the sternum, but the diagnosis of its cause would rest upon data already discussed under the heading of mitral regurgitation (see p. 210).

2. Diseases of the Lung.

Fibroid Lung gives rise to symptoms and physical signs so characteristic that there is rarely any difficulty in making a diagnosis. The hypertrophy of the right ventricle is of secondary importance, and does not become manifest until late in the disease. The retraction of the lung draws the heart over towards the affected side, and in consequence of the displaced cardiac impulse and the increased area of pulsation, it may appear to be much larger than it really is. When the right lung is affected, there may be well-marked epigastric pulsation, and the cardiac impulse may be to the right of the sternum in the fifth intercostal space, the maximum point being in some cases as far out as the right nipple line. When the left lung is affected, the heart may be pulled over towards the left, so that the cardiac impulse is situated in the anterior, or even in the mid-axillary line. In consequence of the shrinking of the lung, more of the anterior surface of the heart will lie in contact with the thoracic wall, and there may be therefore an increased area of visible pulsation in the second, third, or fourth intercostal space. In addition to displacement of the cardiac impulse, there is diminution in the size and decrease in the movement of the affected side of the chest, the shoulder is drawn down, the spine curved with the concavity towards the affected side ; there is increased tactile vocal fremitus, impairment of note on percussion, and possibly a cracked-pot sound ; and, should there be dilated bronchial tubes, there are cavernous or amphoric breathing, bronchophony, pectoriloquy, and loud crackling râles. With the exception of compensatory emphysema, there may be no sign of disease in the other lung, a point which helps to distinguish this condition from phthisis. The chief symptoms are chronic cough, dyspnœa, abundant expectoration on rising in the morning, the sputum often being fœtid on account of the bronchiectasis so frequently associated with fibroid lung. The patient may be well nourished and show no signs of loss of flesh. Hæmoptysis occurs occasionally, but no tubercle bacilli will be found in the sputum. There is often extreme clubbing of the fingers.

Chronic Bronchitis and Emphysema may so increase the volume of the lungs that they cover the anterior surface of the heart completely ; consequently the cardiac impulse may be invisible, the superficial cardiac dullness diminished or absent, and the heart sounds faint or even inaudible. In these circumstances it is not an easy matter to diagnose enlargement of the heart. Should there be dilatation of the right ventricle as well as hypertrophy, and also tricuspid regurgitation, a systolic murmur may be heard over the lower part of the sternum and in the fourth and fifth left intercostal spaces close to the sternum, and œdema of the legs, ascites, enlargement of the liver, and albuminuria may also be present. If, in addition, there are signs of pulmonary emphysema, viz., the cubical chest, wide epigastric angle, increased tactile vocal fremitus, hyper-resonant percussion note, diminished area of hepatic and cardiac dullness, increased voice sounds, diminished vesicular murmur with prolongation of the expiratory sound, with or without non-consonating râles and rhonchi, and if there are no indications of fibrosis of the heart valves from former endocarditis, chronic Bright's disease, or primary arteriosclerosis, enlargement of the heart with failure of compensation as a result of chronic bronchitis and emphysema may be diagnosed.

3. Diseases of the Right Side of the Heart.

Pulmonary Stenosis.—This is the commonest form of congenital heart disease. In addition to cyanosis, clubbing of the fingers and toes, polycythæmia, dyspnœa, and signs of hypertrophy of the right ventricle, there is usually a well-marked systolic thrill over the second left intercostal space close to the sternum, and a loud, rough systolic murmur in the same position. The murmur is not transmitted to the carotids in the neck, as is that of aortic stenosis.

Pulmonary Incompetence.—This lesion may be associated with congenital pulmonary stenosis, or may be due to infective endocarditis (especially gonococcal), but by far the commonest cause is functional incompetence from dilatation of the pulmonary artery and orifice secondary to the high tension produced in the pulmonary circulation by mitral stenosis. It may be difficult to distinguish from aortic regurgitation ; the early diastolic bruit of pulmonary incompetence is most audible, however, in the third and fourth left intercostal spaces midway between the left nipple line and the left border of the sternum, whereas in aortic disease the diastolic bruit is usually heard best in the third left space close to the left border of the sternum. The visible pulsation of the superficial arteries, and the collapsing pulse, which are so characteristic of aortic incompetence, are not present in cases of pulmonary incompetence. *Herbert French.*

ENLARGEMENT OF THE KIDNEY.—(See Kidney, Enlargement of, p. 352.)

ENLARGEMENT OF THE LIVER.—(See Liver, Enlargements of the, p. 366.)

ENLARGEMENT OF THE LYMPHATIC GLANDS.—(See Lymphatic Gland Enlargement, p. 376.)

ENLARGEMENT OF THE SALIVARY GLANDS (See Swelling of the Salivary Glands, p. 694).

ENLARGEMENT OF THE SPLEEN.—(See Spleen, Enlargement of, p. 628.)

ENLARGEMENT OF THE THYROID GLAND.—(See Thyroid Gland Enlargement, p. 721.)

ENOPHTHALMOS (or Retraction of the Eyeball).—This may occur : (1) In wasting diseases ; (2) In paralysis of the cervical sympathetic ; (3) In various congenital affections.

The enophthalmos in *wasting diseases* is due to the absorption of the orbital fat, and the diagnosis as regards the eye presents no difficulty.

Enophthalmos due to *paralysis of the cervical sympathetic* is always associated with the other well-defined symptoms of this condition, namely, diminution in the size of the palpebral aperture, constriction of the pupil, and absence of sweating and blushing on the paralyzed side. The pupil is constricted owing to the paralysis of the dilator fibres, the pupil therefore not dilating in a feeble light.

In certain *congenital cases* there is well-marked retraction associated with defective or irregular movements of the affected eyeball. The ocular muscles are, as a rule, inserted much farther back in the sclerotic than is normally the case. The condition appears to be due to the absence or defective insertion of the extrinsic muscles of the eye, and may be recognized by its existence since birth. *Herbert L. Eason.*

ENURESIS occurs almost exclusively in children, and although most frequently confined to the night, it may occur in the day. It must be distinguished from incontinence of urine ; the patient has usually full control of micturition during the day, although sometimes the desire to urinate must be satisfied quickly or a little dribbling may take place. The child completely empties the bladder, often without waking, once or several times during the night. The bladder need not be quite filled for micturition to occur, for it takes place in the early hours of the night.

Enuresis is often accompanied, and may be caused, by slight affections, such as phimosis, balanitis, small urinary meatus, vulvitis, constipation, or intestinal worms, the correction of which remedies the trouble, but in other cases there seems nothing to promote the excitability of the detrusor muscle. It is cured not infrequently by an operation for the removal of enlarged tonsils and adenoid growths, or after the administration of small doses of thyroid extract. It has been stated that the condition is due to faulty development or deficient innervation of the sphincter muscle, or to spasm of the detrusor ; but this is difficult to prove. If the sphincter muscle were paralyzed or deficient, there would be true incontinence of urine, whereas this is not so, and the children are often of good development and health. It is probable that the infantile condition in which the detrusor muscle holds the mastery over the sphincter persists, a relative disparity between the innervation of the two sets of muscles allowing the detrusor, which normally is held in check by the sphincter, to overcome the comparatively weak action of the latter. When enuresis persists throughout childhood, it may disappear at puberty, when the prostate gland enlarges and strengthens the action of the sphincteric apparatus.

It is important to exclude both pyelitis, phosphaturia, and oxaluria before a diagnosis of simple enuresis is made. In either case nocturnal micturition may be the chief symptom ; microscopical examination of the centrifugalized deposit will detect the pus cells or the excess of calcium oxalate crystals, and a bacteriological examination of a specimen of urine passed directly into a sterile bottle or obtained by a catheter should be made in order to diagnose or exclude coli bacilluria (p. 69).

In most cases no source of irritation, alteration in the urine, or disease of the bladder can be found. The child is nervous and sensitive from a feeling of shame due to attempts by the parent to cure the trouble by punishment. In some the enuresis may accompany a minor epileptic attack, in which case there may be longer intervals than is usual in simple enuresis, or there may be a history of epilepsy, insanity, or other nerve trouble in the parent. *R. H. Jocelyn Swan.*

EOSINOPHILIA denotes a relative increase in the coarsely granular eosinophile cells of the blood (*Plate II, Fig. L,* p 22); it is determined by preparing blood films and making a differential leucocyte count. Normally, the coarsely granular eosinophile cells vary from 0 to 2 per cent ; the point at which eosinophilia begins is quite arbitrary ; but one may say that although it is unusual, under perfectly healthy conditions, to find more than 2 per cent of these cells in the differential count, they should reach 5 per cent or more before the term eosinophilia is applied to the condition. It is probable that some normal people have upwards of 5 per cent of these cells, but beyond this point they are nearly always pathological.

One may divide the causes of eosinophilia under main headings as follows :—

1. Conditions in which Eosinophilia is slight, inconstant, and of little diagnostic significance :—

Post-febrile states, after :—

Scarlet fever
Pneumonia
Acute articular rheumatism
Measles
Varicella
Malaria.

Affections of the bone-marrow :—

Splenomedullary leukæmia
Sarcoma of bone
Rickets
Osteomyelitis
Osteomalacia

Addison's disease.
From certain chemicals, particularly camphor, sulphuretted hydrogen.
In ovarian maladies.
Gonorrhœa.
During the positive stage of tuberculin reaction.
Some cases of malignant disease, especially when there are metastases—carcinoma, lymphosarcoma.

2. Conditions in which Eosinophilia may be marked.

(*a*). *Spasmodic Asthma.*

(*b*). *Certain Skin Diseases*, more particularly the bullous dermatoses :—

Pemphigus
Erythema bullosum
Dermatitis herpetiformis (Dühring's disease)
Hydroa
Herpes iris, or erythema iris
Herpes gestationis

It is much rarer in other cases of skin disease, but is noted occasionally in psoriasis, eczema, and exceptionally in some other affections of the skin.

(*c*). *Certain Parasitic Affections*, particularly :—

Ankylostomum duodenale
Bilharzia hæmatobia
Bothriocephalus latus
Tænia solium
Tænia mediocanellata
Filaria sanguinis hominis
Trichina spiralis

It is much less constant, and indeed generally absent, in cases of :—

Ascaris lumbricoides
Trichocephalus dispar
Oxyuris vermicularis
Pediculus capitis
Pediculus pubis
Pediculus corporis
Acarus scabiei

The list above almost speaks for itself, and little discussion is needed. None of the conditions named is necessarily associated with eosinophilia, but the coarsely granular eosinophile cells often reach a figure between 5 and 15 per cent in the differential count in many of the diseases that come under headings (*a*), (*b*), and (*c*), whilst sometimes during paroxysmal asthma they may reach 25, 50, or even more per cent, and they are often over 20 per cent in the severer forms of parasitic disease. The eosinophilia of leukæmia has often had stress laid upon it in text-books, but as a matter of fact, although the coarsely granular eosinophile cells per cubic millimetre of blood may be considerably above the normal along with all the other corpuscles, yet when reduced to percentages in the differential leucocyte count, the eosinophile corpuscles seldom number more than 2 or 3 per cent of all the white cells present.

The value of eosinophilia in discriminating between artificial bleb-formation and a true *bullous dermatosis* is mentioned in the article upon BULLÆ (p. 96).

The difficulty sometimes present in deciding whether in a given case the lesion is primary emphysema and bronchitis, or primary *asthma* succeeded by emphysema and bronchitis, is discussed on page 535 ; and the value of eosinophilia in discriminating between truly asthmatic cases and those which simulate asthma but are really cardiac, renal, or bronchitic, is there referred to. It should be noted that the eosinophilia is not confined to the blood, being present also in the cells in the sputum ; it occurs during the paroxysms of asthma, and rapidly disappears in the intervals.

When a patient is suffering from an obscure form of anæmia, and when the blood at the same time exhibits considerable eosinophilia, the latter may sometimes be the first suggestion that there is a serious *parasitic infection* in the case, and careful examination of the fæces or urine for the parasites themselves or for their ova, with the administration of anthelmintic drugs, may then be resorted to for confirmation of the diagnosis (see PARASITES, INTESTINAL, p. 519). Persons who have been resident in the tropics are more liable to unsuspected infection of this kind than are others. *Herbert French.*

EPIPHORA, or overflow of the tears, may be due to (1) *Increased secretion* ; (2) *The puncta lachrymalia not being in close apposition to the globe* ; (3) *Obstruction of the lachrymal canaliculi or duct.*

1. The most familiar cause of epiphora due to increased secretion of tears is the act of *weeping*, in which the flow is due to psychical stimuli. Epiphora may also occur in the lachrymation caused by *conjunctivitis*, *corneal ulcers*, and other inflammatory affections of the eye (p. 231).

2. Tears only find their way down the canaliculi by capillary attraction, the puncta lachrymalia being applied closely to the surface of the globe. In *facial paralysis*, owing to the failure of the orbicularis palpebrarum muscle, the lids are no longer braced up against the eye, and the lower lid droops away from the globe. The tears collect in the sulcus thus formed, and run over on to the cheek. The condition is easily diagnosed by the inability to close the eye entirely, either by passive or active movements. In cases of *chronic marginal blepharitis*, hypertrophy of the lid-edge and the conjunctiva results in a slight eversion or ectropion. The punctum lachrymale of the lower lid is no longer in apposition with the eye, and epiphora follows, causing continual moisture of the edge of the lids and aggravation of the original condition. Cicatricial ectropion from *burns*, *injury*, *sclerodermia*, or *lupus* of the cheek may also result in epiphora ; and so may severe *proptosis* (see EXOPHTHALMOS, p. 229), resulting from tumours or inflammation at the back of the orbit, or from Graves's disease.

3. The lachrymal ducts may be *congenitally obstructed.* The obstruction is usually unilateral, and is due to a plug or septum of uncanalized epithelium situated in the lower part of the duct. The epiphora is as a rule not evident till the seventh or eighth day, at which period the infant first begins to shed tears, and owing to the suppuration of the tears collected in the lachrymal sac the malady may be mistaken for a chronic conjunctivitis. The unilateral nature of the affection, and the presence of tears or pus in the sac, are the diagnostic signs, and the obstruction may generally be cured by a single probing of the duct through the dilated but uncut canaliculus. Congenital absence of one or both canaliculi has been recorded. Stenosis of the lachrymal duct may also occur as the result of *catarrhal congestion* of the mucous membrane, or from some organic obstruction, due to *cicatrization* following abscess in the lachrymal sac or necrosis of the bones forming the walls of the duct. The diagnosis can only be made by syringing through the canaliculi ; in catarrhal obstruction, fluid can usually be forced into the nose, but in organic stricture it is returned through the other canaliculus. In such cases the stenosis can be relieved by the passage of a probe, after slitting the lower or upper canaliculus, or by various operations, which are now on their trial, for the formation of a permanent direct opening from the lachrymal sac into the nasal cavity.

Excision of the lachrymal sac for chronic suppuration is always followed by epiphora, but this condition may often be preferable to the discomfort caused by recurrent lachrymal abscess and to the risk of corneal ulcer with hypopyon.

Injury to the duct or canaliculus may also cause permanent epiphora. *Herbert L. Eason.*

EPISTAXIS—rhinorrhagia, or bleeding from the nose, may be due to local or general causes, or to a combination of both. In many cases it occurs spontaneously and no cause can be indicated.

Local Causes.

Injury.—A blow, fracture of the base of the skull, a foreign body in the nose, operation on the nose, violent coughing, sneezing or nose-blowing, nose-picking.

Ulceration.—Traumatic, syphilitic, malignant, tuberculous, leprous.

New Growth.—Adenoid growths, polypi, fibroma, angioma, malignant disease.

Varicosity of the Veins of the nasal mucosa : multiple hereditary telangiectases.

Acute Infective Inflammation.—Severe catarrh, diphtheria, scarlet fever, influenza.

General Causes.

High Arterial Blood-pressure, such as obtains in granular kidney and chronic renal disease, arteriosclerosis, gout, cirrhosis of the liver, heart-disease.

High Venous Blood-pressure in bronchitis, emphysema, dilatation of the right heart ; in cerebral congestion, when blood passes from the superior longitudinal sinus by an emissary vein going through the foramen cæcum to the nasal mucosa : in 'determination of blood to the head' ; in schoolboys and children after taking violent exercise.

Altered Conditions of the Blood.—Hæmophilia, pernicious anæmia, purpura, scurvy, leukæmia, chlorosis, jaundice, and the onset of acute specific fevers, particularly enteric, scarlet fever, and measles.

Alterations in Atmospheric Pressure.—Mountaineering, diving, caisson disease.

Epistaxis of Obscure Origin, often attributed to congestion, and occurring :—In childhood ; at puberty, especially in girls ; as the alleged vicarious menstruation ; as the result of sexual irritation in either sex ; in women at the menopause.

In some cases the blood issues from both nostrils ; in the majority, particularly when the cause of the bleeding is local, from one only. But it must be remembered that nose-bleeding may occur without any blood coming from the anterior nares ; if the patient is lying down the effused blood runs down the sides or floor of the nose, passing through the posterior nares and entering the nasopharynx ; when this occurs the patient may cough and spit it up, when hæmoptysis will be observed ; if, on the other hand, he swallows the blood, he may vomit it later, when hæmatemesis will take place. In the not uncommon instances in which either of these events occurs from epistaxis, careful enquiry should suffice to make the diagnosis clear ; but it should not be forgotten that either hæmatemesis or hæmoptysis may indicate nothing more serious than an attack of nose-bleeding.

In every case of epistaxis, the history of the attack should be gone into carefully. Particular enquiry should be made as to any sort of trauma that might account for it, and also as to the occurrence of previous attacks of nose-bleeding. More important still is a careful examination of the local conditions of the nose, with use of a nasal speculum to dilate the nares, and of a mirror and lamp to secure a good illumination. In many cases the bleeding point can be seen, whether the hæmorrhage be arterial or venous, the so-called 'seat of election' of epistaxis being a small and perhaps ulcerated spot on the cartilage of the septum not far from its junction with the ethmoid and vomer. In other instances no such bleeding point can be seen, the blood oozing from the mucous membrane generally. The urine should be tested for albumin and the arterial blood-pressure measured instrumentally.

Recurrent Epistaxis at irregular intervals is likely to be due to some local cause. For example, a small ulcer on the septum nasi, due perhaps to injury in the first instance, may scab over from time to time but never heal satisfactorily ; a comparatively trifling injury, such as that occasioned by blowing the nose, may suffice to detach the scab, epistaxis following. Malignant disease of or about the nose, and also adenoid vegetations, often give rise to repeated nose-bleeding. Epistaxis has been a prominent symptom in the rare hereditary disease in which numerous friable telangiectases appear about the surfaces of the body and on mucous membranes.

Considerable aid in diagnosing the probable cause of an epistaxis is afforded by the age of the patient. In *infancy*, the cause is likely to be local injury by a fall, a foreign body, the habit of nose-picking, or syphilitic disease of the nasal bones. In *childhood*, falls and blows on the nose are common, the temptation to insert foreign bodies up the nose still asserts itself, adenoid growths in the nasopharynx are common ; and general causes such as heart-disease, diseases of the blood, or obscure conditions of local congestion, may exist and account for the onset of epistaxis. About the age of *puberty* nose-bleeding may occur in either sex, and particularly in girls, not only in consequence of the causes enumerated already, but also spontaneously. In the healthy, or apparently healthy, *young adult*, almost any of the list of local and general causes may account for nose-bleeding ; diagnosis here must rest upon the results of the examination into the local conditions of the nose, and the general state of the organs of the body. In the *old*, on the other hand, and in middle-aged patients of plethoric habit, high blood-pressure with or without general arterial disease is the most important cause of epistaxis ; it may be a natural remedy for the plethora from which such persons suffer, and not infrequently does relieve them from such symptoms as a sense of fullness and congestion of the head, tinnitus aurium, or the appearance of flashes of light or muscæ volitantes before the eyes. In other instances, it may serve as a warning, drawing attention to the abnormally high blood-pressure and to the chronic interstitial nephritis or arteriosclerosis that underlies it.

A. J. Jex-Blake.

ERUCTATIONS.—(See FLATULENCE, p. 240 ; and HEARTBURN, p. 296.)

ERUPTIONS, BULLOUS, VESICULAR, Etc.—(See BULLÆ, VESICLES, ETC.)

ERYTHEMA signifies a pathological reddening of the skin from vascular hyperæmia, the redness disappearing on pressure, to return when the pressure is removed. There is no strict line at which one can say mere redness of the skin ends and actual erythema begins. For instance, FLUSHING (p. 241) would hardly merit the term erythema in some cases, though it would in others ; clinically, however, there is seldom difficulty in deciding what is erythema and what is not. It may be local or general, and may be due to many causes, including the following :—

1. **Drugs.**

(*a*). *External Applications, including all Rubefacients* :—

- Turpentine
- Poultices
- Mustard plasters
- Ammonia
- Croton oil
- Cantharides
- Capsicum
- Carbolic acid
- Mineral acids
- Alkalies
- Alcohol and alcoholic preparations
- Chrysarobin
- Oil of cade
- Strong mercurials
- Iodoform
- Arnica
- Mesotan
- Methyl-salicylic acid
- Certain soaps
- *X*-ray dermatitis
- Radium applications.

(*b*). *Medicines taken by the Mouth* :—

- Copaiba
- Cubebs
- Sandalwood oil
- Belladonna
- Atropine
- Salicylates
- Aspirin
- Sulphonal
- Veronal
- Trional
- Chloral
- Chloral hydrate
- Butyl chloral hydrate
- Chloralamide
- Antipyrin
- Arsenic
- Quinine
- Boric acid
- Rhubarb
- Benzoic acid
- Mercury
- Bromides
- Iodides
- Pilocarpine
- Valerian
- Chlorate of potash

(*c*). *Therapeutic Agents injected Hypodermically* :—

- Normal horse serum
- Antidiphtheritic serum
- Anti-anthrax serum
- Antistreptococcus serum
- Antitetanic serum
- Antipneumococcus serum
- Salvarsan
- Neosalvarsan
- Atoxyl
- Sodium cacodylate.

(*d*). *Therapeutic Agents injected per Rectum* :—

- Soap and water enemata
- Other enemata

2. **Erythema Artefactum (Malingering).**

3. **Irritants affecting Workers at Certain Trades,** in which they have to handle or come in contact with erythema-producing substances, such as :—

- Turpentine
- Varnish
- Aniline dyes
- Volatile oils
- Scents
- Fishermen handling jelly-fish, sea cats, weevers and other fish
- Tar products
- Resins
- Vanilin
- Satin-wood sawdust
- Leaves of Primula obconica
- Leaves of Rhus toxicodendron
- Juice of knot-grass (Polygonum aviculare)

4. **After Operations sometimes,** perhaps the result of the anæsthetic.

5. **Extremes of Heat or Cold** :—

- Erythema solare
- Erythema ab igne
- Erythema a frigore

6. **Around Inflammatory Foci,** such as :—

Over any abscess as it nears the surface—appendicular, mastoid, dental, hepatic, axillary, inguinal, a pointing empyema, and so on.

Round superficial inflammations of the skin, such as boils, carbuncles, furuncles, malignant pustules, and so on ; or as part of other skin lesions, such as eczema, lupus erythematosus, urticaria, ringworm, tinea versicolor, erythema iris, hydroa, erythema nodosum, phlebitis, cellulitis, lymphangitis.

7. **As part of a General Illness,** in which other symptoms are likely to be even more prominent :—

- Cerebrospinal meningitis
- Ptomaine poisoning
- Malaria
- Leprosy
- Dengue.

8. **As a Prominent Symptom of a Disease** which may or may not present other symptoms at the time.

(*a*). *Localized Erythema* :—

Erysipelas
Small-pox
Gout
Erythromelalgia
Raynaud's disease
Erythema induratum (Bazin's disease)
Polymyositis
Trichinosis
Rat-bite fever
Pellagra
Meige's disease
Angioneurotic œdema.

(*b*). *Generalized Erythema* :—

Scarlet fever
Measles
German measles
'Fourth' disease
Acute rheumatism
Parasitic toxæmias, e.g., from
  Hydatid disease
  Tapeworm
Filariasis
Trypanosomiasis.
Snake bite

9. **Generalized Erythema without obvious cause** :—

Erythema simplex
Erythema multiforme
Erythema exfoliativum seu scarlatiniforme.

In arriving at the cause of erythema in any particular case, the diagnosis is very often obvious when the possibilities are borne in mind. Indeed, many of the conditions mentioned in the list above do not require any further discussion. The appearance of the part affected will often suggest that some external application is the cause: vesiculation may result from almost any of the substances which, in weaker solution, produce erythema only; there is nothing pathognomonic about the naked-eye appearances from which to tell the application used; the history as to what the patient has been applying to the skin is needed; and if malingering by surreptitious application is suspected, the cessation of the lesions when the patient is placed under circumstances where further applications are not possible will confirm this. One point of importance is that the red patches produced by carbolic acid may be such as to simulate tinea circinata; but the absence of spores upon the hairs removed from the patch when they are examined microscopically will exclude this.

Local reaction from the application of *x*-rays on repeated occasions is familiar; it differs from *x*-ray cancer in that, though the erythema may persist for weeks, or may develop into a vesicular eruption (*x*-ray dermatitis or *x*-ray burn), which may be very resistant to treatment, it ultimately subsides, leaving a brown pigmented stain, whereas *x*-ray cancer progresses in spite of cessation of the use of the rays, leads to progressive if slow destruction of the affected parts, and ultimately behaves like an ordinary epithelioma.

Radium burns are less common than they used to be, because of greater knowledge of the methods of screening the skin from the effects of the superficial rays by means of lead or silver sheets; but most radium applications are followed by some degree of local reaction, of which erythema is a prominent feature, for a week or ten days, after which the reaction subsides rapidly though often followed by local brown pigmentation of the skin.

The phenomena of *serum reactions* are familiar; the effects are due to the serum itself rather than to the antitoxin it contains, and they form part of what is known as anaphylaxis. The injection of any foreign proteid into the system is followed by chemical reactions in the body antagonistic to the injected proteid, and when these reactions are at their height the body is extra-sensitive (anaphylactic) to a further injection of the same foreign proteid. In the case of horse-serum this state of anaphylaxis reaches its height about eight or nine days after the original injection; it is at this time that the symptoms of 'serum disease' show themselves clinically. The patient begins to ache all over, with more or less acute pains in the back and limbs, sometimes referred particularly to the joints; headache is usual, and there may be vomiting; the temperature rises moderately, the tongue is coated, and appetite fails; at the same time a blotchy red eruption appears upon the skin, sometimes universally, but generally with maximum intensity round the site of the inoculation. There is intense itching, the patient may not be able to sleep, and may not have enough fingers to scratch himself with. The eruption is sometimes a pure urticaria, but quite as often generalized erythema preponderates, with multiple urticarial wheals amongst the erythema. Vesication is uncommon. These symptoms last but a day in mild cases,—two, three or even four days in others. They nearly always subside spontaneously, but in a few instances the serum reaction has been so intense as to prove fatal.

The erythema which sometimes follows the injection of *salvarsan*, *neosalvarsan*, *atoxyl*, or *sodium cacodylate* may be severe for a time, but it is usually transient, and it only develops in a small minority of cases. It is probably due to the arsenic which is present in relatively high proportions in organic combination in these drugs.

The erythema that may follow *enemata* is generally universal, and for the time being the patient looks very much as if he had scarlet fever. Indeed, the physician may be unable at the moment to make sure that it is not scarlatina, especially if the case is febrile already. The erythema disappears in about twenty-four hours or less, is not accompanied by vomiting, sore throat, or albuminuria, and is not followed by desquamation. The fact that it has followed directly after the administration of an enema is the main point in the diagnosis.

Erythema due to the various trade causes mentioned under Group 3 in the above list may not be relegated to its correct cause unless the nature of the patient's occupation is fully understood: but a general indication which is common to all this group of erythemata is that the patient does not suffer when he is away from his work, but gets recurrences when he returns to his old surroundings. The same applies to the effects of certain garden and hot-house plants, though here the source of the irritant may escape diagnosis unless the possibility is borne in mind; particularly in the case of persons living in houses upon which *Rhus toxicodendron* is growing in place of ampelopsis as a virginia creeper. *Knot-grass* is a common weed in some districts, but it is not likely to produce erythema unless the patient has recently been indulging in extensive weeding operations in the garden.

The *stings of jelly-fish* are familiar to bathers as well as to fishermen, and in addition to intense itching and irritation, acute œdema may result and generalized erythema and urticaria. The skin eruption may not be confined to the part actually stung by the jelly-fish, for sometimes after a latent period of from twelve to twenty-four hours there may be a generalized erythematous eruption although the jelly-fish sting may have been purely local. It is not so much the small, flat, gelatinous jelly-fish that are the worst offenders in this respect, as the much larger ones with long red streamers.

The effects of being pricked by the spines on the gills or fins of certain fish, especially *sea-cats* and *weever fish*, are familiar to most fishermen; in addition to acute irritation of the skin, with or without urticaria, there may be intense swelling, vomiting, headache, and a feeling of such illness that the patient may be confined to bed in a temporarily serious condition for several days. Sometimes, indeed, the local spot which has been pricked may fester and remain a sore for many months.

Most of the trade irritants are apt to go further than the production of erythema, an acute vesicular dermatitis being even commoner as the result of irritants mentioned on page 222.

Little need be said about the erythema following upon *operations*, or that which follows upon *extremes of heat* or *cold*. Local erythema, especially of the feet, was very common amongst those who had to man the trenches in the Great War; in most instances it stopped short of actual frost-bite with gangrene, but the erythema persisted for weeks or months, accompanied by swelling, and great local pain and consequent limping gait.

One need not discuss from the point of view of the erythema itself the reddening that may be associated with abscess formation, phlebitis, cellulitis, lymphangitis, etc., mentioned in Group 6 in the above list. The diagnosis is indicated by other symptoms that will be present.

Ptomaine poisoning is characterized much more by acute vomiting, accompanied or followed by severe recurrent diarrhœa, than it is by erythema; but in some cases in which erythema is a prominent feature it may make the diagnosis less easy than when it is absent. When a single patient is attacked, the nature of the condition may be difficult to determine unless enquiry into the previous dietary shows that some particular food likely to produce ptomaine poisoning has been partaken of; the erythema generally comes on either at once or else twenty-four hours or more after the food in question. It is noteworthy that precisely similar erythema may result in some cases from the ingestion of foods which do not make other persons ill at all, in which respect there are personal idiosyncrasies to crab, strawberries, and occasionally to other quite ordinary foods which the patient can never take without suffering from erythema or urticaria, or both.

In *cerebrospinal meningitis* generalized erythema is only one of many possible skin eruptions ; herpes facialis is much commoner, and a vesicular eruption on the trunk and limbs is more usual than one which is purely erythematous. Most characteristic of all, however, are purpuric spots, varying in size from mere petechiæ to relatively large purple blotches, though this purpura develops in less than half the cases ; at the same time there will in most cases be somnolence, headache, green vomit, and pyrexia to indicate the nature of the malady, the diagnosis of which is confirmed by finding meningococci after lumbar puncture.

In *malaria* acute erythema is not common ; the patient will generally give a characteristic history of recurrent rigors with intervals of perfect health, and he will have lived or be living in a malarial district ; hæmatozoa (*Plate* VI, p. 32) will be looked for in the blood.

Leprosy is discussed on page 403, and *dengue* on page 466 ; patchy erythema is almost constant in the latter, but is not so frequent in the former.

The main characters of each of the various diseases that may be associated with localized erythema mentioned in Group 8 (*a*) in the list above, are described in other parts of this volume : *erysipelas* on page 674 ; *small-pox* on page 560 ; *erythromelalgia* on page 256 ; *Raynaud's disease* on page 256 ; *erythema induratum*, or Bazin's disease, on page 404 ; *polymyositis* and *trichinosis* on page 464 ; and *rat-bite fever* on page 598.

Pellagra, rare though it is in this country, has now been recognized in so many individual patients and in such various parts of Great Britain, that it merits special mention, for although it is much commoner in other countries, especially Italy, it seems likely that it would be diagnosed correctly, and more often, if its chief characters were more familiar. The malady is one of months or years as a rule, and a certain proportion of the cases recover. The disease starts as a rule with acute gastro-intestinal disturbances in the form of nausea, vomiting, and diarrhœa, which last is generally severe and sometimes intractable. Acute ptomaine poisoning may be simulated at this stage; but the diarrhœa, which is seldom absent, persists in a way that will exclude ptomaine poisoning, whilst at the same time there is generally a remarkable soreness of the mouth and tongue and considerable salivation, such as does not accompany ptomaine poisoning. Such an attack may subside, to recur after an interval of days or weeks, and sooner or later nervous symptoms of a serious nature are added. These nerve symptoms may take the form simply of progressive weakness, but more often they may simulate some gross intracranial change on account of the severity of headache with vomiting and giddiness, semi-coma, and even, in exceptional cases, optic neuritis or optic atrophy. Hallucinations are common, and not a few of the patients become actually insane, though this does not happen as a rule until the disease has been present for some time. During exacerbations there may be pyrexia, sometimes of long duration (*Fig.* 101), though generally not of severe degree. The two chief groups of symptoms which call attention to the seriousness of the patient's illness in its earlier stages are the gastro-intestinal on the one hand, and the cerebral upon the other. If either of these were present alone the correct diagnosis would probably never strike one, but the characteristic feature which may very likely call one's attention to the nature of the case, rare though the condition is, are the changes in the skin. A few days, weeks, or months after the first onset there develops on the backs of the hands a remarkable discoloration

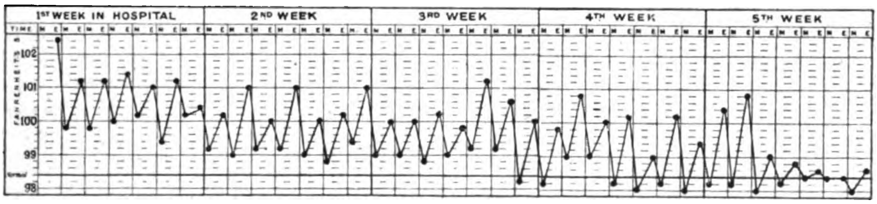


Fig. 101.—Temperature chart of the last five weeks of an exacerbation of pellagra in a case which occurred in the south of England. The patient had been ill, with fever, for seven weeks before he was admitted to hospital.

which, at first red and erythematous, presently becomes more pigmented, so as to resemble a condition of extreme sunburn. The skin soon becomes not only dark but thickened and rough, and presently also cracks or fissures may occur and desquamation follows. Such an attack of erythema of the dorsal surface of the hands may subside, to recur again after an interval, and it may then spread to the face or other parts, remaining nearly always symmetrical (*Plate* IX). There is a well-defined line of demarcation between the erythematous or pigmented parts and the normal skin immediately above it. It is by these skin changes that the disease is recognized, and it is generally upon looking back over the history that one realizes that the previous cerebral or gastro-intestinal attacks were part of the same malady. Cases have been recorded in patients who have never been out of London, whilst in some village districts in Hampshire and in some parts of Scotland several consecutive cases have been recognized in the same valley, and there is a belief, not yet proved, that the disease is microbic, and spread by infection from water derived from particular soil; though there is an alternative theory that it is a malady of diet, pellagra having been attributed to maize in much the same way that beri-beri is to decorticated rice.

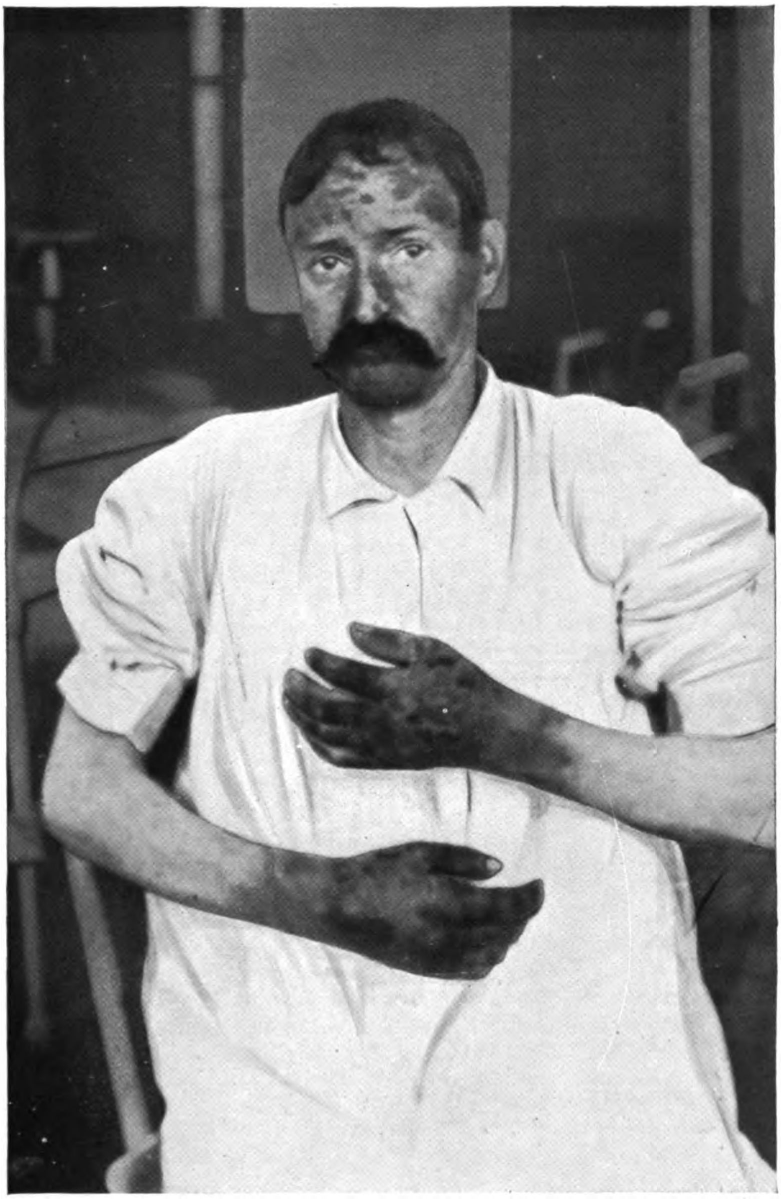
Both *Meige's disease* and *angioneurotic œdema* are characterized by œdema rather than by erythema in the great majority of cases (p. 411); in each of these two conditions, however, which are doubtless in their primary pathology related to one another, both being functional disorders of the vasomotor system, the patient is liable to acute attacks associated with vomiting, possibly diarrhœa, malaise, generalized backache and limb pains, pyrexia amounting to 101° or 102° F., and acute erythematous eruptions which may be localized or general. The erythema is very similar to that of erysipelas, especially when the attack is localized, and it is more than probable that when the first attack occurs erysipelas will be diagnosed. The attack may last a day or two, or a week, and then subside either completely, or, in Meige's disease particularly, there may be a temporary or even permanent increase in the localized œdema. The exact nature of such an attack may escape recognition until familiarity with them is established by their recurrence in the same patient at intervals of months or years, each subsequent attack being very similar to the one before; another point which may assist the diagnosis even in a first attack is the occurrence of similar pyrexial and erythematous bouts in other members of the same family, for both Meige's disease and angioneurotic œdema are familial disorders.

The erythemata due to parasitic toxæmia, especially to *hydatid disease*, *tapeworm* infection, *filariasis* and *trypanosomiasis* may sometimes be extreme, and in a case of severe erythema with constitutional symptoms in which no apparent cause can be found, examination of the fæces for tapeworm ova should not be omitted. In some cases of hydatid disease, generalized erythema has been the first symptom to draw attention to the fact that anything was wrong, but to attribute such erythema to hydatid infection would be exceedingly difficult unless upon physical examination some evidence of a cyst in the liver, in the peritoneum, or elsewhere could be found. If the possibility were suspected, but no confirmatory signs discovered, an additional test is the specific hydatid serum reaction, for which blood can be taken from the patient, as in testing the Wassermann reaction, and sent to a special laboratory for examination. If there were acute erythema from hydatid disease, the hydatid would almost certainly be active and the patient's blood serum would give a positive hydatid precipitin reaction; whilst EOSINOPHILIA (p. 218) would probably be pronounced also. The erythema of filariasis and of trypanosomiasis may occur early in the infection; the nature of the malady would be proved by the discovery of filaria embryos (*Plate* XXVIII, *Fig. F*, p. 614), or of trypanosomes (*Plate* XXVIII, *Fig. G*, p. 614) in the patient's blood, though the nature of the infection would first be suggested by the history of residence in countries in which one or other of these parasites is prevalent.

There remain for discussion erythema simplex, erythema multiforme, erythema exfoliativum seu scarlatiniforme; scarlet fever; German measles; 'fourth' disease; and the erythema of acute rheumatism. When erythema due to any of these causes is met with, the main object in the diagnosis will be to either recognize or exclude *scarlet fever*. It will be found in practice that it is sometimes quite impossible to be certain whether a given generalized erythema is that of scarlet fever or not. So much a matter of opinion may it be that even at a consultation between the highest physicians some will consider

PLATE IX

PELLAGRA



This patient is a man who had a severe attack of pellagra during the summer of 1910, and went into complete remission in about two months. He has not come under observation since. Note lesions on hands, lower part of forearms, forehead, side of the nose, and chin.—*The Photograph is by Dr. Beverley R. Tucker, U.S.A.*

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the lesion to be that of scarlet fever, others will say that in their opinion it is certainly not scarlet fever. The importance of the decision lies in deciding whether the patient should be sent to a fever hospital or not. If the condition is not scarlet fever, then by so sending the patient to a fever hospital one renders him liable to get scarlatina if his erythema is of some other kind. If, on the other hand, his condition is really one of scarlet fever, and it is regarded as non-scarlatinal from the atypical character of the rash, other individuals in the household or community may catch the infection if the patient is treated as suffering from some other form of erythema. The right thing to do under such circumstances is to isolate the patient as though it might be scarlet fever, until the progress of the case and other circumstances prove that it is not; carrying out the isolation in a separate room, in which the patient himself runs no risk of infection from other scarlatinal cases. In not a few such instances there will be doubt for all time as to whether the patient has had scarlet fever or not; because, especially now-a-days, the scarlatinal rash is sometimes almost transient and is often atypical. The important points to pay attention to are: the onset of the illness on the day before the rash appears with vomiting as an early symptom; the extreme redness of the throat, fauces, and pharynx; the coated tongue, perhaps with red fungiform papillæ projecting through the pallor of the fur; pyrexia of 102° or 103° F., which if the case be followed should fall by lysis and reach normal about the end of the first week if there are no complications such as otorrhœa or adenitis; tender swellings in the neck; absence of all rash upon the face, forehead, scalp, or behind the ears, associated with a bright scarlet erythematous eruption all over the trunk and limbs, beginning at the root of the neck and extending thence downwards. This erythematous eruption will be found on careful inspection to be not purely erythematous, but an erythema associated with very fine red dots, which show up best if some affected part of the skin is pressed gently with a glass spatula or microscope slide, so that after the redness of the erythema has disappeared by the compression the minute red dots can still be seen. It is a 'punctate' erythema. If with these characteristics the patient also has a mild degree of albuminuria, the diagnosis of scarlatina is exceedingly likely; and if after ten days or a fortnight the characteristic peeling develops, starting round small pin-prick-like foci and extending thence concentrically away from the central minute hole, the diagnosis is almost certain. Upon the hands and feet the desquamation does not have this pin-hole type, the surface epidermis coming off rather in flakes or casts than in fine scales.

One of the most difficult skin affections to distinguish from true scarlatina is *erythema scarlatiniforme*, which has also been called *erythema exfoliativum*; with this, either at the same time that the rash appears, or a few hours before it, the patient becomes suddenly ill, with shivering and loss of appetite, and there may be reddening of the tonsils and fauces, and a condition of tongue very like the strawberry tongue of scarlet fever, with pyrexia. Erythema scarlatiniforme is apt to recur in the same patient, and the difficulty of diagnosis will be much less after a second or later attack than it is in a first, but most cases are diagnosed as scarlet fever in their first attack. Owing to the close resemblance between the two diseases, it is certainly right that such patients should be isolated in any case. Two points of distinction between the two that are worthy of special note are: first, that with scarlet fever the desquamation seldom begins before the end of the first week and is usually not at all marked until even later than this; in erythema scarlatiniforme desquamation, which may be extreme, generally starts whilst the erythema is still present, often on the second or third day, and nearly always not later than the third or fourth; and secondly, that the erythema of scarlet fever may last twelve hours or less, and seldom has a duration of as much as a week, whilst that of erythema scarlatiniforme may persist for two or three weeks, or in some cases even for a month or more.

Erythema multiforme, which is one of the erythemata which may be classed as apparently idiopathic, seeing that no cause is known, is generally distinguished from other forms by the fact that it is very seldom purely erythematous. There are nearly always vesicles or even bullæ at the same time (p. 756), whilst scarlet fever and the other erythemata we are discussing practically never become vesicular.

Erythema simplex, which may also resemble mild scarlet fever, or be mistaken for it, is perhaps even more liable to be mistaken for mild erysipelas; it is seldom universal; much more often it occurs in local patches. If there are several patches on different parts of the body at the same time, erysipelas would be unlikely, nor is the edge of each red patch as much raised or as sharply defined as is that of erysipelas; in addition to which there are few if any constitutional symptoms, and little pyrexia or none. Sometimes the patches come and go over a period of days or weeks, either in the same or in different parts of the body, and then the term *erythema fugax* is applied. The diagnosis of erythema simplex can only be made when the circumstances of the case lead one to exclude all other possibilities, and the main difficulty will be to be certain that the patient is not having a very mild and atypical attack of scarlet fever. In all such cases very careful watch should be kept upon the condition of the urine, lest there should be nephritis really of scarlatinal origin, which if not looked for in this way may escape detection altogether; the patient coming under observation ten or fifteen years later with a Rose-Bradford kidney (p. 11) arising out of a condition which was so mild as to be diagnosed erythema simplex, when it was really scarlatina.

Acute rheumatism is liable to be associated with various skin eruptions, including not only subcutaneous nodules (p. 405) and erythema nodosum (p. 404), but also generalized erythema. Doubt has been expressed as to whether some of the cases in which joint pains, transient bruits and erythema have been attributed to acute rheumatism are really rheumatic at all; but in the absence of proof to the contrary, one must in the meanwhile allow that the older teaching may be correct, and that acute generalized erythema closely simulating scarlet fever may be associated with rheumatic fever. If there are no joint pains in such a case the diagnosis will be very difficult, but if there are joint pains and pyrexia, and if both the pyrexia and the joint pains disappear within thirty-six or forty-eight hours after the administration of salicylate of sodium in appropriate doses, the probability will be that the patient is suffering from acute rheumatic erythema. The erythema itself is of little moment in such cases; the main point in the case is to eliminate the possibility of scarlet fever with joint pains, and if there is doubt it will be better to treat the patient as a possible case of scarlet fever than to assume too readily that the condition is rheumatic. Sore-throat may be a marked feature in either case.

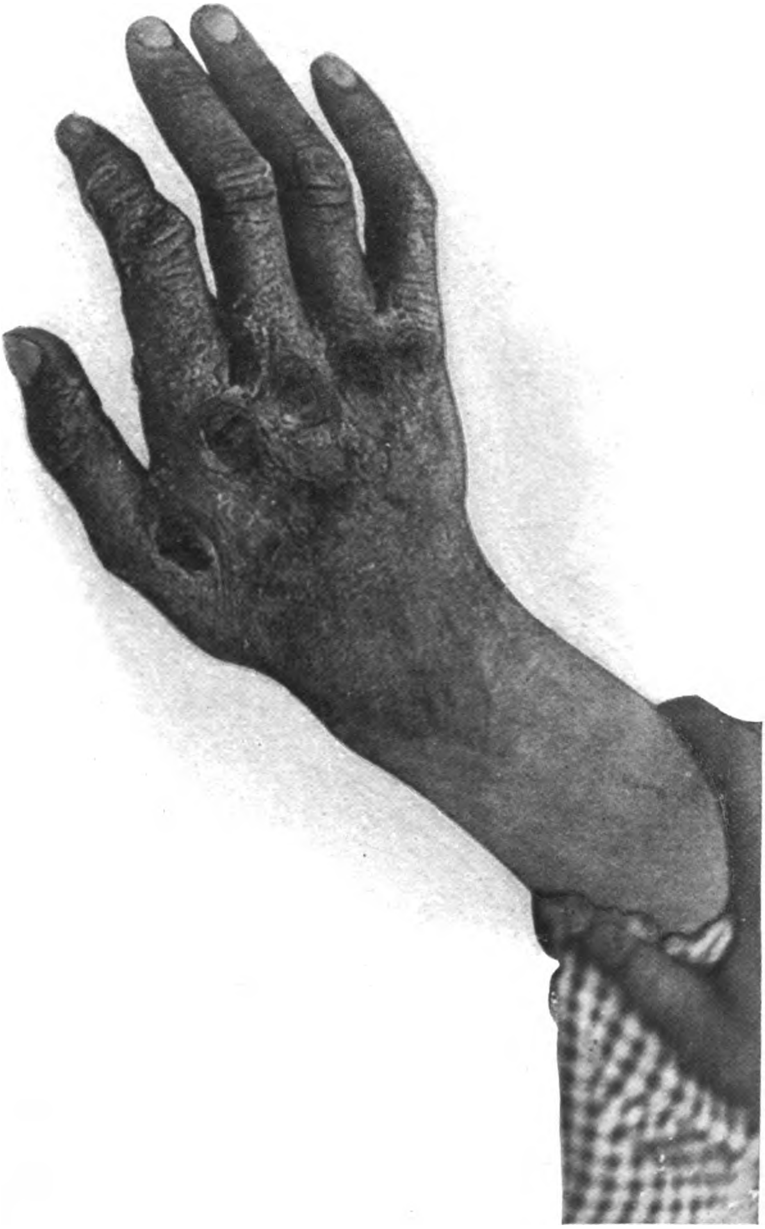
This leaves for discussion *measles*, *German measles*, and '*fourth*' *disease*. Both measles and German measles are nearly always maculo-papular rather than purely erythematous as regards the skin eruption, and the rash in both these diseases affects the face and the neck as much as, and even sooner than, the body or limbs; whereas in scarlet fever the actual exanthem does not attack the face at all, though the latter often has a characteristic appearance of pinkness of the cheeks with pallor round the nose and mouth, the general tone and appearance having been described as the 'peach-blossom' facies. In measles, moreover, there will almost certainly have been some constitutional symptoms for about three days before the rash appears, especially running at the eyes and nose simulating a common cold; in addition to which a very helpful point in differential diagnosis is the development of Koplik's spots (*Plate* VIII, p. 178) within the mouth. These may occur singly, but more often they are in groups varying in number from two or three to a score or more, each spot having a pale, almost white, centre, the size of a small pin's head, surrounded by a deep-red injected periphery; they are to be expected upon the inner surface of the cheeks, upon the mucous surface of the lips, and sometimes upon the gums, generally at some little distance from the teeth. They may also develop upon the mucous membrane of the soft or hard palate, though here they may be simulated by particles of milk if care is not taken to see whether or not they are removable by means of a soft brush.

German measles is more apt to simulate scarlet fever than ordinary measles is, and with German measles there are no Koplik's spots to help one. The rash, however, if inspected carefully in different parts, will generally be found to be a characteristic macular one somewhere, and it will be found upon the face or forehead, or behind the ears, which will not be the case with scarlet fever. The constitutional symptoms are generally slight, even though the rash is very extensive, and a very helpful point in diagnosis is the presence in German measles of generalized enlargement of the lymphatic glands, including not only those in the neck, axillæ, and groins, but also those in the occipital region. In scarlet fever, although the glands in the neck may be swollen and very tender, those in the occipital region and elsewhere are not generally enlarged.

The chief remaining difficulty is in connection with what has been called '*fourth*' *disease*. Though this is accepted by many observers as being a distinct entity, its existence is not allowed by all. It has struck many observers, however, that patients who

PLATE X

PELLAGRA



The hand of a pellagrin. Note the areas of sloughing, and the deep fissure between the index and middle fingers.—*The Photograph is by Dr. Beverley R. Tucker, U.S.A.*

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have been known to have had German measles and ordinary measles and scarlet fever definitely in the past, may yet develop an acute erythematous exanthem which has some of the characteristics of measles, some of the characters of German measles, and some of the characters of scarlet fever, without nevertheless being typical of any one of these three. The malady may spread through a school or a household or institution, and produce similar characteristics in other individuals who, not having had scarlet fever or measles before, if seen by themselves would be diagnosed as suffering from one or other of the three better known maladies. As, however, in such an epidemic it may attack those who have had scarlet fever before and also those who have had measles before, and those who have had German measles previously, in addition to those who have had all three before, those who have had most to do with cases of this kind incline to the belief that there is a definite 'fourth disease' distinct from the other three. It is a relatively mild malady, with some pyrexia and some constitutional disturbance, but not much of either, and in all cases there is a widespread erythematous or maculo-erythematous eruption. There are no points which are distinctive of the malady, however, and if it exists at all it can only be diagnosed when circumstances suggest that patients are suffering from it who ought not to be liable to any one of the three better known exanthems of similar type. The weak point in the argument is that it is a well-known fact that although German measles, measles, and scarlet fever protect against subsequent attacks of the same maladies in most individuals, there are some who may have not only two but three or even several separate attacks of either German measles or measles or scarlet fever ; and it is on this account that one cannot say definitely that there is such a disease at all as the 'fourth' disease. Hence its diagnosis in any particular instance must necessarily be one of opinion only. *Herbert French.*

ERYTHRÆMIA.—(See POLYCYTHÆMIA, p. 532.)

ERYTHRASMA.—(See FUNGOUS AFFECTIONS OF THE SKIN, p. 251.)

ERYTHROPSIA.—(See VISION, DEFECTS OF, p. 762.)

EXOPHTHALMOS (or Proptosis)—May be bilateral or unilateral.

Bilateral Exophthalmos.—The commonest cause of this condition is *Graves's disease*, in which the exophthalmos is associated with other general symptoms, such as tachycardia, swelling of the thyroid gland, fine tremors, and general nervousness. The eyes are pushed forward to a varying extent (*Fig.* 114, p. 236), in some cases the protrusion being so great that they cannot any longer be covered entirely by the lids. The protrusion causes the upper lid to be unusually raised, and the eyes look wide open, giving the patient an expression of alarm or astonishment (Stellwag's sign, due to spasm of the levator palpebræ superioris). When the eyes are lowered, the upper lids do not descend to the same extent as the cornea, but leave a broad portion of the sclerotic visible above the cornea (von Graefe's sign). Winking takes place less frequently, and convergence of the eyes is sometimes rendered difficult (the sign of Moebius)

Bilateral exophthalmos may also be caused by *thrombosis of the cavernous sinuses.* This condition is usually secondary to some furuncle or carbuncle of the skin of the face in the region of the eye, to orbital cellulitis, or suppuration in the accessory sinuses of the nose. It usually starts on one side, and invariably spreads to both in the later stages of the attack. The eyes are protruded and fixed, the eyelids are red and engorged, and the frontal and ophthalmic veins are dilated and full. Movements of the eyes are very limited, and there is much swelling and induration of the orbital tissues. In association with the orbital infiltration there is often some swelling in the region of the mastoid process, owing to the exit in this region of an emissary vein in connection with the sinuses that communicate with the two cavernous sinuses. This condition is nearly always fatal, as it is followed by a suppurative meningitis.

Unilateral Exophthalmos may be due to :—

Orbital cellulitis
Thrombosis of the cavernous sinus
Orbital periostitis
Meningocele and encephalocele
Gumma
New Growth
Exostosis
Tubercle
Arteriovenous aneurysm
Distention of the accessory sinuses of the nose.

The diagnosis of *orbital cellulitis* and *thrombosis of the cavernous sinus* presents little difficulty, owing to the symptoms of acute inflammation that are present, orbital cellulitis being distinguished from cavernous sinus thrombosis by the fact that it is usually unilateral and there is no œdema in the mastoid region.

Orbital periostitis, especially in more chronic cases, may give rise to varying degrees of proptosis, and in the absence of any obvious thickening of the orbital margins the diagnosis may be obscure. In any periosteal inflammation of long standing, a skiagram will usually show a definite increase of density in the affected bone.

Meningoceles and *encephaloceles* may in some cases be difficult to diagnose from dermoid cysts. The latter are usually placed anteriorly in the orbit, and do not therefore cause any proptosis, though they may displace the eyeball. A meningocele usually presents itself through a gap between the ethmoid and the frontal bones (*Fig.* 102), and is attached to the bone. An opening may sometimes be found through which the meningocele communicates with the cranial cavity. Meningoceles sometimes pulsate in association with the arterial and respiratory oscillations. They may also be diminished in size by pressure of the fingers, as the fluid can be squeezed into the cranial cavity. In many cases an exploratory puncture is the only means of making a certain diagnosis.

A *gumma* of the orbit can only be diagnosed from the patient's general history, evidence of specific disease elsewhere, a rapid improvement in the condition after the administration of salvarsan, mercury, or iodide of potassium, and perhaps a positive Wassermann's serum reaction.

A *growth of the orbit* has usually no distinctive feature, and can only be diagnosed by means of an exploratory operation and the removal of a portion for microscopical examination ; but it is to be remembered that tumours of the optic nerve can usually be diagnosed with accuracy by the fact that they always produce some compression of the eyeball in the antero-posterior diameter. Cases of proptosis, therefore, in which there is increasing hypermetropia on the affected side, may be ascribed to a primary tumour of the optic nerve.

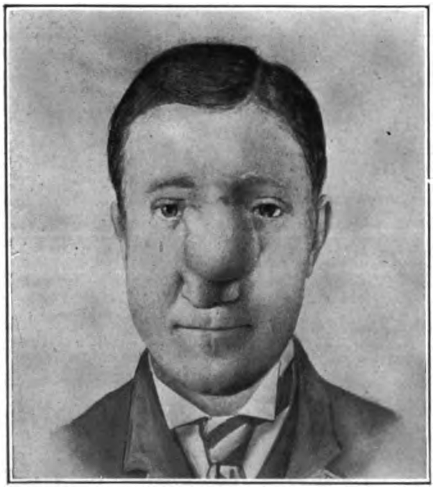


Fig. 102.—Meningocele projecting into the face from the anterior part of the base of the skull. (*From a drawing in the Gordon Museum, Guy's Hospital.*)

Ivory exostoses or *osteomata* usually arise from the frontal bone and are attached by a broad base, so that their removal presents very great difficulty ; the diagnosis depends on their slow growth and excessive hardness ; a skiagram shows their presence with great certainty.

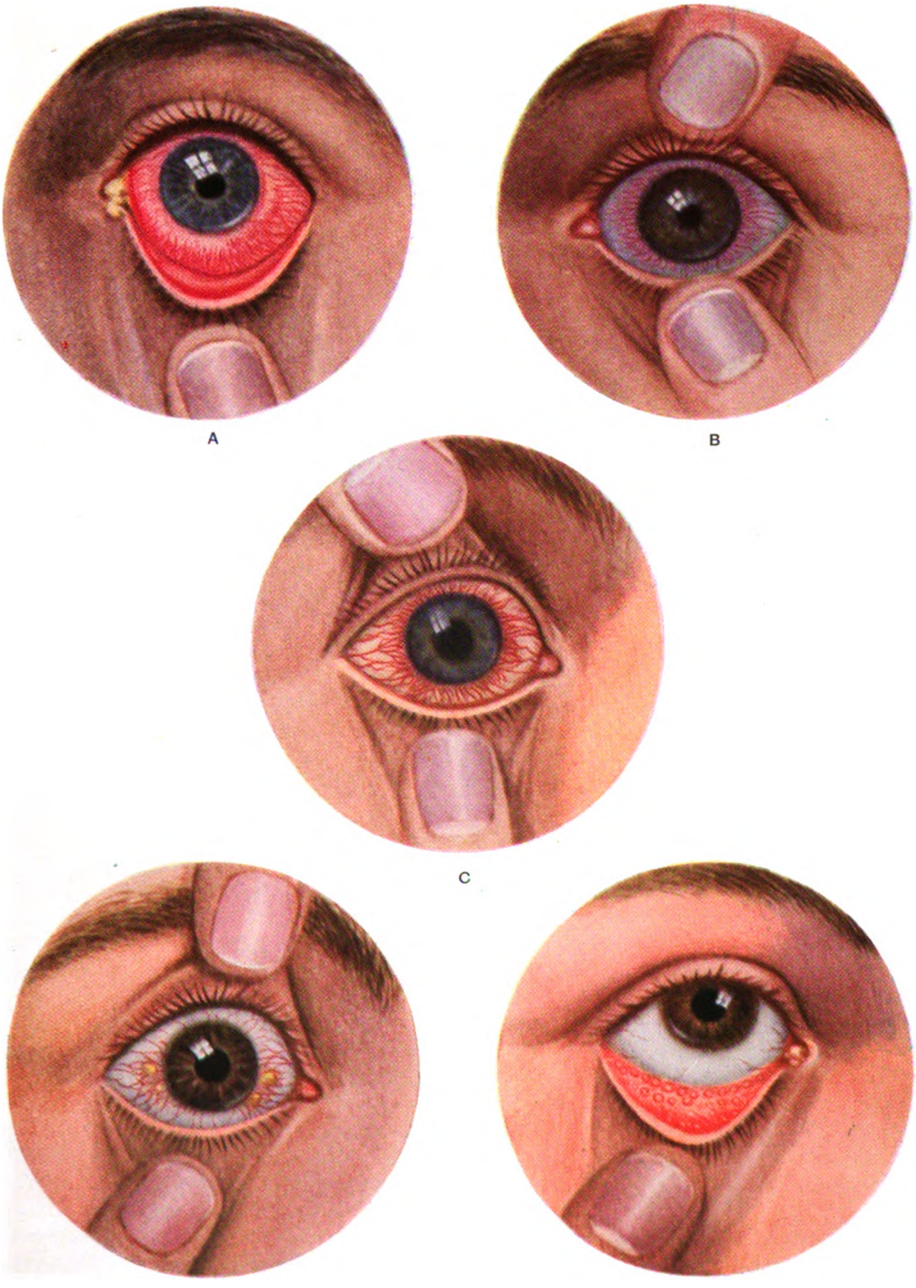
Some cases of *tuberculous disease of the orbit* may closely simulate orbital cellulitis or distention of the accessory sinuses of the nose, and the diagnosis can only be made with certainty after excision of a portion of the infiltrated tissue and a microscopical examination of the fragment.

An *arterial aneurysm* is nearly always associated with a pulsating exophthalmos, in which there is protrusion of the eyeball and dilatation of the blood-vessels of the retina, lids, and conjunctiva. There is distinct pulsation of the eyeball, and a loud blowing murmur on examination with the stethoscope. Compression of the carotid on the same side diminishes the pulsation and the sound. The usual cause of arterial aneurysm is the rupture of the carotid into the cavernous sinus as the result of an injury. Rare cases are also seen of intermittent exophthalmos, which appears only at intervals or when the head is depressed. These are usually due to varicose veins in the orbit not in communication with an artery.

The protrusion of the eyeball in *dilatation of the accessory sinuses* of the nose is, as a rule, less an exophthalmos than a displacement of the eyeball downwards and outwards. In dilatation of the frontal sinus there may be some thickening and fullness of the supra-orbital ridge associated with pain and tenderness over the eyebrow. In dilatation of the ethmoidal cells there is usually a definite swelling to be felt at the inner side of the orbit,

PLATE XI

ACUTE INFLAMMATIONS OF THE EYE



A, Acute conjunctivitis. **B**, Acute iritis. **C**, Glaucoma. **D**, Phlyctenular conjunctivitis. **E**, Follicular conjunctivitis.

INDEX OF DIAGNOSIS—*To face p.* 230

which is compressible though not distinctly fluid. Dilatation of the sphenoidal sinus is sometimes accompanied by neuritis or atrophy of the optic nerve. In all cases of proptosis due to sinus trouble of any duration, there is evidence in the nose of inflammation of these cavities, the usual symptom being the existence of polypi or of definite swellings in the region of the infundibulum. *Herbert L. Eason.*

EXPECTORATION.—(See Sputa, p. 641 ; and Hæmoptysis, p. 285.)

EXTENSOR PLANTAR REFLEX.—(See Babinski's Sign, p. 68.)

EYE, ACUTE INFLAMMATION OF.—Acute inflammation of the eye may be due to three main types of disease, *conjunctivitis*, *iritis*, and *glaucoma*. The character of the inflammation varies with the type of the disease, but certain symptoms, such as *pain*, *photophobia* (intolerance of light), and *lachrymation*, are common to all inflammatory conditions, and are therefore of little diagnostic value.

In *conjunctivitis* the conjunctival vessels are dilated, bright red, and injected ; they are freely movable over the subjacent sclerotic, and the injection is most evident in the equatorial region of the ball of the eye, the circumcorneal portion of the conjunctiva, owing to its firmer attachment to the sclerotic in this region, being relatively paler. The cornea is usually clear and polished, unless there are corneal ulcers (see below); the anterior chamber and iris are normal in appearance, the pupil is black, and the iris active. There is always more or less secretion of purulent material, which collects at the inner angle of the palpebral aperture and on the edge of the lids, especially after sleep. The eye feels hot and dry, and, owing to the œdema of the inner surface of the lids and the enlargement of the lymphoid follicles, there is a feeling of grittiness as of sand or dust in the eye.

In the ordinary *infectious* or *catarrhal* ophthalmia (' pink eye ') the inner conjunctival surface of the lids is velvety and swollen, but there is little or no œdema of the conjunctiva covering the eyeball. In *gonorrhœal conjunctivitis* by contrast, a brawny œdema of the lids and intense swelling and œdema of the conjunctiva, which is raised all round the corneo-sclerotic margin (*chemosis*), is a prominent symptom. In the earlier stages of the infection, the discharge is yellow, serous, and blood-stained, but rapidly becomes purulent and extremely profuse. The cornea ulcerates as a rule, its substance apparently melting away in a characteristic manner. Gonorrhœal ophthalmia of the new born (ophthalmia neonatorum) exhibits similar symptoms, early and destructive ulceration of the cornea being one of its most serious complications. In *follicular conjunctivitis* the conjunctiva, especially of the lower lids, is studded with small raised lymphoid follicles, which are transparent and gelatinous in appearance. In *trachoma* the conjunctiva is also studded with enlarged follicles, but in this disease they are found particularly on the under surface of the upper lid and in the upper conjunctival fornix. The follicular enlargement is associated with considerable thickening and œdema of the tissues of the upper lid causing a partial ptosis, with profuse lachrymation and, in the later stages, with a vascular opacity (*pannus*) of that part of the cornea which is usually covered by the upper lid. In the later stages of trachoma the infiltration is followed by the formation of fibrous tissue, causing bending of the tarsal fibro-cartilage, entropion, and trichiasis. In *phlyctenular conjunctivitis* there are to be seen one or more round yellowish raised masses at the corneo-sclerotic margin surrounded by a localized area of vascular conjunctiva. In some cases the phlyctenules may encroach upon the corneal surface, being followed by a trail or leash of conjunctival vessels. *Chronic conjunctivitis* in adults is sometimes characterized by being confined to the inner and outer angles of the palpebral aperture (angular conjunctivitis), the infection being due in this case to the diplobacillus of Morax-Axenfeld. In this form of conjunctivitis the edges of the lids as well as the conjunctiva are moist and red, especially at the inner and outer canthus.

In *membranous conjunctivitis*, which may be due either to the diphtheria bacillus or more commonly to staphylococci, the under surface of the lids is covered with a yellowish-white membrane which can be peeled off, leaving a raw bleeding surface.

Corneal ulcers are always apparent as greyish or white opacities of the cornea over which the cornea has lost its polish. There may be only infiltration of the cornea, or in more serious cases actual loss of substance, which may ultimately lead to perforation of the cornea. In certain cases of corneal ulcer there may be pus in the anterior chamber (hypopyon). The diagnosis presents no difficulty, as their existence is always obvious if the cornea be carefully examined.

In *iritis* the inflammation of the eye presents rather different characteristics. As the iris receives its blood supply from the deeper ciliary vessels, the dilatation of these shows a marked contrast to that of the conjunctival vessels. The injection is most evident in the circumcorneal region, the equatorial region of the eyeball being paler, and the colour of the injection being not bright red, but rather of a more dusky or violet character. The cornea retains its polish, but the aqueous is usually turbid, and there may be actual punctate deposit of fibrin and leucocytes on the posterior surface of the cornea (*keratitis punctata*) or a deposit of pus at the lower part of the anterior chamber between the cornea and the iris (*hypopyon*).

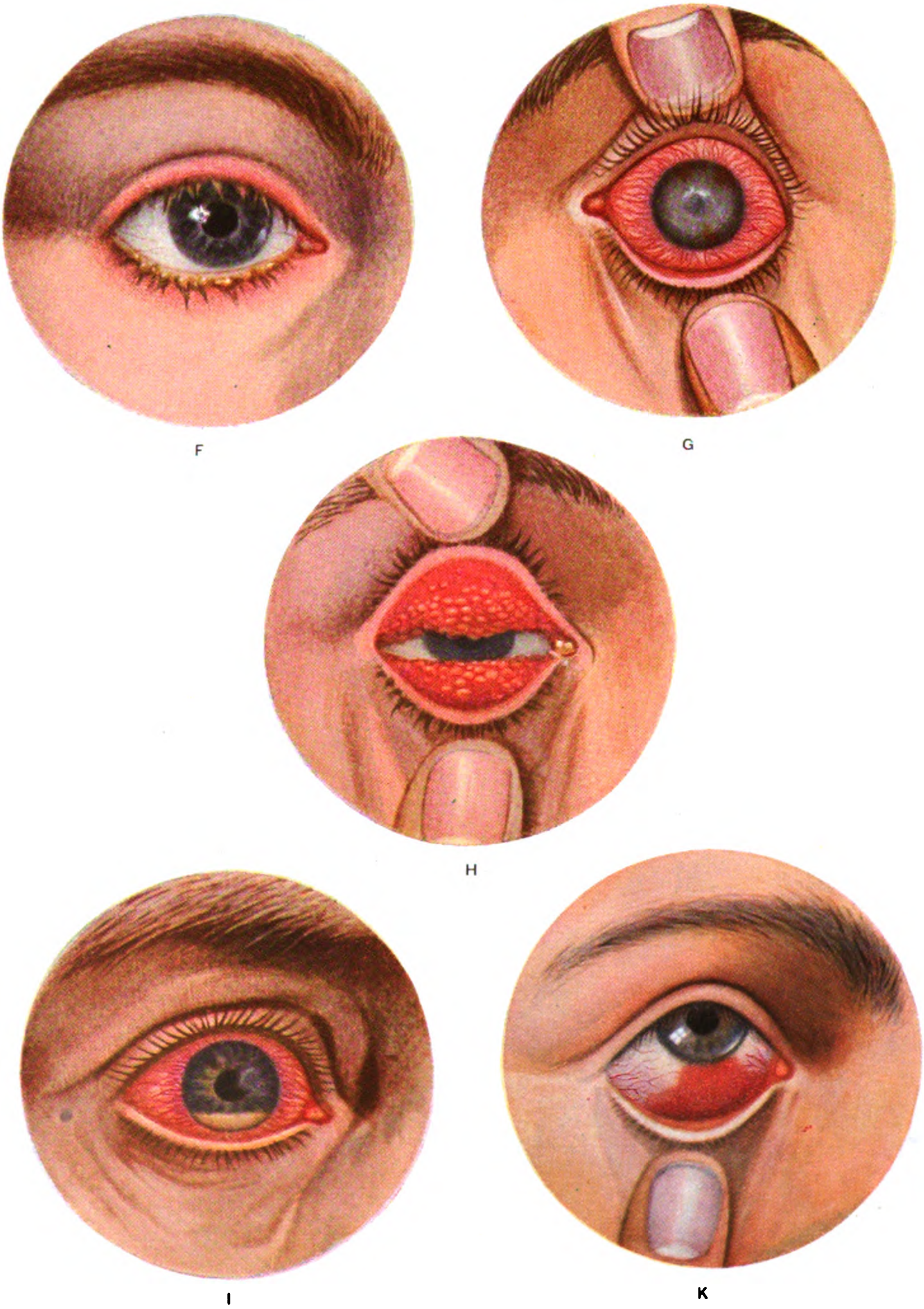
Owing to the increased vascularity of the iris, and to the exudation into its substance, its volume is increased and its mobility impaired; hence the pupil is small and sluggish or inactive. The presence of blood and exudate in the substance of the iris also changes its colour,—a blue iris becoming greenish, and the fine detail of the iris structure is blurred and obliterated. In the later stages *adhesions* will occur between the iris and the lens at the point of their immediate contact, the edge of the pupil; in the constricted state of the pupil these may not be seen, but on dilatation with atropine these adhesions or *posterior synechiæ* will prevent the enlargement of the pupil at certain points, and it will therefore be irregular in shape; small masses of iris pigment may also be seen on the anterior surface of the lens where the mydriatic may have broken down some of the weaker adhesions. Lymph may be exuded into the pupillary aperture, where it will be recognized as a filmy grey membrane completely or partially blocking the pupil.

	CONJUNCTIVITIS	IRITIS	GLAUCOMA
Conjunctiva ..	Conjunctival vessels bright red and injected; movable over subjacent sclerotic: injection most marked away from corneo-sclerotic margin; colour fades on pressure	Ciliary vessels injected, deep or bluish-red; most marked at corneo-sclerotic margin; colour does not fade on pressure	Both conjunctival and ciliary vessels injected
Cornea	Clear, sensitive	Clear, sensitive	Steamy, hazy, insensitive
Anterior chamber	Clear, normal depth	Aqueous turbid, anterior chamber slightly shallow	Very shallow
Iris	Normal colour	Injected, swollen, adherent to lens, and muddy coloured	Injected
Pupil	Black, active	May be filled with lymph, small, fixed	Dilated, fixed, rather green
Tension	Normal	Normal	Raised

Inflammatory glaucoma is an acute disease of the later years of life, attacking women more frequently than men, hypermetropes rather than myopes, and especially those who use their eyes for close work to a considerable extent. It comes on in bouts, often precipitated by ocular strain or indiscretions of diet or regimen. It may affect one eye only at first, but later both eyes are usually attacked.

PLATE XII

ACUTE INFLAMMATIONS OF THE EYE



F, Chronic blepharitis. **G,** Interstitial keratitis. **H,** Trachoma. **I,** Hypopyon ulcer of cornea.
K, Subconjunctival hæmorrhage from injury.

At first the chief complaint is of attacks of temporary obscuration of vision, the appearance of halos or rainbows round lights, and unusually rapid increase of presbyopia, or failure of accommodation for near vision. During a mild attack there is often a feeling of tension in the eyes and a dull frontal headache in addition to the loss of vision. In severe attacks the pain is very violent, radiating from the eye to the head, the ears, and the teeth, and is associated with sickness, the latter symptom often causing the condition to be mistaken for migraine or sick headache. The lids may be œdematous and the conjunctiva injected. The cornea is hazy and anæsthetic, the anterior chamber is shallow, the iris discoloured, and the pupil dilated and fixed. The eye is hard to the touch and very tender. Vision fails rapidly, diminishing in a few hours from normal to the bare perception of light. In the acute stages the optic disc is not visible owing to the opacity of the cornea, aqueous, and vitreous; but ultimately, when the media clear, the optic disc will be seen to be white and excavated (*Plate* XX, *Fig. v*, p. 418).

Subacute or simple glaucoma, but for its slower course and the absence of severe attacks, resembles acute glaucoma.

The importance of discriminating between iritis and glaucoma cannot be over-emphasized ; the use of atropine or some similar mydriatic is the *sine qua non* of the treatment of iritis, whilst in glaucoma it is disastrous.

The points which serve to differentiate these three conditions from one another are summarized in tabular form on page 232.

Herbert L. Eason.

EYE, PAIN IN.—(See Pain in the Eye, p. 445.)

EYES, BLACK SPECKS BEFORE THE.—(See Black Specks before the Eyes, p. 71.)

FACE, SWELLING OF THE.—(See Swelling of the Face, p. 673.)

FACE, ULCERATION OF THE.—(See Ulceration of the Face, p. 735.)

FACIAL PARALYSIS.—(See Paralysis, Facial, p. 491.)

FACIES, ABNORMALITIES OF.—The study of the face in health and disease, while it cannot replace careful systematic examination of the body as a whole, may in many cases direct the experienced observer's attention to the most likely field in which to find data for his diagnosis. Observation and experience alone can teach the student to detect all the features of a face. Photographs and drawings can only illustrate the coarse and obvious defects which are present when the face is at rest or when some particular movement is being sustained. The more subtle abnormalities of expression, the play of the emotions, and the response of the features to intelligence, are often too fleeting and too mobile to allow of reproduction on paper, and sometimes so intangible as to defy any effort to describe them. Even if the pen of a skilled artist could succeed in portraying the passive vacant aspect of a chronic alcoholic, it must necessarily fail to depict the traitorous tremor which hovers about the corners of his mouth when he opens it to proclaim

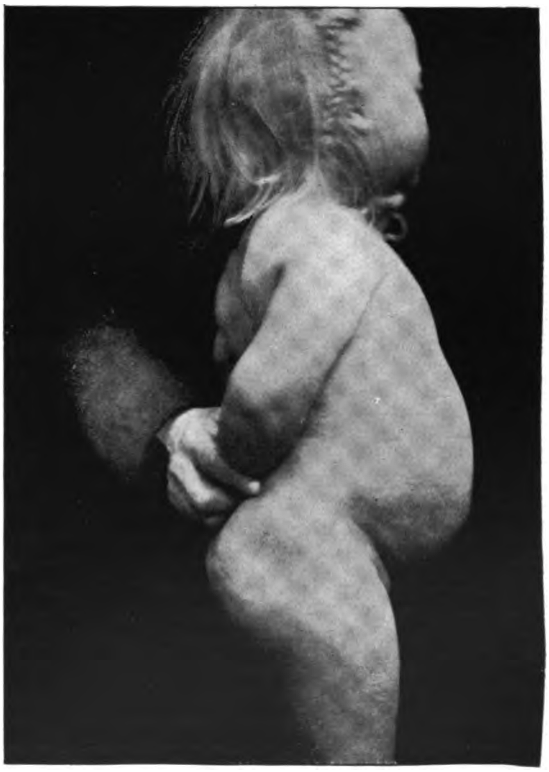


Fig. 103.—A female cretin, to show the 'frog-belly.

his temperance. The shifty eyes of the drug-taker, the fatuous placidity of the patient with advanced insular sclerosis, the anxious look born of abdominal disease, the explosive suddenness with which the victim of double hemiplegia bursts into laughter or tears, are only a few of the many familiar and striking lessons of the face which must be seen in real life if they are to be learned and utilized. On the other hand, there are facies the description and illustration of which may serve to impress their more important features on the minds of those to whom they are not familiar.

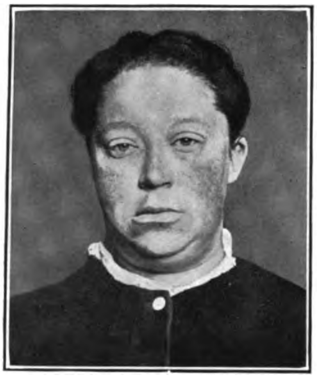


Fig. 104.—Myxœdema: the characteristic facies, illustrating the broadening of the features and the malar flush. (Compare *Fig.* 105.)

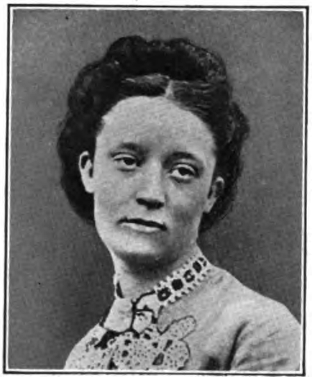


Fig. 105.—The same patient as *Fig.* 104, previous to the development of myxœdema (*From photographs kindly lent by Dr. Hale White.*)

Cretinoid Facies.—Compared with the general stunted growth of the rest of the body, the head is relatively large. The face is broad and remarkable for thick eyelids, broad flat nose, thick lips, and large coarse ears. The mouth is usually open and expressionless, the tongue may be more or less constantly protruded, and the chin is poorly developed (see DWARFISM, p. 186). The hair is scanty and brittle, the skin coarse, dry, and often almost yellow. Confirmation of the diagnosis may be sought in the dwarfed size of the child, the pendulous 'frog belly' (*Fig.* 103), and the thick pads of subcutaneous tissue especially frequent above the clavicles. The lack of mental development, the slow pulse, and subnormal temperature complete the clinical picture.

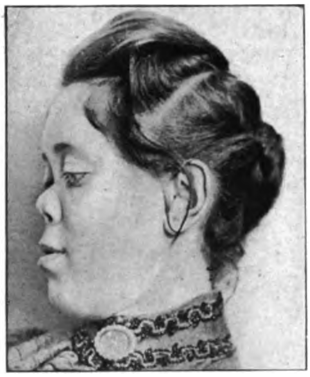


Fig. 106.—Congenital syphilis, showing prominent forehead and depressed nasal bridge.
(*Photo by Dr. Rendle Short.*)

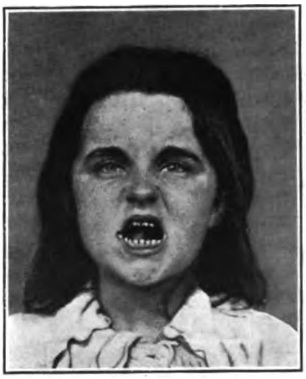


Fig. 107.—Facies of congenital syphilis, showing notched teeth and sore angles of the mouth.
(*Photo by Dr. S. A. K. Wilson.*)

Myxœdematous Facies.—The dulled intelligence of the patient is betrayed by the apathetic physiognomy (*Fig.* 104). *Fig.* 105 shows the same patient previous to the attack. The skin of the myxœdematous face is coarse, dry, and sallow, with occasional cyanotic areas over the cheeks. The puffiness of the eyelids may suggest nephritis, but the subcutaneous tissue is everywhere of firm consistence, and podgy rather than œdematous. The nose is broadened, the ears thickened, and the lips so much swollen that more than the usual amount of mucous membrane is exposed. The hair is scanty, receding from the forehead, and the eyebrows poorly marked. Similar conditions of hair and skin, together with brittle, striated nails, are found elsewhere. Masses of fatty tissue, like those described in cretins, may be found scattered about the neck and trunk. The slow speech, the expressionless face, and the general attitude of the patient may suggest paralysis agitans, but the diagnosis may be made by

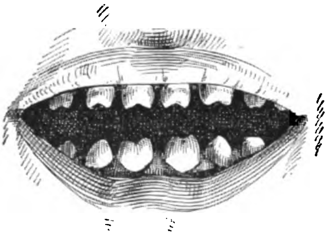


Fig. 108.—Hutchinsonian notched teeth. (From *Introduction to Surgery*, Prof. Rutherford Morison.)

paying attention to the features just mentioned, and by observing the slow pulse and subnormal temperature, and the effects of thyroid treatment.

Congenital Syphilitic Facies.—The victims of congenital syphilis, after ten or twelve years of age, may present a facies which is characteristic—an overhanging forehead, perhaps frontal bosses, a depressed nasal bridge (*Fig.* 106), striated scars radiating from the corners and other parts of the lips (*Fig.* 107), with a sallow, earthy complexion. Closer observation of the eyes and teeth may detect the opacities of old keratitis and the changes in the upper incisors which are claimed by Hutchinson to be pathognomonic (*Fig.* 108). These teeth are wide-gapped, irregular, and so deficient in enamel over the anterior and median parts of their cutting edge that the resulting crescentic notch gives them a striking appearance. Such a facies may accompany deafness, mental deficiency, physical infantilism, tibial deformities, and chronic arthritis, especially of the knee joints. The diagnosis may be clinched if the blood gives a positive Wassermann reaction.

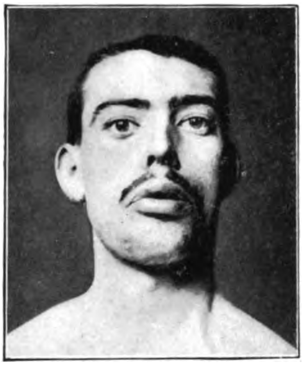


Fig. 109.—Myopathic facies: the loose pout due to weakness of the orbicularis oris.
(*Photo by Dr. S. A. K. Wilson.*)

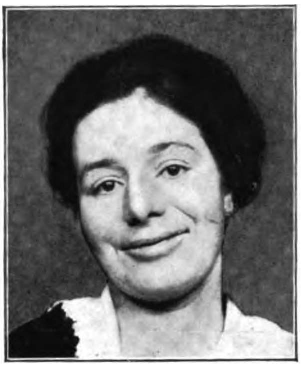


Fig. 110.—Myopathic facies: the transverse smile.
(*Photo by Dr. S. A. K. Wilson.*)

Myopathic Facies.—Many cases of myopathy show no characteristic facies; others are remarkable for the loose pout of their lips at rest (*Fig.* 109), and the 'transverse' character of their smile (*rire en travers, Fig.* 110). Both features are due to deficient facial musculature, and particularly to weakness of the orbicularis oris. The paresis of the orbiculares palpebrarum is only striking when an attempt is made to close the eye, although it may sometimes lead to prominent and perhaps staring eyeballs. In other instances there is a droop of the upper eyelids rather than any tendency to exophthalmos. The inability on the part of the patient to whistle or to blow out his cheek quickly demonstrates the weakness of the orbicularis oris, if it is not made obvious by the large amount of labial mucous membrane exposed while the mouth is at rest.

(*Reproduced from Blocks kindly lent by Messrs. Macmillan & Co.*)

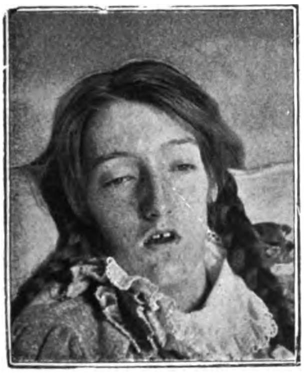


Fig. 111.—Myasthenic facies: the appearance of fatigue produced by the drooping of the eyelids and dropping of the jaw is very apparent.

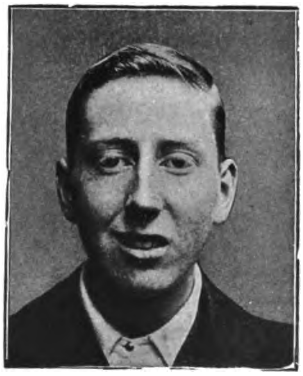


Fig. 112.—Myasthenic facies: to illustrate a 'nasal' smile on the left side of the face, and a natural smile on the right.

Myasthenic Facies.—In patients suffering from myasthenia gravis there are two types of facies which can hardly be reproduced by other diseases. The first illustrates the exhaustion of the patient (*Fig.* 111); she can hardly keep her eyes open, and her chin tends to drop exhausted on her chest. The second depends on the characteristic myasthenic smile, sometimes more appropriately named a sneer (*Fig.* 112). This unfortunate and misleading facial expression is the result of deficient action on the part of the zygomatic and risorius muscles, and exemplifies the curious way in which some muscles are affected and others escape, in this disease, even when they derive their innervation from the same source. The accompanying photograph shows how a slight asymmetry in the muscular affections may be responsible for very different expressions on the two sides of the face.

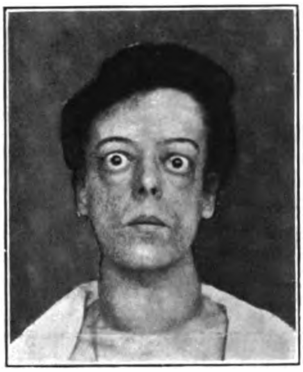


Fig. 113.—Exophthalmic goitre. (*Photo by Dr. S. A. K. Wilson.*)

The Facies of Exophthalmic Goitre.—The facial appearance in Graves's disease depends chiefly upon the 'stare' (*Fig.* 113). Surprise or fear is suggested by the prominence of the eyeballs and the retraction of the eyelids. The degree of exophthalmos varies greatly, and it is not present in all cases; sometimes it occurs on one side and not on the other. Close observation shows that the sclera is visible between the edge of the iris and the eyelids, and that the usual harmony of movement between the eyeball and the eyelid is lacking. Normal winking is frequently much diminished or entirely in abeyance. The surface of the conjunctiva may be abnormally bright and glistening, and the secretion of tears may be excessive. In contrast with the white of the eyeballs, there is often considerable dark pigmentation of the eyelids, which may also be the site of some œdema. The size of the pupils varies, undue dilatation occurring only in exceptional cases. A moist skin and a readiness to flush may often be remarked in the face.

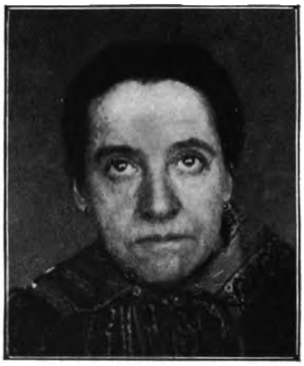


Fig 114.—Facies of paralysis agitans, showing the fixed staring expression.

The Facies of Paralysis Agitans.—In this disease a cardinal symptom is muscular rigidity, which affects the skeletal muscles generally as well as those of the face. The ocular muscles, however, escape. It is due to this fact that while the face as a whole is expressionless, 'starchy' or 'masked' (*Fig.* 114), the eyes appear to move with natural or even abnormal rapidity; for instance, they will turn in the direction to which the patient desires to look, before the head has assumed a corresponding position. Frequently the face has a staring expression, the eyelids being constantly retracted by the tonic spasm of the orbiculares palpebrarum. An absence of normal winking has been noted and ascribed to the same cause. In contrast with the slow development of facial expression under the influence of emotion, there is sometimes marked want of control over the fully-developed emotional movement, and the patient complains that the exuberance of his laughter or tears is entirely out of proportion to his feelings of merriment or sorrow.

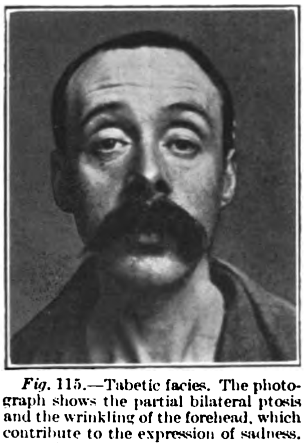


Fig. 115.—Tabetic facies. The photograph shows the partial bilateral ptosis and the wrinkling of the forehead, which contribute to the expression of sadness. (*Photo by Dr. S. A. K. Wilson.*)

Tabetic Facies.—In a considerable percentage of cases of locomotor ataxy the appearance of the face is sufficiently striking, to a close observer, to afford a clue to diagnosis. The small size or the inequality of the pupils may first attract attention. The slight drooping of the upper eyelids, combined with some wrinkling of the forehead (*Fig.* 115), due to a compensating effort on the part of the frontalis muscle, gives a sad expression. This drooping of the eyelid, which may be termed pseudo-ptosis or hypotonic ptosis, is not due to any paresis of the levator palpebræ superioris, as may be shown by the raising of the lid when the patient is looking up. It really depends on the fact that this muscle, like most of the muscles of the body, is in a condition of hypotonia. This allows the action of gravity to assert its influence, with the result that the lid hangs like a half-raised curtain in front of the eyeball. In other respects the face may be normal, but the majority of tabetics have a sallow complexion and very little subcutaneous fat, two facts which contribute to their generally unhealthy aspect. The writer believes that many victims of this disease exhibit a deficiency of the emotional reflex movements of the facial muscles. During conversation, the play of their features in response to the subject of their talk is not so noticeable as that of healthy individuals.

Facies of Acromegaly.—In the course of acromegaly, changes in appearance frequently take place to such a degree that the patient becomes unrecognizable by friends who have known him only before the onset of his disease. These changes are the result of abnormal growth on the part of the bony and subcutaneous tissues in many parts of the body, and especially in the skull and extremities. The characteristic facies is brought about by osseous hyperplasia of the frontal ridges, the mastoid, zygomatic, malar, and nasal processes, while the lower jaw is usually enlarged in all directions. The prominent, arched brows, with retreating and wrinkled forehead, the massive nose, the long, thick upper lip, and the heavy chin (*Fig.* 116) form the most conspicuous features. The lower set of teeth may project some distance in front of the upper, and they are unduly wide apart. The tongue may be so enlarged as to keep the mouth open and to display many fissures and indentations as the result of its pressure against the teeth. The increased weight of the lower part of the face tends to make the head lean forward and perhaps ultimately to rest upon the sternum. In some cases the lower jaw is not affected, and the face may be described as abnormally square (*type carrée*).

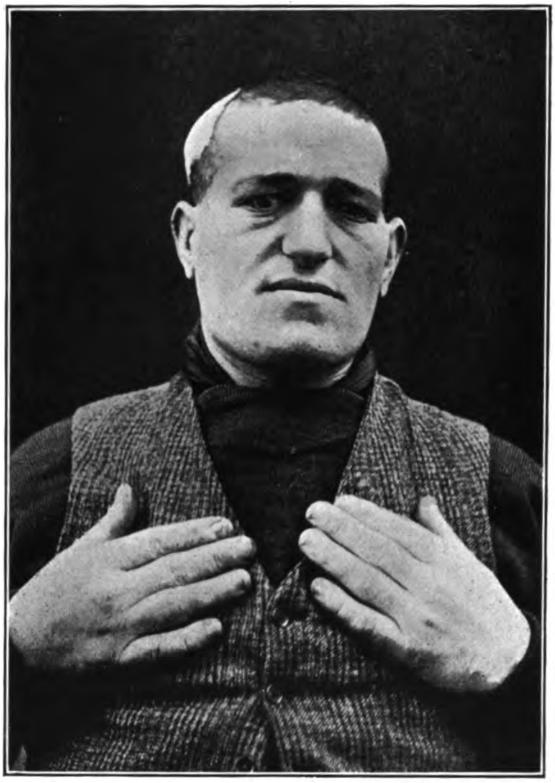


Fig. 116.—Acromegaly.

Facies of Mongolian Idiocy.—This facies is so characteristic that the diagnosis may often be made at sight (*Fig.* 117 ; see also Dwarfism, p. 186). The head is brachycephalic ; the palpebral fissures slant obliquely inwards and downwards towards a broad flat nose,

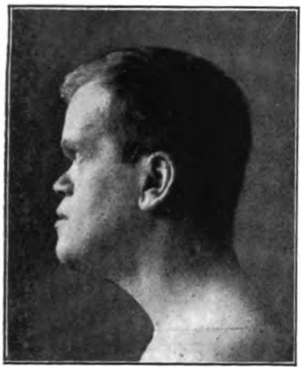


Fig. 117.—Achondroplasia.
(*Photo by Dr. S. A. K. Wilson.*)

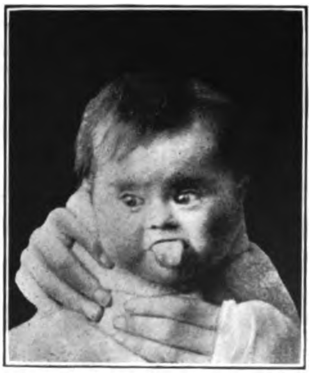


Fig. 118.—A Mongolian idiot in infancy. The photograph shows the oblique palpebral fissures and the large protruding tongue.

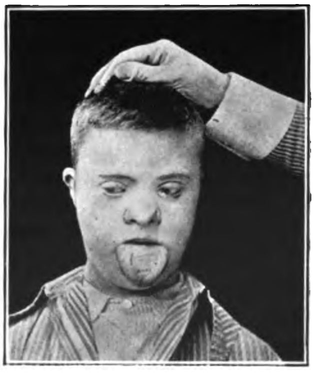


Fig. 119.—A Mongolian idiot, showing a large flabby tongue, which is deeply fissured.
(*Photo by Dr. S. A. K. Wilson.*)

rendered even broader by the presence of epicanthus; the eyelids show signs of chronic blepharitis; the ears are large and pitcher-shaped; the lips are fissured and often left open to allow a coarse tongue to protrude (*Figs.* 118, 119); the forehead is downy, and the hair of the scalp scanty, wiry, and frequently mouse-coloured; the complexion is florid and mottled. The almond-shaped eyes, the presence of epicanthus, the florid complexion, and the absence of fatty masses serve to distinguish the Mongolian from the cretinoid idiot; in case of doubt the benefit or otherwise of thyroid treatment may clinch the diagnosis.

Facies of Familial Lenticular Degeneration.—The characteristic facies of this disease is only seen in advanced cases, and may be described as one of fixed emotion. The slightest attempt to engage in conversation may evoke an expression of exaggerated mirth (*Fig.* 120) which takes a long time to wear off and is quite unlike that seen in other diseases of the nervous system, although perhaps related to the spastic smile of double hemiplegia. The accompanying photograph also illustrates the tendency to fall to one side or the other when in the sitting position.

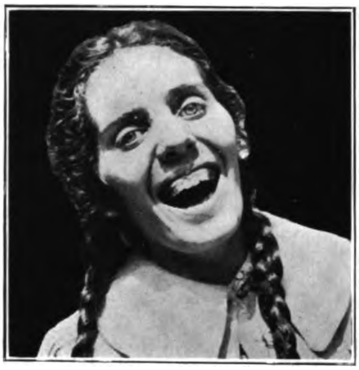


Fig. 120.—Facies of familial lenticular degeneration.

E. Farquhar Buzzard.

FÆCES, BLOOD IN.—(See Blood per Anum, p. 75; and Melæna, p. 385.)

FÆCES, FAT IN.—(See Fatty Stools, p. 239.)

FÆCES, INCONTINENCE OF.—(See Incontinence of Fæces, p. 313.)

FÆCES, MUCUS IN.—(See Mucus in the Stools, p. 398.)

FÆCES PASSED PER URETHRAM.—Fæces or fæcal fluid are only passed per urethram when the bladder is in fistulous communication with some part of the bowel, or with some fæculent abscess cavity infected with the *Bacillus coli communis*. Pneumaturia (p. 529) is liable to occur at the same time. The chief causes are as follows:—

Cancer of the bladder opening into the rectum or into some loop of bowel which has become adherent to the bladder.
Cancer of the rectum } opening into the bladder either directly, or through the medium of an intervening abscess.
Cancer of the sigmoid colon }
Cancer of the cæcum }
Cancer of the uterus opening both into the bladder and into the rectum.
Proctitis and periproctitis leading to the formation of an abscess which opens into the bladder.
Prostatitis and prostatic abscess opening into the rectum.
Rectovesical fistula from injury and sloughing, particularly after childbirth.
Caseous tuberculous disease opening both into the bladder and the rectum.
Appendicular abscess opening into the bladder.
An abscess resulting from acute diverticulitis (Telling's disease) opening into the bladder.

The passage of fæces in the urine may be simulated by some cases of very fœtid cystitis, when the bladder has been infected by the *Bacillus coli communis*.

If the symptom is due to cancer, it matters little which viscus is the primary site by the time the growth has involved both bladder and bowel. The diagnosis resolves itself, therefore, into one between malignant conditions on the one hand and non-malignant on the other. If malignant disease is not obvious, it will nearly always be advisable to resort to surgical measures in the hope of curing the primary condition—rectal, appendicular, prostatic, or otherwise. The commonest causes other than malignant are local sloughing of the parts after labour, and fæculent appendicular abscess opening into the bladder. The diagnosis will be suggested by the history and confirmed by local examination or exploration.

Herbert French.

FÆCES, PUS IN.—(See Pus in the Stools, p. 557.)

FÆCES, SAND IN.—(See Sand, Intestinal. p. 599.)

FÆCES, WORMS IN.—(See Parasites, Intestinal, p. 519.)

FAINTING ATTACKS.—(See Coma, p. 117.)

FAT IN URINE.—(See Chyluria, p. 108.)

FATTY STOOLS.—All stools contain a little fat; many contain more than they should, the fact being discoverable on analysis, although it may not be obvious to the unaided eye: the relative proportions of saponified and of unsaponified fats may have an important bearing on the diagnosis of pancreatic lesions (see Cammidge's Pancreatic Reaction, p. 100). Fatty stools in which the fat is obvious to the naked eye are rare; when they do occur they indicate one of three things: either that enormous amounts of fat are being ingested—more than can be absorbed by the normal mucosæ; or that the secretions are defective, so that even ordinary amounts of fat remain unabsorbed; or that the food is being hurried through the alimentary canal so fast that much fat remains undigested.

It is easy to exclude the first of these three possibilities by regulating the diet; the other two factors generally occur together, and the chief diseases in which fatty stools may be a prominent feature are:—

1. Those associated with severe diarrhœa, especially where the patient may be having an abundance of milk, as in:—Typhoid fever; Infantile diarrhœa; Sprue.
2. Those associated with jaundice, especially where the cause of the latter also prevents the pancreatic secretions from entering the duodenum, such as:—Chronic pancreatitis; Carcinoma of the head of the pancreas; Carcinoma of the duodenum, including the ampulla of Vater.

The diagnosis of Group 1 need not be discussed further here, for it will be indicated by other symptoms than the fatty stools. The different maladies belonging to Group 2, on the other hand, may be indicated directly by the fatty condition of the stools. If, for instance, there is doubt as to whether the patient is suffering from gall-stones obstructing the common bile-duct, or from chronic pancreatitis, the occurrence of pale abundant stools upon the surface of which an iridescent scum of fat is obvious, will be in favour of the latter, for fat can be digested to a far greater extent without bile but with pancreatic juice than it can be without the latter. The symptom affords no means of distinguishing inflammation from new growth, however; the distinction between these will depend mainly upon the duration of the symptoms—growth of the pancreas kills within a few months of producing fatty stools, whilst chronic pancreatitis may continue for years, or even get quite well. Other points to be on the watch for would be the presence of a tumour, of a dilated gall-bladder, or of secondary deposits. The age of the patient is seldom much help, for neither disease is common before adult life. The distinction between carcinoma of the head of the pancreas and carcinoma of the ampulla of Vater and duodenum may be next to impossible without laparotomy or post-mortem examination; although carcinoma of the head of the pancreas, rare though it is, is much commoner than new growth starting in the duodenum.

Herbert French.

FEVER.—(See Pyrexia, p. 563 and p. 571; and Hyperpyrexia, p. 309.)

FINGER, SORE.—Digital lesions may be erythematous, papular, vesicular, bullous, pustular, squamous, or ulcerative, representing a long list of cutaneous affections. The *erythematous* affections which may attack the fingers are erythema, lupus erythematosus, eczema, urticaria, chilblains, and frostbite; the *papular*, lichen planus and lichen annularis, pityriasis rubra pilaris, angiokeratoma, eczema, and papular syphilides; the *vesicular*, scabies, cheiropompholyx (dysidrosis), eczema, dermatitis herpetiformis, chilblains, the irritation set up by the habitual handling of sugar, or (in washerwomen) by immersion in water containing soda, or by contact with such vegetable irritants as rhus, mustard, thapsia, the common orange, eucalyptus leaves, arnica, etc.; the *bullous*, pemphigus, epidermolysis bullosa, dermatitis herpetiformis, scabies, leprosy, and syphilis (chiefly in infants); the *pustular*, scabies, boils, whitlow, impetigo contagiosa, eczema, and pustular syphilide;

the *squamous*, psoriasis, eczema, ichthyosis, lichen planus, syphilis, acanthosis nigricans, and verruca necrogenica; the *ulcerative*, bedsore, chilblains and frostbite, *x*-ray ulcer, dissection wounds, lupus vulgaris, lupus erythematosus, leprosy, chancre and syphilitic ulcer, epithelioma, Raynaud's disease, diabetic gangrene, trophic ulcer, and scleroderma.

The diagnosis of these various affections will be found under the names of the primary lesions—papules, vesicles, etc.—and here it is only necessary to particularize bedsore, diabetic gangrene, verruca necrogenica and dissection wounds, and chancre. *Bedsore* on the fingers is caused by friction between the knuckles and the bedclothes as the patient raises himself to the sitting position. It begins as erythema, and its significance can hardly be mistaken, though its presence in such a situation may take the nurse by surprise. *Diabetic gangrene* most frequently attacks the toes or other part of the foot; but occasionally it has been observed in the penis, and I have seen cases in which the fingers have been affected. *Post-mortem wart*, or post-mortem pustule, the condition sometimes met with chiefly on the knuckles and in the interdigital folds in those who have to handle dead bodies, whether of human beings by mortuary attendants or of the lower animals by butchers and slaughterers, is a form of tuberculosis, caused by infection with living bacilli from the dead tissue. It is sometimes met with also in colliers, in whom the site of inoculation is probably an abrasion received in the handling of coal. The pustule, beginning as a flat papule, dries up and forms a scab, which, when it falls off, leaves a surface that is made irregular by overgrowth of papillæ. These grow and become harder, until they form a warty mass. The avocation of the patient will suggest the true nature of the lesion. Of *dissection wounds*, consisting of pustules or small abscesses on the site of a puncture or scratch, or of lymphangitis and cellulitis, which may be followed by pyæmia, the history will supply the diagnosis. In *chancre* of the finger, usually met with in midwives, nurses, and medical men, but occasionally in others, a favourite situation of the sore is at the lateral nail-groove, and in many cases the lesion first attracts notice as a persistent fissure. If the sore undergoes induration, and there is general enlargement of glands with the other well-known secondary symptoms, the diagnosis can no longer be doubtful.

Dairymaids, milkers, and other farm hands sometimes develop acute or chronic sores upon their fingers due to *cow-pox* caught from the teats or udders of infected cows, and such patients may inoculate others who have no work that is connected directly with cows. The appearances are those of persistent boils or whitlows, and the diagnosis may be very difficult unless the source can be traced. Two other varieties of whitlow may pass entirely without recognition unless bacteriological methods are resorted to, namely, onychia or perionychia due to Klebs-Loeffler bacilli (digital diphtheria) and similar trouble due to the *Bacillus coli communis*. *Malcolm Morris.*

FINGERS, CLUBBED.—(See Clubbed Fingers, p. 111.)

FINGERS, DEAD.—(See Dead Fingers, p. 162.)

FINGERS, NUMBNESS OF.—(See Sensation, Abnormalities of, p. 604.)

FITS.—(See Convulsions, p. 143.)

FLATULENCE.—It is important to distinguish between (1) *Gastric flatulence*, in which wind is eructated; and (2) *Intestinal flatulence*, in which it is passed per anum.

Gastric Flatulence.—Before concluding that excess of gas is being produced in the stomach, it is necessary to exclude the possibility of *air-swallowing* (*aërophagia*, *eructatio nervosa*). This is common, but is apt to be interpreted wrongly. It is met with often in women about the menopause; it is also by no means infrequent in young men prone to be 'neurotic,' or to exhibit signs of neurasthenia or psychasthenia, though otherwise healthy. Eructatio nervosa is recognized by the violence of the belching and the excessive amount of wind expelled. It comes on in attacks both by day and by night, sometimes waking the patient. If a patient can belch 'to order,' one may conclude with almost perfect certainty that he is suffering from this form of neurosis; and by watching him during the attack one can recognize that he is gulping down air.

True gastric flatulence is present to a greater or less degree in many—one might almost say in all—forms of gastric disorder. For purposes of diagnosis one must distinguish between the cases in which gas is produced by *fermentation* in stagnating gastric contents, and those in which no such fermentation is taking place. In the former the stomach is dilated, vomiting is almost certainly present ; if examination of the gastric contents shows delay in their transmission, and the presence, probably, of sarcinæ (*Fig.* 121) and yeasts, one may diagnose pyloric obstruction, either simple or malignant. In these cases the eructations are sometimes offensive, revealing the existence of putrefaction in the gastric contents. Bismuth and x-ray examination will confirm the delayed emptying of the stomach.

Non-fermentative flatulence occurs in almost all forms of functional disorder of the stomach ; but is specially prone to occur in gastric atony. In that case there will be a well-marked splash over the gastric area, even some hours after a meal, but without any evidence of actual dilatation of the organ, although there may be some gastroptosis. (See also INDIGESTION, p. 315.) In other forms of gastric disorder flatulence is only a minor symptom, and of little diagnostic value.

Flatulence is also not an uncommon symptom in *emphysema of the lungs*, and in cases of *cardiac disease*, especially when due to degeneration of the heart muscle. In elderly persons these conditions should always be looked for. In *angina*, also, flatulence may be a prominent symptom, but in that case the attacks tend to come on after exertion, and are accompanied by the characteristic pain of angina.

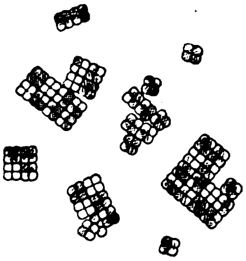


Fig. 121.—Sarcinæ Ventriculi. (Medium power of the microscope.)

Intestinal Flatulence may be either *acute* (see METEORISM, p. 388), or *chronic* (intestinal flatulence proper). In the latter case it is often attended by colicky pain, which is relieved by the passage of wind. It is important to note that flatulence is not a feature of ordinary constipation. When marked, it is suggestive either of chronic obstruction or of intestinal fermentation.

If *obstruction* be present, coils of intestine undergoing peristaltic contraction are often to be seen, and there is pronounced constipation, sometimes alternating with diarrhœa. A diagnosis of the exact cause of the obstruction may necessitate the use of the sigmoidoscope, bismuth and the x-rays, or even of an exploratory operation. In cases of *intestinal fermentation*, either constipation or diarrhœa may be present. Microscopic examination of the stools is often of help in elucidating the nature of the fermentative process, undigested muscle fibres (proteid fermentation or putrefaction) or an excess of starch cells (carbohydrate fermentation) being seen. (See also DIARRHŒA, p. 170.) *Robert Hutchison.*

FLUSHING.—The difference between flushing and blushing is that the former only occasionally, the latter invariably, arises from emotion—shyness, shame, and modesty. A flush may begin instantaneously in all the parts in which it is felt, or, arising in a lower region, it may ascend to the head, or, beginning in the head, it may descend to some part of the body, or it may pass both upwards and downwards. The sensation varies in severity, and may be actually painful. The nerve-storm generally ends in a cold stage, though this may precede the hot stage. The cutaneous symptoms may be accompanied or followed by nausea, vomiting, fainting, a sense of suffocation, numbness, tremors, tinnitus, giddiness, palpitation, paresis. The physical states and conditions from which flushing arises include menstruation and menstrual irregularities, the climacteric, pregnancy, lactation, chlorosis, indigestion, feeble circulation, general debility ; it may also be an expression of emotion, may be caused by alcoholic indulgence, or may merge into an epileptic aura. If it becomes chronic, the skin of the face, especially of the flush area—the middle third of the face—is reddened permanently, and the case becomes one of *rosacea* ; sooner or later the superficial vessels undergo dilatation ; hypersecretion and retention of sebaceous matter follow, and inflammation may be set up ; the inflammatory process, becoming chronic, may give rise, especially if the patient is much exposed to the weather, to hypertrophic thickening of the skin of the nose, with lobulation (rhinophyma).

The condition or habit which is the cause of rosacea will be deduced from the history, especially as regards tea, alcohol, and dyspepsia, and from examination of the patient. Rosacea is distinguished from *acne vulgaris* by the absence of comedones, the redness of the affected surface, the limitation of the eruption to the face, the telangiectasis, the hypertrophy, and by its being an affection of middle life rather than of puberty. It differs from *lupus erythematosus* in the absence of scaliness and of atrophic scarring, in the border which is not raised and shows no signs of active spreading, and by its fluctuations. *Seborrhœic eczema* may be met with in the flush area, but it is usually associated with seborrhœa capitis, there is no telangiectasis, and the affected surface is oily or scaly. From *tertiary syphilides*, rosacea is distinguished by its slow course, its symmetry, the dilatation of blood-vessels, and the absence of any tendency to ulceration and scarring, or to atrophy. In syphilis, further, there will be the stigmata or the history of earlier lesions.

Malcolm Morris.

FOOT-DROP.—(See PARAPLEGIA, p. 510; and PARALYSIS OF ONE EXTREMITY (LOWER), p. 496.)

FOOT, ULCERATION OF THE.—(See ULCERATION OF THE FOOT, p. 735.)

FOREHEAD, ENLARGEMENT OF.—(See ENLARGEMENT OF THE FOREHEAD, p. 203.)

FORGETFULNESS.—(See AMNESIA, p. 19.)

FORMICATION.—(See PRURITUS, p. 540.)

FOUL BREATH.—(See BREATH, FOULNESS OF THE, p. 86.)

FRACTURE, SPONTANEOUS.—Spontaneous fracture signifies fracture of a bone from causes which ordinarily would have been inadequate. Tremendous muscular efforts sometimes lead to the breaking of bones without any external violence, but this variety would not be included under the heading of spontaneous fracture if the degree of muscular effort seemed adequate. A man has been known, for instance, to dive into shallow water, and in order to bring himself to the surface quickly, and prevent his head from striking the bottom, he has used his neck muscles so strenuously in bending his head back as actually to fracture his vertebræ. This fracture is not spontaneous, but due to excessive muscular exertion. There are three main groups of causes for true spontaneous fracture, namely, excessive brittleness, or innate lack of strength of the bones—*fragilitas ossium*; general paralysis of the insane; and unsuspected lesions of the bones, particularly myeloid sarcoma, chloroma, tuberculous caries, or secondary deposits of carcinoma or sarcoma.

Fragilitas Ossium.—When the first fracture occurs in such a patient, there may be doubt as to the diagnosis; but when repeated breaking of different bones occurs, in each case from apparently trivial causes, the diagnosis becomes clear. The undue fragility may show itself in early life, but more often not until the patient has reached adult stature and weight. There is a very remarkable familial type of the disease, in which successive generations contain some members who have fragile bones amongst others who are healthy; the latter have white sclerotics, whilst those exhibiting fragilitas ossium have sclerotics that are definitely blue—sometimes even dark blue. Both bones and sclerotics lack part of their proper matrix; the diagnosis is easy, though the condition, termed 'blue sclerotics with brittle bones,' is rare.

Two maladies which differ from fragilitas ossium, and yet which may cause undue bending, or partial or green-stick fracture of bones, are *rickets* in children, in which disease, for a time at least, there is excess of preparation for bone formation, but deficiency in completing the ossifying process, so that the bones, being unduly soft, not only bend, but also give way as a green stick would, causing the partial or green-stick fracture; and *mollities ossium*, a rather rare affection in this country, though reported to be less uncommon in certain parts of the Continent, notably in the Rhine valley, coming on especially after pregnancy, and associated with concentric thinning of the bones from the marrow outwards, so that they eventually consist of a mere shell, which bends with undue ease, and may sometimes break spontaneously. The relationship to pregnancy may suggest the diagnosis, and there is no other disease which produces the same degree of pathological softening and fragility of the bones in adults; active rickets is practically confined to young children, only a few cases having been recorded during adolescence and none in adult life.

Spontaneous fractures in *general paralysis of the insane* occur, like the aural hæmatomata of this disease, at a late stage when the patient is bedridden. They may arouse suspicion that the attendants have been unduly rough in their handling of the patient; but so atrophic do the tissues, and particularly the bones, become, that the latter may fracture from slight and otherwise inadequate causes. The diagnosis will have been made months or more previously, by reason, first, of the mental changes of the patient, particularly ideas of grandeur; and secondly, by the occurrence of convulsive seizures after there have been changes in the patient's mental condition for a longer or shorter time. The case is generally that of a man who has suffered previously from syphilis, for which treatment was not very prolonged, and whose business has entailed much mental hard work, and possibly worry, in a city. Confirmatory evidence may be obtained, if need be, by finding relatively large numbers of small lymphocytes in the cerebrospinal fluid removed by lumbar puncture, and Wassermann's serum reaction for syphilis may be positive.

Before concluding that spontaneous fracture of a bone is due either to neurotrophic causes, or to fragilitas ossium, it is important to exclude the possibility of primary or secondary *new growth* in the affected bone, or *tuberculous caries*. It may be that the patient is already suffering from a bony swelling, such as myeloid sarcoma, before the fracture takes place, or it may be known that there is, or has been, a primary growth elsewhere; for instance, in the pelvis, breast, stomach, or thyroid gland, in which case the spontaneous fracture of a bone would suggest that a second metastasis has occurred at the site of fracture, eroding the bone until it finally broke from a trivial cause. The chief difficulties arise, first, when there are no symptoms of the primary growth itself, for instance in the case of a diffuse carcinoma of the stomach of the indiarubber-bottle type; and secondly, when the patient is really suffering from tuberculous caries whose existence has been entirely unsuspected. As an instance, one might mention the case of a woman fifty years of age, who, seeming to be in perfectly robust health, was standing in her kitchen, when her son entered unexpectedly, causing her to start suddenly, giving her body a twist at the same time. This movement was followed immediately by paralysis of both legs, and it seemed as though the sudden muscular exertion had led either to a hæmorrhage or to a fracture-dislocation of the spine; the cause for the fracture was in itself inadequate, however, and it would not have produced the symptoms had there not been spinal caries which had been slowly eroding the bones for some time previously, until they now gave way as the result of what would otherwise have been a trivial movement. The diagnosis in cases of the kind depends chiefly upon remembering the possibilities, and not omitting a most careful examination of every part of the body. When the *x*-rays are available, they may sometimes be of considerable value in detecting a neoplasm (*Fig.* 286, p. 673) or a tuberculous focus (*Fig.* 195, p. 460) in the affected bone *Herbert French.*

FRAGILITAS OSSIUM.—(See FRACTURE, SPONTANEOUS, p. 242.)

FREQUENCY OF MICTURITION.—(See MICTURITION, ABNORMALITIES OF, p. 393.)

FULLNESS, SENSE OF.—A sense of fullness is experienced when the tension exerted on the muscle fibres of the stomach or intestines is greater than normal.

Localization.—A sense of fullness felt in the upper part of the abdomen, in the neighbourhood of the umbilicus, and in the lower part of the abdomen is generally due to distention of the stomach, small intestines, and colon respectively.

1. **Gastric Fullness.**—The bulk of gastric contents, whether fluid or gas, required to produce a sense of fullness depends upon whether the tone of the muscles of the stomach is (*a*) Normal, (*b*) Excessive, or (*c*) Deficient. The sensation is the same in each case; the patient commonly believes it is due to excess of gas in the stomach, and it is generally thought that tone is deficient; but the former is rarely true, and the latter is certainly not more commonly the case than the reverse.

(*a*). In normal individuals the sensation of fullness is produced by eating very rapidly, as the intragastric pressure rises owing to the relaxation of tone, which should proceed *pari passu* with the increasing bulk of the gastric contents, taking place with insufficient rapidity.

(*b*). When the tone of the stomach is increased, a comparatively small quantity of food produces a sensation of fullness unless the food is eaten with extreme slowness. In rare cases the stomach is abnormally small, owing to infiltration of its walls with cancer (leather-bottle stomach, *Fig.* 132, p. 270): as its capacity cannot then increase at all by relaxation of its muscular coat, a very small quantity of food produces an immediate rise in intragastric pressure and a corresponding sensation of fullness.

(*c*). In atonic dilatation of the stomach the muscle-fibres are relaxed completely before any food is eaten ; the weight of the food, however slowly it is eaten, stretches the fully relaxed fibres from the minute it is eaten, and a sensation of fullness is felt.

In slighter cases (hypotonus) the muscle-fibres are not relaxed completely, but complete relaxation occurs as soon as a small quantity of food has been eaten ; any further addition to the gastric contents produces a sense of fullness.

From these considerations it is clear that : (1) If a sense of fullness is only felt when an excessive quantity of food is eaten, the size of the stomach is probably normal, and the excess is the cause of the symptom ; (2) If it is only felt when food is eaten very rapidly, the size of the stomach is probably normal, and the bolting is the cause of the symptom ; (3) When it is felt in spite of the food being normal in quantity and eaten at the normal rate, it is due to hypertonus or hypotonus if it can be prevented by eating small meals very slowly, and to atony or to leather-bottle stomach if this is not the case.

The distinction can only be made with certainty, however, by estimating the tone of the stomach directly, or indirectly from its size, a hypertonic stomach being small, whereas a hypotonic or atonic stomach is large.

Although percussion gives some idea of the quantity of gas in the stomach, it does not help in the determination of its size, and auscultatory percussion and friction have been shown by means of the *x*-rays to be quite valueless so far as the stomach is concerned.

Splashing and succussion occur in the normal stomach after an ordinary meal ; if, however, they can be produced after drinking two ounces of water on an empty stomach, atony is probably present.

Inflation is the only method apart from the *x*-rays which gives definite information as to the size and tone of the stomach. The patient drinks on an empty stomach two quantities of water in rapid succession : 1½ drachms of sodium bicarbonate are dissolved in one and 1½ drachms of tartaric acid in the other. At the body temperature and atmospheric pressure, 1700 c.c. of carbon-dioxide are evolved. The normal stomach has a capacity of 600 to 1200 c.c. when filled rapidly : the 1700 c.c. of gas are therefore subjected to a considerable degree of tension ; a certain amount of discomfort is felt, and the gas is expelled rapidly on sitting up. When the tone of the stomach is excessive, the capacity is less than 600 c.c., and when it is deficient it is more than 1700 c.c. ; in the former case a sensation of painful fullness is produced, and the gas is expelled violently on sitting up ; whereas in the latter case no discomfort, and sometimes actual relief, is experienced, and the gas can be expelled only with difficulty. The tumour formed with a hypertonic stomach is generally situated too high to be accessible for palpation, but when it can be reached it is found to be firm and well defined ; with a normal stomach it is also firm and well defined, and is often visible through the abdominal wall, whereas with a hypertonic stomach it is soft, and its outline is less easy to determine by palpation and percussion. When the inflated stomach is outlined by means of palpation and percussion, the distance between the lesser and greater curvatures of the stomach should be between 3 and 4 in., and the greater curvature should reach within an inch of the umbilicus. The distance between the curvatures in a hypertonic stomach is less than 3 in., and the greater curvature is more than an inch above the umbilicus ; in atonic dilatation the distance is more than 4 in.. and the greater curvature often reaches below the umbilicus.

The size and tone of the stomach can be determined most accurately with the *x*-rays after a meal of porridge containing 2 oz. of barium sulphate. Owing to the adaptation of the tone of the normal stomach to the volume of its contents, there is little difference in the upper level of the semi-fluid chyme as seen in the erect position, whether the volume is 5 oz. or 2 pints, and the greater curvature is not more than an inch above or an inch below the umbilicus (*Fig.* 122). A hypertonic stomach is diagonal, or even horizontal, instead of almost perpendicular, as in normal individuals, and its lowest extremity is situated at least an inch and often considerably more above the umbilicus. An atonic stomach does not adapt itself to the volume of its contents : food taken when it is empty drops at once to its most dependent part, instead of being held up for a few seconds by the tonic contraction of the body of the stomach. As more and more food is taken, the upper surface of the gastric contents gradually rises, but it never reaches the height observed in normal and hypertonic stomachs after the first two or three mouthfuls of food are swallowed. The gastric tone is insufficient to withstand the weight of the food, and the greater curvature consequently sinks as the quantity of gastric contents increases.

Most patients ascribe a sense of fullness in the epigastrium to 'wind,' and try to relieve their discomfort by eructation ; as, however, it is rare for excess of gas to be present, the attempt leads to aërophagy. The sense of fullness is thus often aggravated by aërophagy, though primarily due to some other cause. In addition to ascertaining the tone and size of the stomach, it is therefore necessary to discover whether the sense of fullness is in part due either to excess of gas produced by fermentation or to secondary aërophagy. The presence of excess of gas in the stomach can be ascertained most readily by means of the *x*-rays, as it is often difficult to distinguish by percussion whether a resonant area corresponds to a collection of gas in the stomach or in the splenic flexure.

Excess of gas is only produced in the stomach when evacuation is delayed : this never occurs in a hypertonic stomach, and only in a normal stomach when an excessive quantity of food has been eaten. In atonic dilatation of the stomach stasis is never sufficiently great for much fermentation to occur, the only condition in which it is really active being dilatation due to pyloric obstruction. This can readily be distinguished from atonic dilatation with the *x*-rays, even before the onset of visible peristalsis and the characteristic vomiting of large quantities of food eaten many hours earlier, by the excessive activity of peristalsis, the occurrence of retro-peristalsis, and the presence of a large residue in the stomach six hours after a barium meal. The diagnosis of pyloric obstruction is confirmed if the passage of a stomach-tube at 9 a.m. shows that remnants of a dinner taken at 9 p.m. the previous evening are still in the stomach, nothing having been eaten or drunk in the interval.

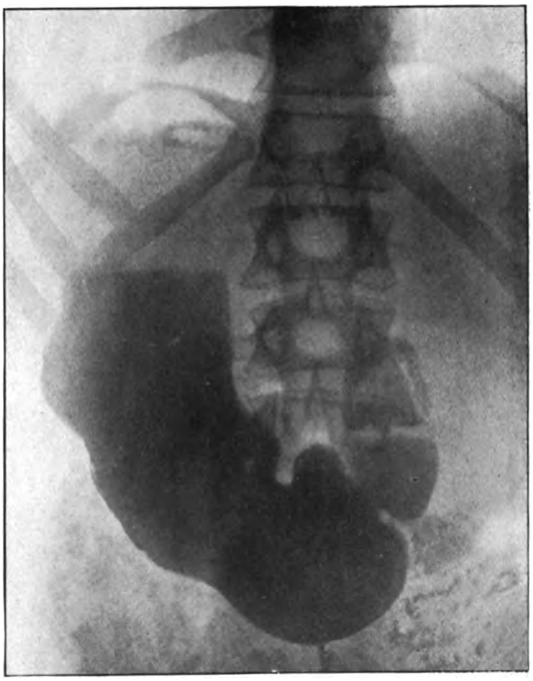


Fig. 122.—Skiagram taken in the vertical position, showing a bismuth meal in a normal adult stomach.
(*Skiagram by Dr. C. Thurstan Holland.*)

Aërophagy can be diagnosed with certainty if eructation occurs six or more times in rapid succession, or if it occurs before breakfast, unless food is present in the stomach as a result of pyloric obstruction. Finally, it is quite easy to watch the whole process of aërophagy with the *x*-rays.

2. **Intestinal Fullness.**—A sense of fullness in the lower part of the abdomen is almost invariably due to the excessive tension on the intestinal walls produced by the presence of an excessive quantity of gas. The only symptom which proves conclusively that this is the case is the passage of excess of flatus, particularly if it is found to relieve the discomfort. In the absence of evidence of a hypertonic condition of the colon, such as occurs in spastic constipation (*Fig.* 52, p. 124), a sense of fullness in the lower part of the abdomen may be presumed to be due to the pressure of an excessive quantity of gas. Spastic constipation is much more often accompanied by pain than a sense of fullness : it can be recognized by the contracted condition of parts of the colon, the situation and degree of the spasm varying from one examination to another, and with more certainty by means of the *x*-rays after a barium meal or a barium enema.

Intestinal flatulence may be due to the excessive production of gas in the intestines from excessive fermentation or putrefaction, retention of gas behind a fæcal mass in constipation, the passage of some of the swallowed air into the intestines in severe cases of aërophagy, and deficient absorption of gas due to the impeded venous circulation in cirrhosis of the liver and heart failure. The stools should always be examined: if they are bubbly, acid in reaction, and contain obvious excess of vegetable residue, intestinal fermentation is probably the cause; if they are alkaline in reaction, have a putrefactive odour and contain obvious fragments of meat, excessive putrefaction is probably present. On incubating some of the stool made thin with water for twenty-four hours in an apparatus in which the gas evolved can be collected, it is found that little or no gas develops and the stool remains neutral in reaction if there is no abnormal bacterial activity. When excess of gas is evolved, it is due to fermentation if it is odourless and the stools have become very acid, and to putrefaction if it has an unpleasant odour of putrefactive products and the stool is very alkaline.

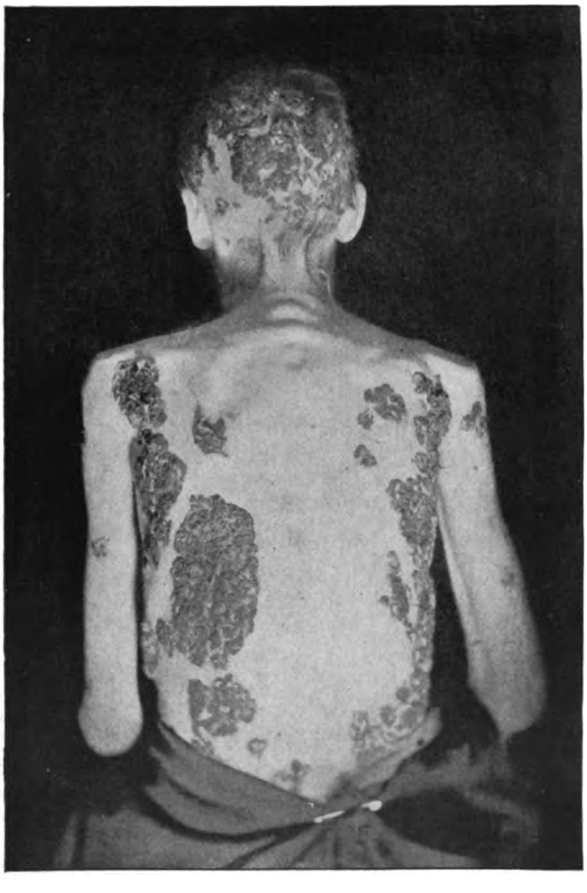


Fig. 123.—Favus: showing the very wide distribution over the body, as well as the scalp.
(From *Diseases of the Skin*, Sir Malcolm Morris.)

In the absence of excessive fermentation or putrefaction, a history of constipation would suggest that that is the cause of the flatulence (*vide* CONSTIPATION, p. 121). Aërophagy only leads to intestinal flatulence when it is so well marked as to be easily recognized (*vide supra*), and an examination of the liver and heart show whether it is due to deficient absorption of gas from cirrhosis or heart failure. *Arthur F. Hurst.*

FUNGOUS AFFECTIONS OF THE SKIN.—We here include (1) *Favus;* (2) *Ringworm;* (3) *Eczema marginatum;* (4) *Tinea imbricata;* (5) *Tinea versicolor;* and (6) *Erythrasma.*

1. **FAVUS** in man is due in about 99 per cent of cases to inoculation with the *Achorion Schonleinii.* Four other achorions, of animal origin, have been identified, and it has been proved that the affection can be communicated from animals to man, but the instances are so rare as to be negligible. Between the achorions on the one hand and the microsporons and trichophytons on the other (see p. 247), there are close morphological resemblances, but the clinical differences are well marked.

Favus, while showing a distinct preference for the scalp, may attack any part of the skin (*Fig.* 123), and even a mucous membrane. The characteristic lesion—a tiny sulphur-yellow disc with a cup-like depression in the centre, resembling both in colour and in shape a honeycomb, and in hairy parts pierced by a hair—can hardly be mistaken on account of its peculiar mousy smell. The lesion begins as a collection of whitish material, somewhat resembling a pustule, which grows and presently becomes dry and friable. The cup-like disc can then be detached from the epidermis, leaving a pimply, smooth, greasy surface.

As they grow, the discs often run together. In a later stage roughish crusts are formed, separated by pale, bluish-pink scars. The crusts, when broken up, are seen under the microscope to consist of spores, varying much both in size and shape, and of short threads of mycelium, which may penetrate into the mucous layer of the epidermis, and may even reach the derma ; this never occurs in trichophytosis. Hairs affected with favus are discoloured and lustreless ; they may fall out, but do not break off as in ringworm. Under the microscope one may see in favus-hairs segments of fungus 12–15 μ in length, dichotomized at an acute angle. If the nails are affected, the ungual cells will be found to be separated by irregular threads of mycelium, or by spores.

In the less characteristic cases the lesions must be examined closely under a good lens for remains of the yellow discs of favus or the broken hairs of ringworm. If, owing to applications to the skin, the crusts are lacking, treatment should be stopped for a few days, when the whitish points and the discs will usually reappear. In prolonged cases the crusts may be replaced by an irregular, lumpy, dirty-yellowish accumulation, but the odour of favus will still remain. At this stage the disease may resemble *psoriasis* of the scalp ; but there is a much greater loss of hair, the scales are less pearly, and even when no discs or sulphur-yellow scabs can be seen about the edges, the lustreless hair and the atrophic scarring left by the scabs are sufficiently distinctive of favus. The scarring may suggest *lupus erythematosus* of the scalp, but in that affection the crusting and the mouse-like odour are absent, while generally there are characteristic lesions on the face. From both *eczema* and *seborrhœa* favus is differentiated by the fact that its lesions are never diffuse, but always have a definite margin. In *alopecia areata* there is no scaling, crusting, or cicatrix.

2. **RINGWORM**—whether of the scalp, the beard, the hairless skin, the mucous membrane, or the nails—is due to fungi belonging to two different families, the microspora and the trichophyta, each of them comprising a number of different species. In the one case the affection is styled *microsporosis*, or tinea with small spores ; in the other, *trichophytosis*, or tinea with large spores. Eleven species of ringworm microsporons have been identified ; of the trichophyta, upwards of thirty. Only four species of the microspora, and the same number of species of the trichophyta, are of importance. The four microsporons are *M. Audouini*, *M. felineum*, *M. canis*, and *M. tardum* ; the four trichophytons, *T. crateriforme*, *T. acuminatum*, *T. sulphureum*, and *T. violaceum*. In both families some of the species are of animal origin, and it is these which account for nearly all the *inflammatory* forms of ringworm, including kerion.

Of the microsporons, the type species is *M. Audouini*, which is the cause of some 90 per cent of the juvenile ringworm of London. It is also the cause of much of the juvenile ringworm of Paris, though of much less than was the case a few years ago. *M. felineum* and *M. canis*, closely allied species, are responsible for an appreciable percentage of human ringworms—the one in England, the other in France. *M. tardum* is met with occasionally in France. Of the four clinically important species of trichophytons, the one encountered most frequently is *T. crateriforme* ; next comes *T. acuminatum*, then *T. sulphureum*, which, however, is not known in France ; and lastly *T. violaceum*. A fifth species of trichophyton, *T. rosaceum*, which chiefly affects the beard, but also the hairless skin, is said to be relatively not infrequent in Northumberland and Durham.

The division of the ringworms into a small-spored and a large-spored group may easily lead to confusion in diagnosis, for among both microsporons and trichophytons the spores vary considerably in size, according to the species. Those of the microsporons may be as large as 4 μ, while those of the trichophytons may be as small as 3 μ ; the limits of the one are 2 to 4 μ, and of the other, 3 to 8 μ. Clinically, therefore, microsporosis and trichophytosis are to be differentiated from each other not alone by the size of the spores, but also by their shape and arrangement and modes of growth.

First, as to *shape* : In microsporosis the spores are, speaking generally, round or ovoid ; in trichophytosis, they tend to be square with rounded angles, or oblong with sharper angles. Still more important, for diagnosis, is the *arrangement* of the spores. In microsporosis they are dotted about irregularly, and the mycelium interwoven with them is curved and branching, and irregularly jointed. In trichophytosis they are arranged in regular chains, and the mycelium is short and regularly jointed. In microsporosis the fungus forms a greyish sheath around the hair—whether of the scalp or of the body—which it eats away, fraying the edges, penetrating to the interior of the shaft, and growing downwards towards the root. Presently the hair breaks off, at some distance from the follicular orifice, and the parasitic sheath is disintegrated and may be seen as a patch of ash-coloured scales on the epidermis. In trichophytosis, the parasite attacks the root of the hair first, and grows upward. The hairs are broken off short, and no sheath is to be seen outside the follicular orifice. It should be added that some small-spored trichophytons form a sheath outside the hair like that of microsporosis, but the spores observe the chain-formation which is characteristic of trichophytosis, and this is never present in microsporosis. These small-spored trichophytons are all pyogenic, and are the cause of many cases of kerion.

Trichophytons may be either *endothrix* or *endo-ectothrix*. If the parasite penetrates the hair between the cuticle cells and develops entirely within the hair-structure, it belongs to the endothrix class. If it develops not only within the hair, but also continues to proliferate in the follicle outside, it must be allocated to the endo-ectothrix class. The great majority of the cases of scalp trichophytosis, both in London and in Paris, are due to endothrix infections ; but the endo-ectotriches are responsible for most of the ringworms of the hairless skin, for nearly all the adult ringworms, and for the majority of specially inflammatory cases.

Sabouraud divides the endotriches into (1) True endotriches, and (2) Neo-endotriches, the distinguishing feature between them being that in the latter the early (*neo*) stage of the attack, the stage in which the outside of the hair is assailed, is prolonged. If the parasite is a true endothrix, it is easy to miss the invasion stage, so short is it ; if it is a neo-endothrix, the invasion-stage is so protracted that it is possible to mistake the case for one of trichophytosis due to an endo-ectothrix. The endo-ectotriches are subdivided into those with large spores (megaspores), and those with small spores (microïdes). It is the latter which, as mentioned above, may be mistaken for microsporons, unless the chain-formation be looked for. In the case both of the microspora and of the trichophyta cultures may have to be grown to distinguish between the different species. There are four microspora of human origin, and these give either a small or medium culture ; to this group belong *M. Audouini* and *M. tardum.* The seven species of animal origin yield a large, enduring culture ; in this group occur the two remaining species of clinical importance, *M. canis* and *M. felineum.* In the first group pleomorphism is never met with ; in the second, on a suitable medium, a white downy pleomorphism, quite different from the mother culture, is exhibited.

Of trichophyton cultures there are four main types : (1) The crateriform or acuminate ; (2) Those with large white growths, either powdery or velvety ; (3) The faviform ; (4) A single species, *Epidermophyton inguinale*, which is the cause of eczema marginatum. In the first group, to which belong all the four clinically important species, the culture resembles the crater of a volcano, and is white, cream, or primrose-coloured, or it is like a mountain peak (' acuminate ') and is grey or yellowish in colour. The parasites of this cultural group are all endotriches. In the second group the cultures are very large and white, some of them powdery, others velvety. The species which yield cultures of this type are all endo-ectotriches, and are of animal origin. The three species which give cultures like those of the parasites of favus, although the clinical course of the lesions and the appearance of the fungus in the hair leave no doubt that they are trichophytons, are also of animal origin. The *Epidermophyton inguinale* yields a yellow-orange culture, dry and powdery, but often white and velvety as the result of pleomorphism.

Ringworm of the Scalp (*Tinea tonsurans*).—Both the small-spored and the large-spored ringworm of the scalp begin alike as a small red papule, which develops near the orifice of a hair-follicle ; the size, and yet more the shape and arrangement of the spores, and the way in which the hair is attacked, help to distinguish between them ; in trichophytosis there is a much smaller number of stumps to be seen with the naked eye, and on the surface of the scaly patches, among the remaining healthy hairs, one may detect those dark points to which the affection owes its name of ' black-dot ringworm.' These dots are pigmented, coiled-up hair-stumps. If the whole scalp is thus affected, the case becomes one of ' disseminated ringworm.' In trichophytosis, again, the scales are scantier, or may even be absent, and the outline of the lesions is not so rounded or so well-defined. As a rule it is not difficult to distinguish tinea tonsurans, whatever its form, from other scalp affections, the clinical picture—the broken hairs, the black dots, the slight scaliness, the prominent follicles, the baldness, in varying degrees, of the involved area—being sufficiently distinctive. In *favus* there is the same dull and brittle condition of the hair, but the patches are not generally circular, while in ringworm the cup-shaped crusts are absent, there is no mousy smell, nor is the skin atrophic. The broken hairs distinguish tinea tonsurans from *pityriasis* of the scalp and from *psoriasis* of the hairy skin, in both which affections the hairs fall out unbroken. In psoriasis, too, there is a greater degree of scaliness, generally it is not the scalp only that is affected, nor is loss of hair usual, though it occurs sometimes. In the anomalous form of ringworm known as *tinea decalvans*, or bald ringworm, in which the hair falls out in places leaving smooth bare patches, confusion with *alopecia areata* may be avoided without much difficulty; the billiard-ball smoothness of the patches in the latter condition is not present in ringworm. Another differential feature is the shape of the short hairs found at the edge of the patches: in tinea tonsurans they are bent, whereas in alopecia areata they may be compared to a note of exclamation. In the latter condition, too, the hairs that remain are free from fungus. In the infrequent cases of inflammatory ringworm, a condition somewhat resembling *impetigo* or *eczema* may be set up; but the broken stumps and the limited area of the affection, together with the history of the case, should prevent confusion with those affections. In these forms of ringworm again, the lesions are sharply defined, and the pustules are invariably situated round the hairs. *Seborrhœa* can be ruled out by remembering the greasiness of the scales, the diffusion of the condition over the whole scalp, and the absence of patches of baldness.

Ringworm of the Beard (*Tinea sycosis*).—From ordinary *sycosis* this affection is distinguishable by its more rapid spread, and the greater lumpiness of the affected surface. In sycosis vulgaris, too, the pustules are usually pierced by a hair, and are quite small, and unless there is much more suppuration than is usual, the hairs do not fall out. Tinea sycosis differs from *eczematous folliculitis* in the absence of the serous discharge that marks the latter affection. In the eczematous condition, again, there is but slight if any loosening of the hairs, so that if they are extracted they bring with them their root-sheaths. The affection is not confined to the hairy parts, as in tinea sycosis, nor do the patches assume the ring-like form. The ring-formation is absent also in *seborrhœa*, nor is the hair involved as in beard-ringworm. In the *circinate tubercular syphiloderm*, the border of the lesion is darker in colour and more infiltrated, and there is either atrophy or pigmentation, or both. Occasionally the severer cases of ringworm of the beard take the form of a single tumour-like formation which may be mistaken for a *carbuncle*, but the inflammation is almost always less active than in carbuncle, and the swelling and pain are correspondingly less. In any clinically doubtful case examination of the hairs under a microscope will show whether or not the case is one of beard ringworm by revealing the presence or the absence of the trichophytic fungus.

Ringworm of the Body Skin (*Tinea circinata*).—The small, red, slightly raised spot which is the first visible lesion of ringworm of the body, gradually spreads at the edge and becomes scaly. Fading away at the centre, the redness leaves a slightly discoloured branny area which forms the inside of a red ring. The circle slowly enlarges without any widening of the edge. Usually, though not always, there are several rings, sometimes, though seldom, arranged concentrically, and those adjoining each other may run together. Frequently, as the edge advances, there is no involution in the centre, the lesions then appearing not as rings but as patches. As a rule inflammation is present in varying degrees, and the neighbouring lymphatic glands may be slightly enlarged.

These symptoms, with the tingling and itching, form an *ensemble* which can hardly be mistaken for any other affection. In *eczema seborrhœicum* the scales are greasy, and often there are projections into the glandular openings. In *psoriasis* the skin is affected in ring-like areas, but all the other characters are different. From the *circinate tubercular syphiloderm*, ringworm of the body may be distinguished in the same way as ringworm of the beard (see above). As a rule microscopic examination will disclose the ringworm fungus—usually a trichophyte—without difficulty; but occasionally the parasitic elements are deep-seated, and must be sought in a section of the affected tissue.

Ringworm of the Nails (*Onychomycosis*) usually, though not invariably, appears in association with trichophytosis of the beard or of the body skin. The first visible lesion shows as greyish stains under the borders of the nail and at the root. Inflammation of the matrix follows and the structure of the nail degenerates, becoming thickened, spongy, and more or less brittle, with a dulled surface. When exfoliation occurs a mass of disintegrated nail substance is seen, in which the fungus may be found.

Similar changes may arise in connection with gout, rheumatism, and other constitutional disorders—those for instance in which there is impaired nutrition—as well as in such inflammatory affections as eczema and psoriasis. From all such cases, the presence of the parasitic elements will suffice to differentiate onychomycosis. In the onychomycosis of favus, the stains under the borders of the nail are yellower, and the mycelial elements shorter and less regular.

3. **ECZEMA MARGINATUM.**—In this form of ringworm of the body, more frequent in tropical climates than in Europe, the parts attacked chiefly are the lower portion of the abdomen, the groins, the buttocks, the fold of the nates, and the axillæ—parts, that is, where the skin surfaces are in contact. The hair is never involved. The characteristic feature of the lesions is their broad, bluff margin ; it is scaly, and as a rule papular. They are often eczematoid, but they can be distinguished from eczema and from eczema seborrhœicum by their gradual spread and broad, elevated margin and by the ring-like formation of the early stage. If any doubt remains, the microscope will clear it up by revealing the parasite, the *Epidermophyton inguinale.*

From eczema marginatum, *dhobie's itch* is differentiated very imperfectly. It is in fact a popular name for all epiphytic skin diseases of warm climates, but usually it connotes diseases of this group of which the sites are the inguinal regions and the axillæ. Castellani distinguishes two fungi as the cause of dhobie's itch, besides *Epidermophyton inguinale,* namely *E. Perneti* and *E. rubrum* ; and Manson holds that, in many cases, the parasites concerned are *Microsporon minutissimum* and *M. furfur.* For practical purposes, dhobie's itch may be regarded as another name for eczema marginatum.

4. **TINEA IMBRICATA**—known also as Tokelau ringworm—was formerly peculiar to certain oceanic tropical climates in the East, but now has a wider distribution. The fungus has not yet been classified definitely. Sabouraud holds it to be a trichophyton allied to species of animal origin met with in Europe ; by other authorities it is regarded as a lepidophyton ; so far it has not been cultivated. The affection to which it gives rise is characterized by a concentric arrangement of closely-set rings of scaly epidermis. The conditions from which it has to be distinguished are *tinea circinata* and *ichthyosis.* From the former it is differentiated by the greater abundance of the fungus elements, the tendency of the process to spread centripetally, the absence of marked inflammation or congestion of the rings, their concentric disposition, and the greater size of the scales. From the latter, by the presence of the fungus, the concentric arrangement of the scales, and the fact that the attached border of each scale is towards the periphery, the free border being towards the centre of the circle, or group of circles, to which the scale belongs.

5. **TINEA VERSICOLOR**—often styled pityriasis versicolor—is caused by the *Microsporon furfur,* the mycology of which is little understood. The disease is contagious, but only in a low degree. The lesions, confined to the horny layer of the epidermis, take the form of roundish, scaly patches, with a definite margin, and of a colour varying from fawn to liver—in coloured races, grey or white ; in persons who have lived in warm climates, it may be black. The hair is not assailed, nor are the hands and feet. As a rule the lesions are limited to the trunk, particularly the front of the chest, but occasionally they extend to the upper parts of the limbs : they have been mistaken for secondary syphilides, but the colour and distribution, and the large patches in which they are found, should serve to obviate the confusion. In exceptional cases the face may be invaded, and the affection might then be confounded with chloasma. From *pityriasis rosea* and from *eczema seborrhœicum* it may be distinguished by the absence of inflammatory reaction, except in persons who perspire freely ; in pityriasis rosea, too, the upper parts of the limbs are affected equally with the trunk. The lesions of tinea versicolor offer some resemblances to the pigmentary patches sometimes met with in *leprosy* ; but from these, as from the other cutaneous manifestations mentioned, they may be differentiated almost certainly by the ease with which the scales can be detached by a stroke of the finger-nail, and quite certainly by the fungus elements which may be detected in the scales after these have been treated with potash. The spores are rounded and, like the mycelium, have a double contour with a diameter of 3 to 5 μ ; they are generally grouped together in masses, suggesting a resemblance to bunches of currants.

6. **ERYTHRASMA,** due to the *Microsporon minutissimum*, presents several points of resemblance to tinea versicolor. In both it is only the horny stratum of the epidermis that is affected, nor is the hair ever attacked. In both there is but a low degree of contagiousness. The lesions offer some likeness to those of tinea versicolor, but they are reddish-brown in colour, and their usual site is the genito-crural region or the axillæ, or both, though occasionally, in fat subjects, there may be extension to the abdominal and submammary folds and those of the large joints. In rare cases, erythrasma resembles one type of eczema marginatum, but is distinguished from that affection by its low degree of contagiousness and slow evolution, and by the absence of inflammation, which also distinguishes it from eczema seborrhœicum and from pityriasis rosea. Any doubt between erythrasma and any other affection, including tinea versicolor, may usually be cleared up by examination of a preparation under a microscope of sufficiently high power. The spores of *M. minutissimum*, like the threads of mycelium, are extremely minute, having a diameter of about 0·6 μ. The mycelial threads, of the same diameter, are so abundant and so twined together as to form, here and there, a network over the epidermic cells.

Malcolm Morris.

GAIT, ABNORMALITIES OF.—As a genuine aid to diagnosis the gait cannot be of much real assistance. There are, however, several diseases and affections which produce manifest, and in some cases peculiar, alterations in gait. In some respects, indeed, the gait is a diagnostic point in identity, though this probably also depends on the total back or front view of the individual, rather than on real peculiarities of gait as such.

In analyzing gait for diagnostic purposes, we find that it consists of co-ordinate and painless movements of the muscles of the lower limbs and pelvis—often, indeed, sinking into purely reflex, or at least subconscious, movements—and these are associated, in easy and ordinary walking, with rhythmical movements of arms, body, and head. The directions, therefore, in which it can be disordered are: (1) *Inco-ordination*; (2) *Local loss of power*; (3) *Pain calling attention to the movements.*

1. **Inco-ordination.**—The test for the presence of this is the complaint of the patient that he feels unsteady in walking, especially on turning or walking on uneven ground, or on walking or standing with the eyes shut; and if co-ordination only be at fault, it will then be found that on testing the legs for simple movements, such as flexion and extension, the power of the muscles is unimpaired. Having discovered inco-ordination, the next question is, to what may this be due? *Tabes dorsalis*, *ataxic paraplegia* (combined lateral and posterior sclerosis), *disseminated sclerosis*, and *hereditary ataxy* (Friedreich's disease), are far and away the commonest causes of this, in the order of mention; their differential diagnosis depends on many other symptoms and signs, discussed elsewhere. *Cerebellar disease* causes rather a reeling in the gait than a simple inco-ordination in the individual movements; and here again, other symptoms will be to the front. Localized *paralyses of eye muscles* may also cause inco-ordination; this will probably cause complaints of double vision, and may be diagnosed by the fact that the patient walks better with one eye shut than with both open—in cases of some duration it is quite likely that this simple test will not discover which is the affected eye.

2. **Local Loss of Power** is well illustrated by the waddling gait of *pseudo-hypertrophic paralysis*, calculated to get the weight of the body as speedily as possible on the foot as a basis. The diagnosis depends on the peculiar way in which the patient climbs up himself (see PARAPLEGIA, p. 510). Another condition in which the loss of power is due, not to the muscles themselves, but to the position of their attachments, is seen in *congenital dislocation of the hips*; the gait here, too, is waddling, the lower part of the back exhibits extreme lordosis, and the belly is thrown forward through attempts to balance the pelvis on the loose supports at the hips.

Other forms of local loss of power betray themselves by a limp or by a dragging of the foot or leg, and (or) peculiar positions of the feet, and possibly by wasting of muscles generally or locally; measurements must of course be made if wasting be suspected. *Infantile paralysis*, and *old hemi-* or *mono-* or *para-plegias* are the common causes of this, if it be unassociated with pain, and enquiry must be made as to mode of onset and duration, in completing diagnosis.

3. **Pain on Walking** is at once obvious, because complained of by the patient; acute inflammatory troubles of muscles, joints, or tissues will be obvious on examination, and

chronic joint troubles, osteo-arthritis, etc., may be discovered easily, chronic gonorrhœa or pyorrhœa alveolaris not being forgotten as possible causes of these. One thing that may escape observation is hip-joint disease, when pain in the knee may be the complaint.

The only other caution we can administer here, is to warn practitioners against any hasty conclusions as to the nature of a disease from the gait; the high-stepping gait of tabes, the shuffling gait of lateral sclerosis, the festinating gait of paralysis agitans, are all easy enough of recognition when a diagnosis is made, but are too frequently absent or atypical to allow much diagnostic superstructure to be built on them alone.

Fred J. Smith.

GALL-BLADDER ENLARGEMENT.

Physical Signs.—The only physical method of examination which is of material assistance in detecting enlargement of the gall-bladder, is palpation; inspection, percussion, and auscultation seldom help. On careful palpation one may feel an oval, smooth swelling, which may be no larger than a hen's egg, or as big as a swan's, moving downwards close behind the anterior abdominal wall when the patient inspires, descending either from beneath the right costal margin near the tip of the ninth rib, or approaching the under surface of an enlarged and palpable liver in the right nipple line. The tumour generally extends inwards as well as downwards as it grows, so that it may ultimately cross the middle line below the level of the umbilicus. It may be large enough to be palpable bimanually in a thin patient; but it seldom fills out the loin in the way that a renal tumour would. It may or may not be tender, according as the cause of the enlargement is associated with inflammation or not; it feels firm and tense rather than hard; on careful percussion it may be found to give an impaired note, but it is seldom quite dull unless it is very big.

Diagnosis from other Swellings.—It has to be distinguished particularly from four groups of conditions which may simulate it:—(1) *From carcinoma* arising in the bile-ducts or gall-bladder, and replacing the latter with new growth; (2) *From tumours* in or attached to the liver in the neighbourhood of the gall-bladder; Riedel's lobe; secondary new growth; or more rarely gumma, abscess, or hydatid cyst; (3) *From movable kidney* or *hydronephrosis*; (4) *From tumours in organs in the neighbourhood*, such as carcinoma of the pylorus, carcinoma of the duodenum, carcinoma of the transverse colon, carcinoma or sarcoma of the right suprarenal capsule or right kidney.

Carcinoma of the Gall-bladder.—It may be difficult to decide whether a given mass is merely an enlarged gall-bladder, or a growth replacing the latter; in either case there may be a history of gall-stones, with biliary colic, pyrexia, and even jaundice, extending over years; for primary new growth of the gall-bladder is nearly always secondary to, and associated with, gall-stones. The rapidity of the enlargement, in the absence of any definite cause, may suggest growth, particularly in a person of the cancer age; careful palpation may show that the mass is not smooth as most gall-bladder enlargements themselves are, but more or less nodulated or covered with bosses or irregularities, which in themselves suggest new growth; in some cases there may be secondary deposits in the liver, and sometimes the enlargement of the left supraclavicular gland points to malignant disease with metastasis. Notwithstanding these points, however, the differential diagnosis may be so difficult that laparotomy will be resorted to in order to decide it.

The Tumours attached to or in the Liver that are most likely to be mistaken for enlargement of the gall-bladder, or vice versa, are Riedel's lobe, secondary carcinoma or sarcoma of the liver, and much more rarely gumma, abscess, or hydatid cyst. A *Riedel's lobe* (see p. 366) may be quite impossible to distinguish by physical examination from an enlarged gall-bladder or from a movable kidney. Owing to the absence of symptoms, there is seldom need for laparotomy; but sometimes the lobe arouses such alarm lest it be some more serious condition that laparotomy may be resorted to and the diagnosis verified in that way.

Secondary new growth in the liver, whether carcinoma or sarcoma, nearly always causes very considerable, and sometimes enormous, enlargement and great hardness of the organ, not infrequently associated with JAUNDICE (p. 331), ASCITES (p. 52), or both. The diagnosis depends, first, upon the discovery of a primary growth, which in the case of carcinoma is likely to be in the stomach, duodenum, pancreas, colon, or rectum; or in the case of sarcoma, the eye—some of the greatest enlargements of the liver being due to secondary deposits of melanotic sarcoma, secondary to a primary ocular growth ; and secondly, on the discovery in the liver of several separate nodules, some of which may be felt to be umbilicated, that is to say, depressed in their central part and raised around the edges.

Gumma of the liver is not very frequent nowadays, and when it occurs is apt to be mistaken for new growth unless there is an obvious history of syphilis, or the effects of tertiary lesions are visible elsewhere, especially gummatous lesions of the skin or tongue. The diagnosis may be confirmed by obtaining a positive Wassermann's serum reaction, or by the beneficial effects of giving salvarsan, or potassium iodide and mercury, though these drugs do not always cause a gumma of the liver to disappear rapidly. In cases that have come to laparotomy the diagnosis between gumma and new growth is by no means easy even when the liver is inspected.

Abscess of the liver (see p. 369), if it is to simulate an enlargement of the gall-bladder, is likely to be a single large one, which if it has not arisen in some pre-existent mass, such as a gumma, new growth, or hydatid cyst, is likely to have been acquired in a tropical country, where the patient may have suffered from amœbic dysentery. The diagnosis may not be evident until laparotomy is resorted to, or until the mass is punctured with an exploring needle, when the chocolate-and-milk appearance of the pus obtained may be characteristic.

Hydatid cyst of the liver is seldom situated in such a position as to cause difficulty of diagnosis from gall-bladder enlargement, the cyst being more often embedded in the liver substance, or projecting from its upper surface. The diagnosis might be arrived at if the patient were known to have had hydatid cysts elsewhere ; but in most cases it is only when laparotomy has been performed that the correct diagnosis can be made. It might have been suggested by the occurrence of eosinophilia, and also by a specific hydatid serum reaction, though neither of these is likely to be found unless the hydatid cyst has produced toxic symptoms, because latent hydatid cysts cause no symptoms.

The Distinction between an Enlarged Gall-bladder and a Movable Kidney or Hydronephrosis might seem to offer no difficulty ; but clinically the distinction is not always easy. There is often no jaundice to suggest gall-bladder trouble, nor need there be any obvious urinary changes to suggest kidney, so that the diagnosis has to be made chiefly by palpation. One would lay stress upon the fact that the gall-bladder is more easily felt anteriorly than posteriorly, whilst the reverse is the case with the kidney ; that the kidney is the more freely movable of the two, as a rule ; that it is seldom possible to demarcate the upper pole of an enlarged gall-bladder in the way that a movable kidney can sometimes be made out ; that with a kidney tumour the loin is dull, whilst with gall-bladder enlargement it is resonant ; and that, on rather firm bimanual palpation, the peculiar sickening sensation that the patient may complain of is more characteristic of kidney than it is of gall-bladder.

Tumours of other Organs simulating Enlargement of the Gall-bladder have to be distinguished partly by the fact that new growths of the pylorus, duodenum, transverse colon, or suprarenal capsule, big enough to simulate an enlargement of the gall-bladder, will seldom have the smooth oval outline that the latter nearly always possesses. There may, moreover, be distinct symptoms attributable to the primary growth, such as dilatation of the stomach, coffee-ground vomit, or there may be secondary deposits in the liver, in the left supraclavicular gland, or elsewhere, to indicate the diagnosis. It is not easy, however, to exclude enlargement of the gall-bladder without resorting to laparotomy in some of these cases.

The Cause of Enlargement of the Gall-bladder.—Having decided that a given tumour is an enlargement of the gall-bladder, it is necessary to determine to which of the following causes it is due :—

Empyema of the gall-bladder
Chronic pancreatitis
Carcinoma of the head of the pancreas
Cholecystitis from :— (i) Gall-stones ;
(ii) New growth
Typhoid Fever
Obstruction of the common bile-duct by a gall-stone
Obstruction of the cystic duct by gall-stone
Simple mucocele.

It is noteworthy that *gall-stones* lead to enlargement of the gall-bladder far less often than might be expected ; if the inflammation they lead to, and which leads to them, does not go on to empyema of the gall-bladder, the latter usually becomes thick-walled, contracted, and embedded in dense adhesions, the latter preventing it from dilating even when the cystic or common bile-ducts become obstructed by a stone. It is the exception to find a very big gall-bladder with gall-stones. Indeed, in a middle-aged patient in whom there has not been any very definite attack of biliary colic, the occurrence of progressive and considerable enlargement of the gall-bladder, associated with a deepening jaundice and no ascites, should always arouse serious suspicion of there being a *lesion of the head of the pancreas* which has extended along the pancreatic duct so as to occlude the common bile-duct gradually, the commonest cause of these symptoms being either *chronic pancreatitis* or *carcinoma* of the head of the pancreas. The greater the epigastric pain in such a case, especially if it is paroxysmal, and such as to suggest gall-stones, the more likely is the lesion to be chronic pancreatitis rather than new growth, and the suspicion may be confirmed by CAMMIDGE'S PANCREATIC REACTION (p. 100). There are, of course, cases in which gall-stones are the cause of the enlargement; but when this is so, there is nearly always tenderness over the gall-bladder, and pain when it is palpated firmly, associated with a rise of temperature, possibly with rigors, especially if the inflammation has spread to the bile-ducts (infective or suppurative cholangitis). Leucocytosis, with a relative increase in the polymorphonuclear cells, would indicate that in addition to gall-stones there is suppurative inflammation—that is to say, *empyema of the gall-bladder*—requiring surgical treatment.

Another important cause for empyema of the gall-bladder is *typhoid fever*. The diagnosis is not difficult as a rule, for there will be no question of new growth or of gall-stones in most of the cases, and the patient will have been suffering from a prolonged asthenic fever which will have been diagnosed already by Widal's test. Infection of the gall-bladder by typhoid bacilli is relatively common, and seeing that gall-stones are seldom if ever primary, but rather the result of preceding microbial inflammation in the gall-bladder, it is not surprising that gall-stones are more common in patients who have previously had typhoid fever than in other persons. Apart from gall-stone formation, however, slighter degrees of inflammation of the gall-bladder by *Bacillus typhosus* are common, and it is thought that the continued infectivity of the excreta in typhoid-carriers is due to the constant discharge of infected bile from the gall-bladder, persisting sometimes for thirty years or more. In a certain number of typhoid patients rapid enlargement of the gall-bladder occurs owing to the bacillary infection, and there are instances in which the distention has become so great that the gall-bladder has ruptured spontaneously and produced general peritonitis. Sometimes the inflammatory products discharge themselves naturally by the bile-passages; but it is often necessary to open and drain the gall-bladder, the diagnosis of the nature of the empyema being settled by bacteriological examination of its contents. It is noteworthy that, whereas in uncomplicated cases of typhoid fever Widal's reaction rapidly becomes negative during convalescence, when there are persistent bacillary complications the serum test may remain positive, or at least partly positive, over much longer periods. When an empyema of the gall-bladder due to typhoid fever remains latent for weeks or longer, the nature of the case may be suggested by the previous history, and by the persistence of the positive serum reaction.

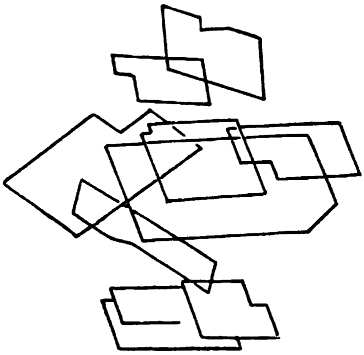


Fig. 124.—Cholesterin crystals. (High power.)

Simple mucocele of the gall-bladder is probably the result of former catarrh of the cystic duct, or of a gall-stone which has disappeared; in many cases it may be impossible to determine the precise cause; the gall-bladder may become greatly distended with perfectly colourless mucoid fluid, free from bile pigment, though sometimes containing crystals of cholesterin (*Fig.* 124). The fluid is sterile. There are usually no symptoms; the patient may by chance have discovered the tumour for herself. Such a mucocele may be mistaken for a movable kidney, and the diagnosis of the nature of the mass is sometimes obscure until operation is resorted to.

Herbert French.

GANGRENE.—When any necrotic tissue becomes infected with putrefactive micro-organisms, the resulting condition is known as gangrene—dry, moist, or spreading. (See also GANGRENE OF THE LUNG, p. 259).

CAUSES OF GANGRENE.

Local Traumatic Causes:—

Severe bruising or crushing of the tissues
Prolonged pressure – splint-sores, bed-sores
Extreme heat or cold—burns, frostbite, etc.
The action of strong chemicals—acids, alkalies, phenol, etc.
The action of powerful electric currents, or of lightning.

Lowered Vitality of the Tissues, either (*a*) *Local*, or (*b*) *General*.

Local: adjacent to the infected area in such acute infections as—

Septic wounds
Erysipelas
Anthrax
Gonorrhœa
Syphilis
Diphtheria
Scarlet fever
Cancrum oris.

General: occurring after some slight injury, as a complication or sequela of—

Diabetes
Enteric fever
Small-pox
Chicken-pox
Measles
Infantile marasmus
Cholera
Plague
Yellow fever
Malaria
Poisoning by snake-venom.

Disturbances of the Innervation of the Tissues, such as occur in—

Raynaud's disease
Erythromelalgia
Peripheral neuritis
Syringomyelia
Tabes dorsalis
Leprosy
Hemiplegia
Myelitis
Meningo-myelitis
Lesions of the spinal cord and cauda equina.

Stoppage of the Circulation, due to—

Embolism
Thrombosis
Endarteritis; senile gangrene
Occlusion of vessels, complete or partial, by—Ligature, tight bandages, splints; Pressure of new growths; Pressure of aneurysms or effused blood
The arterial spasm of ergotism, the so-called 'epidemic gangrene.'

Speaking generally, more than one of the causes enumerated above will be at work in the production of gangrene in any particular instance. Thus, in the gangrene following severe injury to one of the extremities, stoppage of the circulation through the affected part is usually observed in addition to the direct injury caused by the mechanical crushing of its tissues. Again, in *cancrum oris* or *noma*—the name given to the spreading gangrene of the soft tissues of the mouth and cheek occurring in debilitated children after measles or scarlet fever—great feebleness of the circulation contributes to its production, in addition to the lowered vitality of the necrotic tissues (*Fig.* 24, p. 74). A diabetic patient with gangrene may owe it partly to the impoverished or altered quality of his blood, partly to the arteriosclerosis that is often associated with diabetes, and partly to peripheral neuritis occurring as a further complication of his disease.

In *dry gangrene*, or *mummification*, the affected part of the body, usually the distal end of a limb, becomes livid and cold, and gradually blackens as the blood-pigment diffuses out of the blood-corpuscles and enters the tissues; the part withers as the fluid in it evaporates. It is a slow process; putrefaction is little in evidence, and there is no markedly offensive odour about the part, for it is too dry to afford a satisfactory culture-medium for the bacteria of putrefaction; between this dry gangrenous tissue and the adjoining healthy part of the limb is an inflammatory zone: the line of demarcation (*Plate XIV*, p. 258). Dry gangrene is common in cases of embolism or other complete obstruction of the arteries, in senile gangrene, and in Raynaud's disease (*Plate XIII*); the affected part is converted ultimately into a shrunken, black, and mouldy-smelling mass.

Moist gangrene, *sphacelus*, or *sloughing*, may often be seen after severe crushing of a leg or an arm, when the distal portion of the limb dies and putrefies. At first hot, red, and painful, the crushed extremity presently becomes mottled, purplish, and cold, as the circulation through it stops. Putrefaction soon appears in the dead tissue, the skin rising into discoloured blebs, which, on rupture, give issue to offensive sanious fluid. A dusky red line of demarcation separates the gangrenous from the adjoining healthy part. 'Sloughing' is the name commonly given to the putrefactive separation of smaller parts of the soft tissues from the body; sloughs are the localized gangrenous patches that result from most of the injuries described under the first heading.

Spreading gangrene is the form due to infection by special virulent bacteria such as *Bacillus aërogenes capsulatus*, which cause death of the tissues in which they grow and spread. Fatty acids, sulphides, and gases are among the chemical compounds formed by these micro-organisms, and it is to them that the offensive odour is due.

THE DIAGNOSIS.

Traumatic Local Causes and Lowered Vitality of the Tissues.—Gangrene being an infective necrosis of some part of the body, producing changes obvious to the eye and nose, the fact of its occurrence can rarely be difficult to determine. The history of exposure to one or another of the forms of severe injury or infection, or of exposure to some injury or infection that would be unimportant if it occurred in a healthy person, but may lead to gangrene in severely debilitated patients, ought to be elicited readily.

Disturbances of the Innervation of the Tissues.—Gangrene due to disturbances in the innervation of the tissues is commonly described as a trophoneurosis or trophic change. It may be either *chronic* or *acute* in its onset.

Gangrene of a Chronic Type.—In *Raynaud's disease* gangrene may affect the tips of the fingers or the toes, less often the edges of the ears and the end of the nose or tongue. It is often symmetrical, and is preceded by the other two well-known stages of the disease, namely, local syncope, in which the affected extremities become cold, numb, and white; and local asphyxia (*Fig.* 125), in which they turn from white to blue-grey or purple. Rarely, Raynaud's disease is characterized only by recurring attacks of necrosis in the extremities (*Fig.* 126). It is a chronic affection, and gangrene only occurs in marked cases and in their later stages, although it may be seen at any age. As a dry gangrene attacking the superficial and terminal parts of some of the digits, it may bear some resemblance to *senile gangrene* (*Plate XIV*, p. 258); this, however, generally attacks only one limb, usually a foot; it is more extensive and progressive than the gangrene of Raynaud's disease; and it is associated with well-marked disease of the arterial walls.

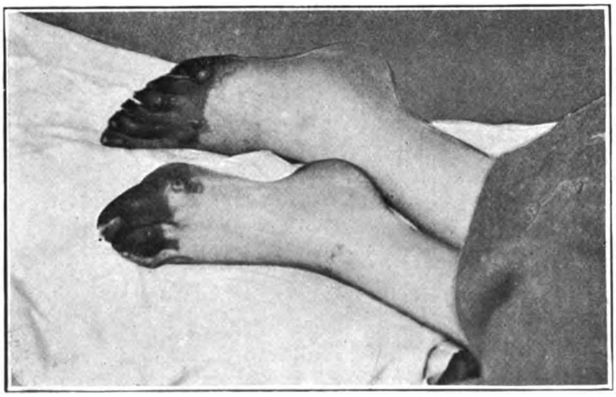


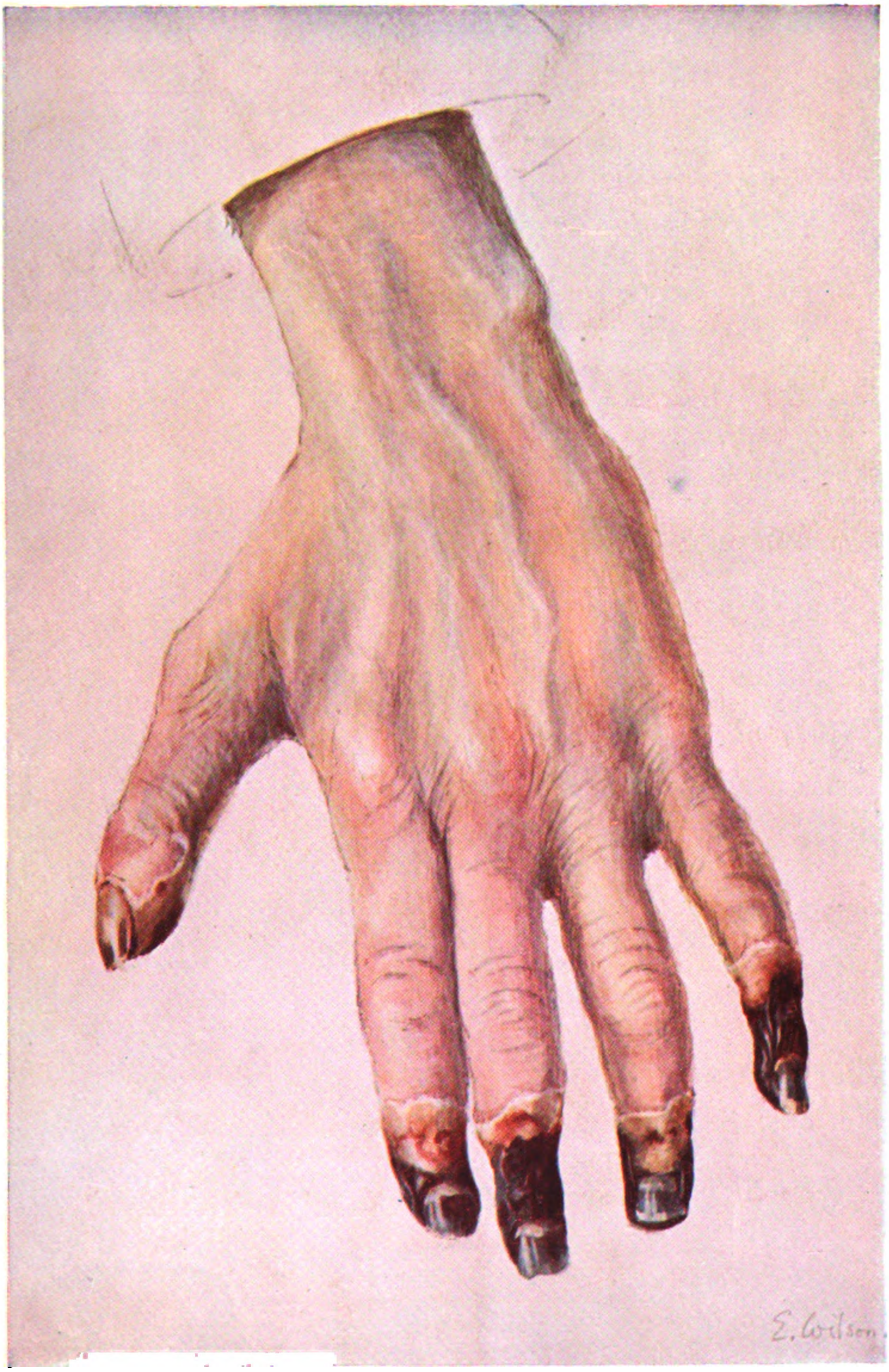
Fig. 125.—Raynaud's disease: stage of local asphyxia.

Gangrene may be a part of the manifestations of *erythromelalgia*, a rare and chronic disease of adults who do hard work while exposed to considerable changes of temperature. It is characterized by pain, heat, and flushing of one or more of the extremities, all aggravated when the limb is allowed to hang downwards. The colour varies from rosy red to purple, and the affected parts are hot: hence the condition should not be confused with Raynaud's disease. The gangrene of erythromelalgia is confined to the extremities and may be symmetrical; as a rule it is more narrowly localized and less superficial than the gangrene met with in Raynaud's disease.

Gangrene is a rare complication of *peripheral neuritis* due to alcoholic, arsenical. or other forms of poisoning (p. 65); it occurs only in patients exhibiting the vasomotor type of neuritis. This closely resembles Raynaud's disease, with which, indeed, some hold it to be identical. The gangrene is symmetrical; the patient will very probably exhibit other symptoms of peripheral neuritis—disturbances of sensation, tremor, paresis, wasting, trophic changes—and a history of alcoholic excess may be obtainable.

PLATE XIII

SYMMETRICAL GANGRENE OF THE FINGERS IN RAYNAUD'S DISEASE



Reproduced by permission of the House Committee of St. George's Hospital from a water-colour drawing by the late Dr. E. A. Wilson.

Gangrene of the skin and superficial tissues of the hands or feet, or of the finger-ends, may be met with in *syringomyelia* ; this disease, if associated with painless whitlows on the fingers, is known as *Morvan's disease*. This gangrene is to some extent traumatic, and may be symmetrical ; but the diagnosis should not be difficult, for in most cases three prominent symptoms are seen in syringomyelia : (*a*) Loss of the sensations of pain and of temperature, tactile sense being preserved over the anæsthetic area—the 'dissociated anæsthesia' of Charcot. (*b*) Trophic changes about the extremities, often originating in some neglected or unnoticed injury ; hypertrophy or atrophy of the skin or nails ; trophic changes in the joints, the so-called 'Charcot's joints' ; brittleness of the long bones, with a tendency to spontaneous fracture. (*c*) Progressive muscular atrophy, invading the hands first, later the forearms, arms, and shoulders ; atrophy of the spinal muscles may ensue, giving rise to spinal curvature. Thus the gangrene of syringomyelia is characterized by its painlessness, and by its combination with other well-marked special symptoms ; in addition the hands often present certain deformities, 'CLAW-HAND' (p. 109) resulting when the muscular atrophy of the hands is marked, 'succulent hand' when much hyperplasia and redundancy of the soft parts of the hand and fingers occur.

Gangrene of the toes may occur in *tabes dorsalis*, usually in connection with a perforating ulcer about the ball of the big toe (*Fig.* 297, p. 736). The process is slow and painless, not symmetrical ; and is associated with the other main signs of tabes (p. 609). Gangrene of a similar sort, and similarly started by some ulceration or a neglected injury, is common in *leprosy* of the smooth or anæsthetic type. It occurs only in the later stages of this disease, and from its rarity calls for no further consideration here.

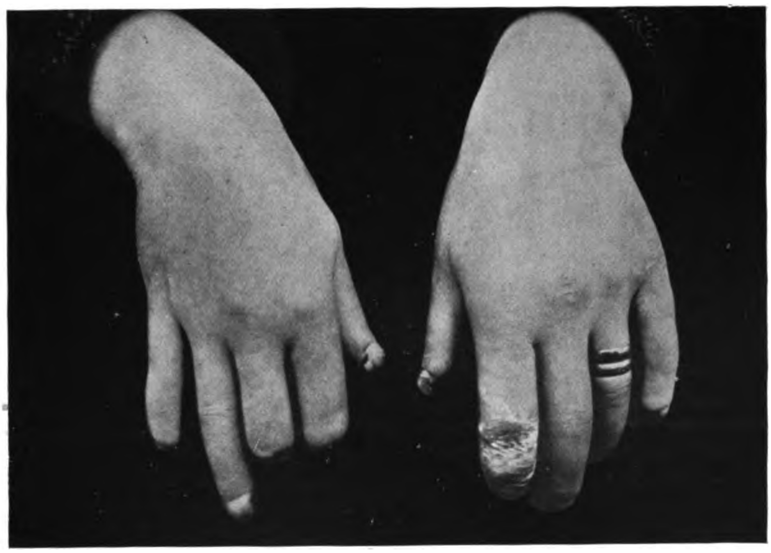


Fig. 126.—The effect of Raynaud's disease after it has produced recurrent necrosis of the fingers.

Gangrene of an acute type, attributable to trophic changes, occurs in the form of *decubitus acutus*, or *acute bedsore* in certain acute disorders or infections of the central nervous system or spinal cord, producing both paralysis and anæsthesia. Within a few days or even hours of the primary lesion, secondary changes are seen in the skin and soft tissues where they are most exposed to pressure—about the buttock, sacrum, coccyx, iliac crest, great trochanter, tibia, or heel, according to the position in which the paralyzed patient lies. When the pressure is unduly great or protracted the skin turns red or purple, and unless most carefully protected presently undergoes extensive and spreading necrosis and gangrene. Hot-water bottles that would expose an ordinary patient to no discomfort or danger, may set up analogous necrosis and gangrene if allowed to remain too long or too closely in contact with the skin of a paralytic patient liable to the formation of bedsores. The prolonged application of an ice-bag may do the same; indeed, the use of ice-bags over long periods may be followed by gangrene even in patients who are free from any nervous disorder, and particularly in patients who are very fat. The chief nervous lesions in which the acute bedsore is seen are the following: *hemiplegia*, whether due to cerebral embolism, hæmorrhage, or thrombosis; acute infections of the spinal membranes or cord, such as *meningitis*, *myelitis*, or *meningo-myelitis*, whatever the nature of the infection; *transverse lesions* of the spinal cord or cauda equina, such as are caused by fractures or fracture-dislocations of the spinal column, or by penetrating wounds involving the spinal cord. These bedsores occur only in the anæsthetic areas, and hence tend to escape the notice of the patient, who may also be unconscious or delirious. It is most important to keep a sharp look-out on the skin over all the bony prominences exposed to pressure in these patients, so that an incipient bedsore may be detected at once, and its spread checked by suitable treatment. Once well established, the acute bedsore tends to spread in area and in depth in spite of the most careful treatment, and brings about the death of the patient by septic absorption, pyæmia, or the exhaustion consequent to prolonged suppuration.

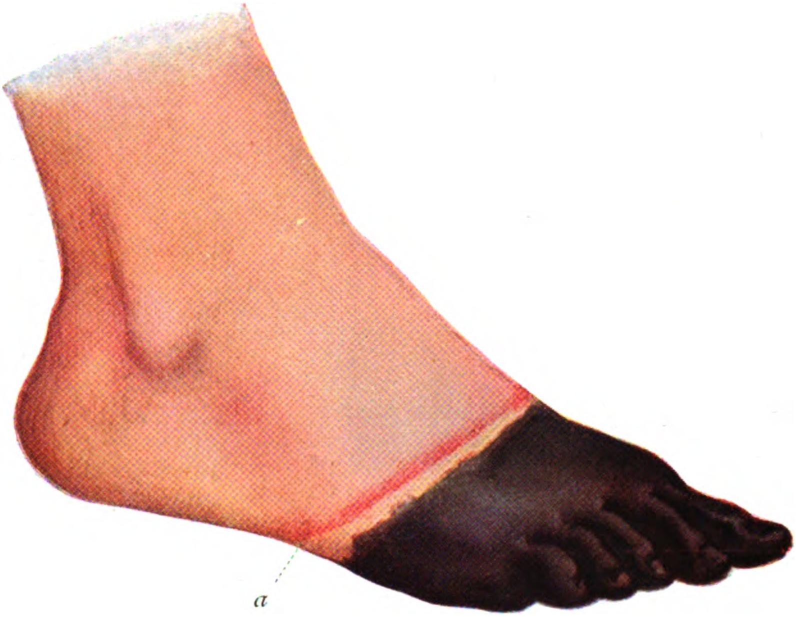
Stoppage of the Circulation.—Among the most important and extensive causes of gangrene are those in which the exciting factor is some more or less complete vascular obstruction, with consequent stoppage of the circulation, and the death of those tissues whose blood-supply is cut off. Occlusion of arteries is more important than that of the veins, but in exceptional cases moist gangrene of some distal part follows blocking of the veins by thrombosis or by pressure from without, while the arteries are still patent. The importance and amount of the pathological changes following vascular obstruction depend on the extent to which collateral channels are able to carry on the circulation through the affected area. If they are ill-developed, the consequences of the stoppage are serious. *Embolism* is likely to occur in patients who have valvular disease of the heart, with vegetations on the mitral or aortic valves that may be swept off into the blood-stream; or the embolus may be derived from a blood-clot formed in a diverticulum of one of the chambers of the left heart, or in an aneurysm, or upon the surface of a rough atheromatous aorta. *Thrombosis*, whether arterial or venous, may be suspected in patients in whom no source for an embolus can be detected, but who exhibit widespread arterial degeneration, phlebosclerosis, or local disease that may spread to some vessel and set up clotting in its contents. The occurrence of arterial embolism, in the leg for example, is marked by a sudden and very severe pain in the limb about the level of the blockage. The parts beyond become numb, cold, insensitive; pulsation can no longer be felt in the arteries distal to the obstruction. The gangrene that follows is usually of the dry type. Very similar symptoms may mark the occlusion of an artery in the leg by thrombosis, but the onset is usually much more gradual, and the pain may be terribly protracted and severe.

Senile gangrene occurs in patients of advanced years with extensive arterial sclerosis; in many instances they also give a history of gout, or suffer from diabetes mellitus. It is in reality a form of occlusive gangrene, due either to the clotting of blood on the diseased and roughened arterial intima, or to increasing obstruction of the arterial lumina by a proliferative endarteritis. It is often of insidious onset and confined to one lower limb, just as embolic gangrene may be; but it tends to spread upwards slowly and indefinitely, a tendency that finds a natural explanation in the extensive character of the arterial degeneration that goes with it. It is not often symmetrical; if more than one limb is affected the lesions are successive in their development.

Little need be said about the gangrene that follows complete or partial occlusion of the vessels by the other causes enumerated above. The gangrene will be secondary to some primary lesion that will seldom fail to be obvious. The *ligature of an artery* in the course of a surgical operation—of the femoral, for example, in the treatment of popliteal aneurysm—has caused gangrene of the leg in patients whose collateral circulation unfortunately proved to be inadequate. The application of *tight bandages* round a limb, possibly to check hæmorrhage, may cause similar gangrene if they are left on too long. *New growth* readily compresses or invades veins or even arteries in exceptional cases, and renders them impervious; in either case gangrene of some distal part may result. The new growth may be primary, or a secondary deposit growing perhaps in a lymphatic gland. Thus carcinoma in the mammary gland, or endothelioma of the lung or pleura, may lead to

PLATE XIV

GANGRENE OF FOOT



Note the line of demarcation at (*a*).

From 'Introduction to Surgery,'
by kind permission of Prof. Rutherford Morison

secondary deposits about the axillary and subclavian vein and artery; and these may be so extensive as to obstruct the circulation through the arm, and set up moist gangrene in the fingers. Similar gangrene of the fingers may result from the vascular obstruction caused by a large intrathoracic *aneurysm*, or by *blood* that has escaped and clotted round the vessels of the arm.

The epidemic gangrene of ergotism is only of historic interest in Great Britain, although it is said to occur still in Russia. It is seen only in persons who consume quantities of mouldy rye; it appears not to occur in human beings as the result of excessive doses of the pharmaceutical preparations of ergot. Minor degrees of ergotism may, however, simulate Raynaud's disease or erythromelalgia. Gangrene due to ergot is dry, chronic in progress, extremely painful, and usually asymmetrical; it results in much disfigurement from loss of tissue, and has had a high mortality in many of its epidemics.

A. J. Jex-Blake.

GANGRENE OF THE LUNG occurs when a portion of this organ undergoes necrosis and then, owing to invasion by one or more of many kinds of bacteria, putrefies. It usually occurs in senile, intemperate, diabetic, or debilitated patients. Most often it affects a circumscribed area of lung tissue only, but it may be a diffuse process involving a whole lung. It occurs as a rare complication of *pneumonia* or *bronchopneumonia*; and as a comparatively common complication of *aspiration pneumonia* due to direct infection of the lung by bacteria contained in food, mucous secretions, or foreign bodies generally (peas, beans, fish-bones, extracted teeth, etc.). that have made their way past the larynx and into the trachea or bronchi. Gangrene may also result from an extension of the infection in such chronic suppurative affections of the lungs as *chronic pulmonary tuberculosis*, *bronchiectasis*, or *fœtid bronchitis*. In other instances the infecting agent reaches the lungs by the blood-stream; thus gangrene may follow *pulmonary embolism* if the emboli contain septic or putrefactive bacteria, secondary, for instance, to lateral sinus thrombosis the result of middle-ear disease; or it may result from *penetrating wounds* of the lung, or from the *spread of infection* from the pleura, peritoneum, or pericardium to the tissue of the lungs.

Gangrene of the lung is characterized by great prostration, irregular fever, cough, and in most cases the expectoration of copious, fluid, frothy sputum of disgusting odour. The sputum settles into three layers on standing, and the lowermost of these contains fragments of elastic tissue. Severe hæmoptysis from gangrenous erosion of a blood-vessel may be noted in chronic cases. In a few cases the sputum lacks the indescribable but characteristic fœtor, oftenest so in diabetics or children. The physical signs of gangrene of the lungs are in no way distinctive; more or less extensive consolidation or infiltration of the affected part will be indicated early in the disease, and later, when the gangrenous tissue has softened and been expectorated, the signs of a cavity may appear. Occurring as a terminal event, shortly before the death of an exhausted and debilitated patient, pulmonary gangrene may not be suspected, and so may escape detection.

As a rule, however, the diagnosis presents no great difficulty, being suggested by the supervention of copious and highly offensive expectoration in a patient known to be suffering from one or another of the diseases already mentioned. The gangrene may, however, be simultaneous with the development of an *aspiration pneumonia*, and this condition may therefore be considered more fully. It is often set up by the entry of a foreign body into the trachea or a bronchus; it may follow stenosis of a bronchus from any cause, such as syphilis, or the pressure of an aneurysm or of a new growth; it may result from the establishment of a fistula from the œsophagus to the trachea or a bronchus as a terminal event in malignant disease of the air-passages or œsophagus; it is seen in patients with spreading infections of the mouth, pharynx, or larynx; it occurs in the insane, or in persons with extensive laryngeal or bulbar paralysis who are constantly exposed to the danger of swallowing food directly into their air-passages; and it is observed occasionally ofter operations, particularly those on the mouth, pharynx, larynx, or trachea, when infective matter—e.g., mucus, sputum, a fragment of a tooth that has just been extracted—has made its way into the bronchi while the patient was under the influence of a general anæsthetic. All the causes just enumerated are dependent on exceptional circumstances or conditions that should be distinguished fairly easily or remembered by the patient; but in not a few instances gangrene of the lung has followed bathing, diving, or accidental immersion, though the patient could not call to mind that he inhaled any water; in the same way the origin of other cases of pulmonary gangrene has remained obscure until the patient has coughed up a piece of bone, a fragment of a tooth, part of an ear of corn, or some other foreign body that he had no recollection of having inhaled down his trachea.

In patients with *pyæmia*, gangrene of the lung due to multiple embolic pulmonary abscesses would be suggested if the patient should develop the signs of pulmonary consolidation, cough, and offensive expectoration. Similar symptoms occurring after *wounds* or *contusions of the lungs* would make the same diagnosis highly probable.

Greater difficulty is experienced in deciding whether gangrene of the lung has occurred in a patient suffering from *bronchiectasis*, *fœtid bronchitis*, *chronic pulmonary tuberculosis with cavity formation*, or *putrid empyema discharging through the lung*, where expectoration of highly offensive sputum was already present. Elastic fibres and shreds of pulmonary tissue may be, and often are, present in the sputa of all these conditions; but they are commonest, and present in greatest amount, in pulmonary gangrene. Again, the onset of pulmonary gangrene is often acute, and accompanied by much prostration, no doubt due to septic absorption; these facts, coupled with evidence of appropriate changes in the physical signs of the patient's lungs, should assist in arriving at the diagnosis.

Pulmonary gangrene may occur so soon before death as to be unsuspected; in a few instances the sputum is not fœtid; in others, particularly in children, the gangrene may lead to no expectoration at all. In these circumstances the diagnosis is impossible, and the gangrene of the lung may be described as *latent*. *A. J. Jex-Blake.*

GASTRECTASIS.—(See DILATATION OF THE STOMACH, p. 173.)

GIDDINESS.—(See VERTIGO, p. 751.)

GIRDLE PAIN, or 'girdle sensation'—which is often a better description of the phenomenon—is a sense of constriction, sometimes of painful constriction, as though a tight band encircled the trunk. The band may be narrow or broad, and may be referred to any level of the thorax or abdomen. Although a common symptom of *tabes*, it is not pathognomonic of that disease, and may occur with any morbid condition involving symmetrically the posterior spinal roots, such as *syphilitic spinal meningitis*.

Another form of girdle sensation, having a different pathological basis, is often described by patients suffering from *spastic paraplegia* due to focal disease within or outside the dorsal region of the spinal cord. In such a case the tight feeling is found to correspond with the highest level of spasticity, sometimes with the highest level of sensory loss. Thus a girdle sensation may be a symptom of *disseminated sclerosis*, of *myelitis*, or of *compression paraplegia*. In the last it may help the physician to localize the level of the disease, but it is rarely so reliable for this purpose as the information which can be obtained from a careful investigation of the distribution of motor and sensory paralysis and of the superficial reflexes (see PARAPLEGIA, p. 510). *E. Farquhar Buzzard.*

GLANDS, LYMPHATIC, ENLARGEMENT OF.—(See LYMPHATIC GLAND ENLARGEMENT, p. 376.)

GLYCOSURIA.—The diagnosis of glycosuria falls naturally into two divisions:—(I) *The recognition of glucose in the urine*; and (II) *The inference as to the disorder with which it is associated.*

I. THE RECOGNITION OF GLUCOSE IN THE URINE.

Certain conditions are met with so commonly in connection with glycosuria that their presence makes it imperatively necessary to test the urine for sugar. Thus irritation (eczema, intertrigo) in the neighbourhood of the glans penis or vulva may be caused by the direct local action of the sugar contained in the urine, while boils and carbuncles arise in the skin owing to diminished resistance to the attack of micro-organisms. Peripheral neuritis, perforating ulcers, and gangrene of the extremities may own a similar cause. Obese persons are often the subjects of a chronic form of glycosuria. Early symptoms of diabetes are: unexplained weakness and lassitude, increased hunger or thirst, increased frequency of micturition owing to the polyuria. Dimness of vision may be the condition of which complaint is made, and may be due either to commencing cataract or to a form of retinitis closely resembling that met with in cases of chronic nephritis.

Urine containing sugar is usually large in quantity, pale in colour, and peculiarly bright and clear in appearance. Its specific gravity is high, any reading over 1030, except in a dark concentrated specimen, suggesting the need for careful examination for sugar. It is acid in reaction, and seldom gives rise to any deposit on standing, but occasionally it may become contaminated with torulæ which form a turbidity, or even a white sediment. In all cases in which sugar is found in the urine it is important to ascertain whether acetone and diacetic acid are also present, such abnormal constituents pointing to a condition of considerable gravity, a true diabetes rather than a simple glycosuria.

The following are the more important chemical tests for the presence of dextrose in the urine :—

1. **Trommer's Test.**—This—and the two succeeding tests, which are modifications of it—depend on the power possessed by glucose of reducing alkaline solutions of salts of copper, with formation of red oxide of copper. To perform Trommer's test, a small amount of solution of potassium hydrate is poured into a test-tube (say ½-in. depth), and to it are added, first, a few drops of solution of copper sulphate, which will produce a precipitate of copper hydrate; and secondly, a small quantity of the suspected urine. On boiling the mixture, a red precipitate or suspension of cuprous oxide appears if glucose be present; while if glucose is absent, black cupric oxide is formed instead.

2. **Fehling's Test.**—This is Trommer's test modified by the addition of sodio-potassic tartrate, which holds the black oxide of copper in solution. Two solutions are prepared as follows: (i) Dissolve 36·64 grams of copper sulphate crystals in distilled water and make up to 500 c.c.; (ii) Dissolve 125 grams potassic hydrate and 173 grams sodio-potassic tartrate (Rochelle salt) in distilled water and make up to 500 c.c. These two fluids should be kept in separate stoppered bottles. For use, take equal quantities of each (say ½-in. deep in a test-tube), mix, and boil. Add to the hot fluid a few drops of boiling urine. If glucose is present a red suspension of cuprous oxide is formed; if it is absent, the fluid retains its blue colour.

3. **Pavy's Test.**—This is practically the same as Fehling's, with the exception that a certain amount of ammonia is added to retain the red oxide of copper in solution. The solution is made thus: Copper sulphate, 4·16 grams; sodio-potassic tartrate, 20·4 grams; strong solution of ammonia, 300 c.c.; and distilled water to 1 litre. On boiling this with urine containing glucose it is decolorized. The blue colour returns on contact with the air. This test is seldom used except for quantitative purposes (see below).

Sources of Error in the Above Tests.—Error may be caused by the presence in the urine of other bodies besides glucose which have the power of reducing copper salts. The most important of these are *Lactose* and *Pentose*. Both of these sugars form 'osazone' crystals with phenyl-hydrazine, but they do not ferment with yeast. Pentoses give a cherry-red colour when heated with hydrochloric acid and a little phloroglucin. They also react with the following solutions (**Bial's Test**): Orcin, 1 gram; 10 per cent solution of ferric chloride, 25 drops; strong hydrochloric acid, 500 c.c. On heating 5 c.c. of the urine with 10 c.c. of this solution a greenish-blue colour is produced, and finally a precipitate of this colour is formed.

Other substances which may cause error in testing with Fehling's solution are—*Glycuronic Acid*, *Uric* and *Hippuric Acids*, *Xanthin*, *Creatinin*, and *Alkapton*. As a rule, however, they do not produce more than a dull greenish-yellow precipitate, instead of the golden colour given with glucose. They are none of them fermented by yeast. Glycuronic acid gives the reactions described as characteristic of pentose. Alkaptonuria is suggested by the dark colour of the urine (see URINE, ABNORMAL COLORATION OF, p. 746). The reduction sometimes seen on testing the urine of patients who have been taking certain drugs, such as morphine, chloroform, chloral, salol, camphor, phenazone, benzoic acid, or carbolic acid, is probably due to glycuronic acid.

If the urine to be tested for glucose by the copper-reduction method contains any large amount of albumin, this should be removed first by boiling and filtration. If the urine be ammoniacal, Fehling's test may be converted unwittingly into Pavy's, and decolorization be produced instead of a red precipitate. Strongly alkaline urine should be rendered slightly acid with acetic acid.

4. **Böttger's Test.**—Put a small quantity of urine (freed if necessary from albumin) into a test-tube, and add an equal quantity of liquor potassæ and a couple of grains of bismuth subnitrate (as much as will lie on the point of a small penknife). On boiling, a black precipitate is formed.

5. **Nylander's Test.**—Make up the following solution: Bismuth subnitrate, 2 grams; sodio-potassic tartrate, 4 grams; caustic soda solution (sp. gr. 1·12), to 100 c.c. On boiling 5 c.c. urine containing glucose with 5 or 10 drops of this solution a black precipitate is formed. Glycuronic acid, pentose, and lactose also reduce bismuth.

6. **Moore's Test.**—Put a small quantity of urine into a test-tube, add an equal amount of liquor potassæ, and boil. If sugar is present, a dark brown colour is produced and gradually deepens to an almost black tint. This test is not of much value, as it requires the presence of a considerable percentage of sugar, and a dark colour may be produced by other substances, such as indican and alkapton.

7. **Picric Acid Test.**—Pour about 5 c.c. of urine into a test-tube; add 2 c.c. of saturated solution of picric acid in water and a few drops of liquor potassæ, and boil. A dark brown colour is produced if glucose is present. This test also is of little value, as a dark colour is produced also by lactose, and even by creatinin; while impure picric acid alone may darken on boiling.

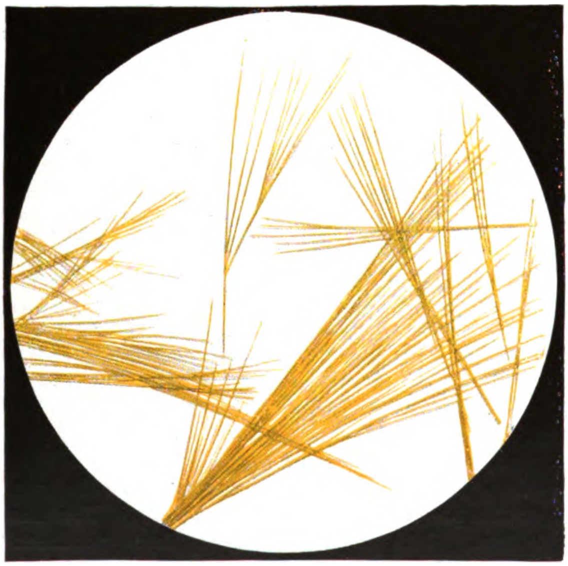


Fig. 127.—Phenyl-glucosazone crystals as seen under the medium power of the microscope. Note their yellow colour, finer rays, and larger size than the sheaves of tyrosin crystals (*Fig.* 148, p. 333).

8. **Phenyl-hydrazine Test.**—Fill a test-tube about a quarter full of urine, and add as much phenyl-hydrazine as will lie on the point of the large blade of a penknife, and a rather larger amount of sodic acetate. Boil some water in a beaker, place the test-tube in this and keep it boiling for half an hour. Then remove it, and allow it to cool. If glucose be present, crystals of phenyl-glucosazone will form in the shape of sheaves of bright yellow needles, as seen under a low power of the microscope (*Fig.* 127). Other sugars, such as lactose and pentose, as well as glycuronic acid, form crystals with this test; but these differ somewhat in shape, phenyl-lactosazone, for example, being shorter and rather in the form of bundles than of sheaves. The melting-points of the different compounds also differ: phenyl-glucosazone crystals melting at 206° F., phenyl-lactosazone at 200° F, phenyl-pentosazone at 160° F. In all cases of doubt the crystals should not only be inspected under the microscope; their exact melting-point should be determined also. Glycuronic acid usually gives rise to an amorphous precipitate, or scales, not to crystals. The urine to be tested should be free from albumin.

9. **Safranin Test.**—Place in a test-tube about a ½-inch of urine (free from albumin); add equal quantities of liquor potassæ and of solution of safranin (1–1000). A dark red fluid is produced, which turns yellow or brownish on boiling if glucose is present. This test is seldom used, but appears to be a sure indication of the presence of glucose (Bedford).

10. **Fermentation Test.**—Boil some urine (200 c.c.) in a beaker, and allow it to cool; stir into it a piece of yeast the size of a small cherry till it is thoroughly mixed. Alkaline urine should first be rendered acid with a few drops of acetic acid. Fill the graduated limb of a ureometer with the fluid, and let it stand for twenty-four hours in a warm place. If glucose is present carbon dioxide gas will be formed and will accumulate at the top of the tube. If performed carefully this test is a certain indication of the presence

of glucose. A small amount of fermentation may be produced by bacterial action on other sugars, but by boiling the urine this error is eliminated. Lævulose may also ferment with yeast, but its presence in urine is so exceptional that it may practically be disregarded. A rough indication of the amount of sugar present may be gained by taking the specific gravity of the urine after fermentation, and comparing it with that of a specimen kept under similar conditions but without yeast. It is said that a fall of one point in the specific gravity takes place for every grain of sugar per ounce of urine ; but this mode of quantitation is very inaccurate.

QUANTITATIVE MEASUREMENT of the glucose present may be made by either Fehling's or Pavy's fluid.

If **Fehling's Fluid** is used, 10 c.c. of the mixed fluid (i. and ii.) are placed in a porcelain dish along with about 40 c.c. of distilled water, and heated to boiling over a flame. A burette is filled up to a known mark with the urine, diluted to 1 in 10 (10 c.c. in 90 c.c. of distilled water), and this is allowed to run slowly, a few drops at a time, into the boiling fluid, which is stirred meanwhile with a glass rod. A precipitate of red oxide of copper forms, and the blue colour is gradually discharged. When this has completely disappeared, the quantity of diluted urine is read off ; and the amount of sugar in this is known to be 0·05 gram. Suppose that, for example, 8 c.c. of urine diluted to 1 in 10 have been used ; then 8 c.c. of undiluted urine will contain 10 × 0·05 gram glucose, or 0·5 gram. Knowing this, the percentage of sugar is easily calculated to be 0·5 × 100 ÷ 8, or 6·25 per cent.

The method of using **Pavy's Solution** is similar, with the exception that it must be boiled in a closed flask, and the nozzle of the burette connected to this by a piece of tubing which passes through the cork of the flask. The complete reduction of the copper is known by the decolorization of the fluid. Ten c.c. of Pavy's solution are equivalent to 0·005 gram sugar (it is thus only one-tenth the strength of Fehling's).

Bang's Method consists in boiling a portion of urine with a standardized solution of cupric sulphate and titrating the amount of copper remaining unreduced with a solution of hydroxylamine sulphate. The reagents are as follows : (i) Dissolve 500 grams potassic carbonate, 400 grams potassic sulphocyanate, 100 grams potassic bicarbonate, by heating in 1200 c.c. water ; add 25 grams cupric sulphate crystals dissolved in 150 c.c. water, and make up the whole to 2 litres. (ii) Dissolve 6·55 grams hydroxylamine sulphate and 200 grams potassic sulphocyanate in water and make up to 2 litres. Thus 1 c.c. of (ii) decolorizes 1 c.c. of (i). To perform the quantitative test, take 10 c.c. of urine (or of urine diluted to 1–5 or 1–10 if there is much sugar present) and boil with 50 c.c. of solution (i) for three minutes. Cool the mixture under the tap : it must remain blue, otherwise the operation must be repeated with 10 c.c. of more dilute urine. Titrate with solution (ii) in the cold till the colour disappears. The quantity of sugar present in the amount of (undiluted) urine originally used is calculated from the following table :—

C.c-hydroxylamine solution needed	Mgr. sugar in urine used	C.c. hydroxylamine solution needed	Mgr. sugar in urine used	C.c. hydroxylamine solution needed	Mgr. sugar in urine used	C.c. hydroxylamine solution needed	Mgr. sugar in urine used
44 ..	4·9	33 ..	15·4	22 ..	27·7	11 ..	41·8
43 ..	5·8	32 ..	16·5	21 ..	29·0	10 ..	43·3
42 ..	6·7	31 ..	17·5	20 ..	30·2	9 ..	44·7
41 ..	7·6	30 ..	18·6	19 ..	31·4	8 ..	46·3
40 ..	8·5	29 ..	19·6	18 ..	32·6	7 ..	48·0
39 ..	9·4	28 ..	20·7	17 ..	33·9	6 ..	49·8
38 ..	10·4	27 ..	21·8	16 ..	35·1	5 ..	51·6
37 ..	11·4	26 ..	23·0	15 ..	36·4	4 ..	53·4
36 ..	12·4	25 ..	24·1	14 ..	37·7	3 ..	55·0
35 ..	13·4	24 ..	25·2	13 ..	39·0	2 ..	57·3
34 ..	14·4	23 ..	26·5	12 ..	40·4	1 ..	59·4

The *Polarimeter* may also be used for quantitative estimation of glucose ; but as the instrument is not likely to be available in ordinary medical practice, it will not be described here.

II. DIAGNOSTIC IMPORTANCE OF GLUCOSE IN THE URINE.

In the great majority of instances, if glycosuria persists for any length of time—e.g., if sugar is found in the urine at frequent examinations during six weeks or two months—

the patient is suffering from some form of diabetes mellitus. Two main varieties may be distinguished :—

1. **True or Acute Diabetes.**—This occurs usually in youngish subjects; the urine is increased largely in amount, and the condition is accompanied by muscular weakness, wasting, marked thirst, and increased appetite. The face may be flushed, and the tongue often looks large and deep-red in colour. The amount of sugar in the urine is influenced but slightly by diet. As the disease advances, acetone, diacetic acid, and oxybutyric acid make their appearance in the urine. (See ACETONURIA, p. 3.) The presence of acetone in the breath may be recognized by the peculiar sweet smell. The knee-jerks are often lost. Death occurs, usually within two or three years, in a condition of coma. It may be hastened by the supervention of acute pneumonia or of a rapidly progressive tuberculosis of the lungs.

2. **Chronic Glycosuria.**—This occurs in elderly subjects, who are often obese, and may show gouty tendencies. The urine is not markedly increased in amount, and does not contain acetone bodies. The amount of sugar present is reduced considerably by strict dieting. There is no wasting, and little alteration of thirst or appetite.

Forms of intermediate severity are met with, the rapidity of the progress of the disease diminishing somewhat as age advances.

There are a few conditions associated with temporary glycosuria which have to be distinguished from true diabetes. The following are the most important :—

1. **Cerebral Injuries, Hæmorrhage, and Tumours** may be associated with glycosuria. In the case of cerebral tumours it may persist till death, and is by some writers described as diabetes due to this affection. It will usually, however, be associated with the cardinal signs of cerebral tumour—headache, vomiting, and optic neuritis. None of these are common in diabetes, though optic neuritis may occur. If a patient is seen for the first time during the coma which is caused by a cerebral hæmorrhage or injury, the presence of glycosuria may lead to a mistake in diagnosis. It must be borne in mind that in diabetic coma there are usually acetone bodies present in the urine; also that cerebral hæmorrhage is most often seen in elderly subjects, diabetic coma in younger persons, and that the amount of sugar found in cases of cerebral disease is not as a rule large.

2. **In Alcoholic Subjects** considerable quantities of sugar may occur in the urine and persist for some weeks, and may yet disappear entirely on careful dieting and complete abstinence from alcoholic liquors. This condition may be due to disturbance of the pancreatic or hepatic functions by the poison, and may really be an early stage of true diabetes which is amenable to treatment. It is well, therefore, not to make a diagnosis of incurable diabetes in an alcoholic subject until the effects of careful régime have been noted.

3. **Pancreatic Disease,** acute and chronic, may be accompanied by glycosuria. Pancreatic hæmorrhage and acute pancreatitis are signalized by severe pain in the upper part of the abdomen, constipation, vomiting, and collapse—symptoms suggestive of acute intestinal obstruction. In only a minority of these cases does sugar appear in the urine; when it does, it is an important aid in diagnosis of the affection present. In chronic pancreatitis—as also in some cases of gall-stones, in which this condition is probably present—glycosuria is encountered; indeed, the condition may go on to true and fatal diabetes. Wasting, pigmentation of the skin, repeated rigors, and the passage of undigested meat-fibres and of an increased quantity of fat (especially neutral or unsplit fats) in the fæces, accompany this form of pancreatic disease. (See under CAMMIDGE'S REACTION, p. 100.) Jaundice is often a marked symptom.

4. Other conditions in which small quantities of sugar may be found in the urine are **Graves's Disease, Starvation,** and so-called **Alimentary Glycosuria,** in which glucose is excreted after meals containing large quantities of this substance or, more rarely, of starchy food. These conditions should not lead to difficulties of diagnosis.

It is well to bear in mind that the onset of true diabetes may be signalized by the *transitory* appearance of glucose in the urine. This symptom may disappear once or twice, with or without special treatment, but may finally return and persist. This occurrence may be observed sometimes in connection with pregnancy, sugar being found in the urine, or increased thirst and appetite noted along with polyuria, the patient subsequently regaining her health; then, at a subsequent pregnancy, the symptoms may recur and persist.

W. Cecil Bosanquet.

GOITRE.—(See Thyroid Gland Enlargement, p. 721.)

GRINDING OF THE TEETH DURING SLEEP is a symptom which troubles the patient little, but may considerably disturb those who sleep with him. In itself it is, however, a symptom of little importance. It is popularly held that grinding of the teeth at night, especially in children, is an indication of the presence of intestinal worms, particularly of the *Oxyuris vermicularis* ; it would be well, therefore, to have the fæces examined in all cases of the kind, both for parasites and for their ova. The popular belief of the association of intestinal parasites with the teeth-grinding habit is seldom verified clinically, however, and the habit may be very bad and persistent in children, or even adults, who are in perfect health. Very often it is rather a rattling of the upper teeth against the lower, owing to lateral movements made by the lower jaw as the patient, when half roused, turns over in bed ; actual gritting of the teeth during sleep is far less common. It is possible that in its beginning there was a gumboil or other local irritation, which led to jaw-movements that persisted as habitual grinding of the teeth long after the primary cause was gone. *Herbert French.*

GUMS, BLEEDING.—(See Bleeding Gums, p. 72.)

GUMS, RETRACTION OF.—(See Retraction of the Gums, p. 589.)

GUMS, SPONGY.—(See Bleeding Gums, p. 72.)

HÆMATEMESIS is a term indicating vomiting of blood. It has to be differentiated from hæmoptysis, but the distinction is not difficult if attention is paid to the points discussed on p. 285.

Having arrived at the conclusion that the patient is suffering from hæmatemesis, the next point is to determine the cause.

CAUSES OF HÆMATEMESIS.

A. **Swallowed Blood.**

- Epistaxis
- Hæmoptysis
- Bleeding from the mouth and throat
- Malingering

B. **Diseases of the Œsophagus** :—

- Epithelioma
- Aortic aneurysm rupturing into the œsophagus
- Rupture of varicose œsophageal veins
- Mediastinal growth perforating the œsophagus and aorta
- Foreign body perforating the œsophagus and aorta

C. **Diseases of the Stomach** :—

- Acute gastritis
- Chronic gastritis
- Toxic gastritis
- Corrosive poisons, such as strong acids or alkalies
- Gastrointestinal irritants, such as arsenic, phosphorus, antimony
- Ulcer
- Gastrostaxis
- Hæmorrhagic erosions
- Carcinoma
- Injuries
- Atheroma
- Abdominal aneurysm opening into the stomach

D. **Diseases of the Duodenum** :—

- Ulcer
- Carcinoma
- Gall-stone ulcerating into the duodenum

E. **Portal Obstruction** :—

- Cirrhosis of the liver
- Pylephlebitis (adhesive) }
- Pressure on the portal veins } quite rarely
- Chronic heart and lung disease }
- Some cases of enlarged spleen }

F. **Acute Febrile Diseases** :—

- Malignant variola
- Malignant scarlet fever
- Malaria
- Yellow fever
- Dengue
- Cholera
- Acute yellow atrophy
- Fungating endocarditis

G. **Blood Diseases**:—

Purpura
Scurvy
Hæmophilia
Leukæmia
Hodgkin's disease
Chlorosis (?)
Pernicious anæmia
Malarial cachexia
Splenic anæmia

H. **Miscellaneous**:—

Chronic Bright's disease
Following abdominal operation
Prolonged jaundice
Syphilis

It may be said at once that there are only three *common* causes of *profuse* hæmatemesis—namely, *gastrostaxis*, *gastric ulcer*, and *cirrhosis of the liver*. The differential diagnosis between these is by no means always easy. The older the patient and the greater the history of alcoholism, the more likely is the symptom to be due to cirrhosis of the liver; at this stage of the malady there may be neither jaundice nor ascites, but the liver may be felt enlarged and unduly firm, and the spleen may also be palpable. Hæmatemesis in a young, anæmic woman is more likely to be due to gastrostaxis than to ulcer, whereas in older patients, especially in males, gastric or duodenal ulcer is the probable diagnosis if cirrhosis can be excluded. The distinction between gastrostaxis and gastric ulcer often becomes one of opinion only, unless operation is resorted to. The longer the preceding history of gastric symptoms, and the more definitely localized the epigastric pains, the more likely does ulcer become.

The diagnosis is often arrived at quickly enough, but sometimes a routine discussion of all the possible causes is required: so that we will take each of the above groups in turn.

A. Swallowed Blood.

Epistaxis.—If there is obvious bleeding from the nose as well as hæmatemesis, the probability would be that some of the blood had trickled down the posterior nares into the pharynx, and had been swallowed and subsequently vomited. It should be remembered, however, that the two commonest causes of epistaxis in adults are cirrhosis of the liver and chronic interstitial nephritis, so that the possibility of bleeding having occurred from the stomach as well as from the nose would have to be considered. If a moderate degree of epistaxis has taken place during the night, blood may have been swallowed unconsciously. In some cases in which no blood has come from the anterior nares, examination may reveal blood trickling from the posterior nares, and the epistaxis may become evident if the patient blows his nose.

Hæmoptysis.—When blood comes from the air-passages, some of it may flow back into the pharynx and be swallowed, especially if the hæmorrhage occurs during sleep. If the patient has a cough, or expectorates blood-stained sputum and presents signs of chronic pulmonary disease, the possibility of swallowed blood must be considered as a cause of the hæmatemesis, though difficulties may arise in forming a correct conclusion, for cirrhosis of the liver, for instance, is not infrequently complicated by phthisis, and so on.

Bleeding from the Mouth and Throat.—The gums, tongue, and fauces should be examined carefully, as blood from any of these sources may be swallowed and later vomited. Bleeding from the gums is most likely to occur when they are spongy, as in scurvy or mercurial stomatitis.

Malingering.—The possibility of blood having been drunk in secret and afterwards vomited with intent to deceive must be considered in some cases when no cause can be found to account for its occurrence. Should fraud be suspected, it may generally be detected by careful observation. The red corpuscles should be examined microscopically in case the oval corpuscles of a bird may reveal their extraneous source.

B. Diseases of the Œsophagus.

Epithelioma.—Hæmorrhage is rare in the commonest form of epithelioma of the œsophagus which leads to an annular stricture, but it may occur from erosion of small blood-vessels as the result of the ulcerative form of the disease, the amount of blood which is brought up being small. When the ulceration is deeper and more extensive it may finally lead to perforation of a larger vessel, even the aorta, a condition which causes sudden, profuse, and rapidly fatal hæmorrhage. The diagnosis of this cause does not, as a rule, give rise to much difficulty; dysphagia is the earliest symptom in nearly all the cases (see p. 194).

An Aneurysm of the Thoracic Aorta compressing the œsophagus may finally erode and open into it, with profuse and fatal hæmatemesis.

Rupture of Varicose Œsophageal Veins.—Varicose veins occur in the lower end of the œsophagus as a result of portal obstruction, especially that form which is due to cirrhosis of the liver, and the rupture of such veins is often followed by profuse hæmatemesis. It is, however, practically impossible to determine whether the blood comes from the lower end of the œsophagus or from the stomach, so that the diagnosis resolves itself into one of cirrhosis of the liver.

Mediastinal Growth perforating the Œsophagus and Aorta.—Hæmatemesis from this cause is exceptionally rare, for mediastinal growth is by no means a common disease, and vomiting of blood is an infrequent complication of it. Hæmorrhage may occur, however, if the growth compresses and erodes the œsophagus. It is most likely to be mistaken for thoracic aneurysm or epithelioma of the œsophagus. The tendency of new growth to compress and invade the large veins, leading to œdema of the neck and upper extremities, cyanosis, and dilated superficial veins, is characteristic, and serves to distinguish it from aneurysm, in which severe venous obstruction is much rarer. The following case of mediastinal sarcoma perforating the œsophagus and aorta is an example of hæmatemesis from this cause :—

Michael H., aged 30, was admitted for dyspnœa and pain in the chest. He had suffered from several attacks of what had been considered to be hæmoptysis similar to that of phthisis, the first occurring seven months before his admission. He was found to have impairment of note, with diminished breath-sounds, over the whole of the left side of his chest. About a month after his admission he suddenly brought up a large quantity of blood, and died. At the post-mortem examination, the stomach and duodenum were full of blood. There was a large sarcomatous mediastinal growth, which had surrounded the lower end of the œsophagus, trachea, and aorta. The wall of the œsophagus at the level of the bifurcation of the trachea had been destroyed, and the aorta was perforated at the origin of the left subclavian artery, so that a direct communication between the œsophagus and the aorta had been established.

Foreign Body perforating the Œsophagus and Aorta.—Copious hæmorrhage, which may cause death, may be produced as a result of a foreign body, such as a pin, fishbone, or tooth-plate, perforating both the œsophagus and some large vessel, or even the aorta. A history of such a foreign body being swallowed, followed by a feeling of discomfort in the œsophagus, would suggest such a condition, which might be confirmed by the use of *x*-rays, bougies, or the œsophagoscope.

C. Diseases of the Stomach.

Acute Gastritis.—The mucous membrane of the stomach in this disease is congested, and small hæmorrhages and erosions may be present. They can be seen clearly with the gastroscope. The hæmorrhage which occurs is slight, in the form of streaks of blood mixed with mucus in the vomit, and it hardly merits the term hæmatemesis. Acute gastritis is caused most frequently by errors in diet, irritating or decomposing foods, alcohol, corrosive or irritant poisons, or sepsis from septic teeth, stomatitis, or pyorrhœa alveolaris. The chief symptoms are : a feeling of discomfort and tenderness in the epigastrium, nausea, eructations, vomiting, constipation ; or in children, diarrhœa : headache, a feeling of depression, furred tongue, foul breath, and concentrated urine. Pus corpuscles, or micro-organisms such as streptococci, pneumococci, or diphtheria bacilli, have been recovered from the gastric contents on appropriate examination in some cases.

Chronic Gastritis.—The mucous membrane of the stomach may be thickened and congested, with hæmorrhagic erosions scattered over its surface. The vomit usually consists of a good deal of mucus, and occasionally a little blood. It may follow acute gastritis, but most frequently is caused by the continual and excessive ingestion of alcohol, tea, coffee, and irritating and indigestible articles of diet. The main symptoms are : tenderness in the epigastrium aggravated by the taking of food, nausea, vomiting—especially in the early morning if due to alcohol—flatulence, foul breath, a furred tongue indented by the teeth at the edges, constipation, concentrated urine, and slight pyrexia.

Toxic Gastritis due to Corrosive Poisons.—Strong acids or alkalies destroy the mucous membrane of the stomach as well as injure that of the mouth, throat, and œsophagus. More or less constant vomiting of blood and blood-stained mucus is one of the most prominent symptoms, and it may be associated with intense pain in the mouth, throat, and abdomen, dysphagia, pain and tenderness behind the lower end of the sternum or in the epigastrium, distention of the abdomen, collapse, and a rapid, feeble pulse. The urine may contain blood and albumin, and, if the poison is oxalic acid, crystals of oxalate of lime. If corrosive poisoning is suspected, an inspection of the mouth and pharynx will show signs of corrosion, and an examination of the vomit will furnish evidence of the nature of the poison.

Arsenic.—The mucous membrane of the stomach is red, inflamed, partly detached, and covered with blood-stained mucus. The chief symptoms are nausea, violent and incessant sickness, burning pain in the epigastrium, diarrhœa, faintness, and depression. The vomit is usually a brownish, turbid fluid, mixed with mucus and streaks of blood. Later, there may be severe diarrhœa, with rice-water stools. Arsenic may be detected in the vomit.

Phosphorus, antimony, and other irritant poisons may also cause inflammation of the mucous membrane of the stomach, and lead to slight hæmatemesis.

Gastric Ulcer.—Hæmatemesis is the most important symptom of gastric ulcer, though it occurs in only about 50 per cent of the cases both in the acute and chronic forms of the disease, being due in the former to erosion of small vessels, and in the latter to the ulcerative process extending to and opening up larger gastric vessels, and occasionally even the pancreatic or splenic artery. The amount of blood varies within wide limits. If the quantity is small, or if it is gradually poured out into the stomach, it may remain there a sufficient time for the acid gastric juice to act on it and convert the hæmoglobin into hæmatin, which gives to the vomit a characteristic dark-brown 'coffee-grounds' appearance. In some cases the blood is not vomited but appears in the stools as melæna (p. 385). If a medium or large vessel is eroded the bleeding may be very copious, a quart or more of blood being vomited, either liquid and arterial in colour or in large red clots. A profuse hæmorrhage causes sudden pallor, a feeling of faintness, restlessness, syncope, and a rapid, feeble pulse. It is rarely fatal unless a large vessel such as the splenic artery has been eroded. Occasionally hæmatemesis is the first intimation of the presence of a gastric ulcer, but in the majority of cases there are other symptoms and signs which have preceded it. The disease was formerly said to occur most frequently in females, especially of the servant class, between the ages of twenty and thirty, but not a few of these cases diagnosed as gastric ulcer are probably examples of gastrostaxis, or bleeding from a spongy, oozing mucosa without any definite and macroscopic ulceration. An analysis of 101 fatal cases showed 59 males and 42 females, and it is quite as common in later as in early adult life. It does occur before puberty sometimes, but very rarely. Like duodenal ulcer, it seems to be commoner in the North of England than in the South, and in mining and manufacturing rather than in rural districts. In addition to hæmatemesis, the signs most characteristic of gastric ulcer are abdominal pain, nausea, vomiting, and melæna. Pain is felt in the epigastrium just below the ensiform cartilage; it usually

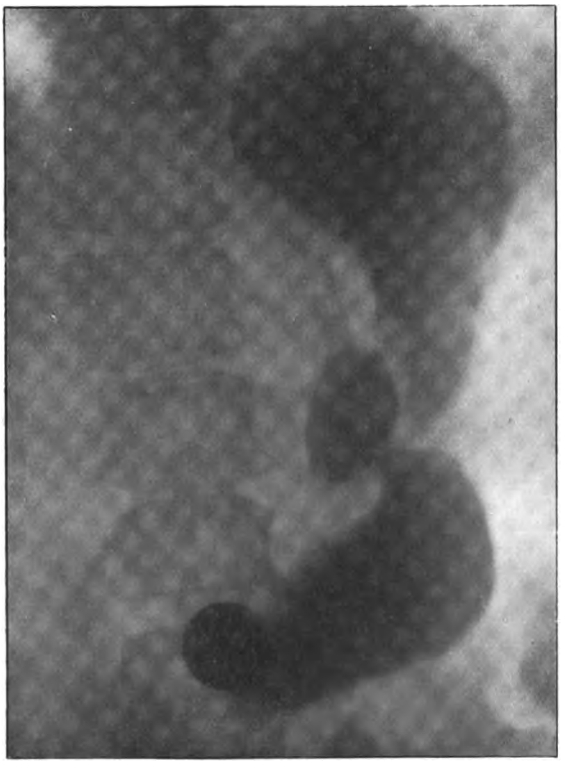


Fig. 128.—Skiagram of bismuth in an hour-glass stomach.

begins a few minutes after the ingestion of food, but in some cases is not experienced until an hour or two afterwards. Pain may also be felt in the back, between the tenth dorsal and first lumbar spines. Its character and intensity are very variable, but it is usually severe. Hyperæsthesia of the skin and tenderness on pressure in the epigastrium may also be present. Vomiting may come on immediately after food is taken, or may be deferred for an hour or two, being preceded usually by a good deal of pain. The vomit has a very acid taste and an abnormally acid reaction. Melæna follows hæmatemesis; occasionally it may precede it, and rarely occurs independently. The tongue in the majority of cases is clean, red, moist, and steady. There is more or less anæmia; the points of distinction between gastric ulcer and anæmic vomiting are discussed on page 36.

Simple ulcer is sometimes difficult to differentiate from cirrhosis of the liver or carcinoma of the stomach. Examination of the stomach with the *x*-rays after a bismuth or barium meal sometimes affords positive evidence of the nature of the lesion (*Fig.* 128), especially when there is some stenosis of the pylorus; but it is possible to have an active ulcer and yet for the *x*-ray appearances of the stomach to be normal.

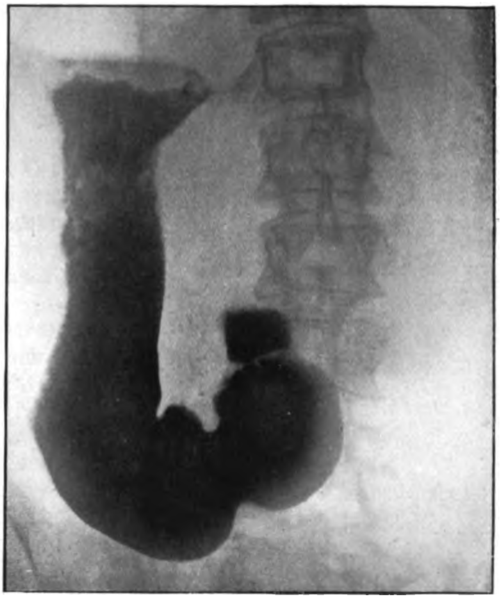


Fig. 129.—Skiagram of a normal stomach after a bismuth meal. Female, age 28.

(*Skiagram by Dr. C. Thurstan Holland.*)

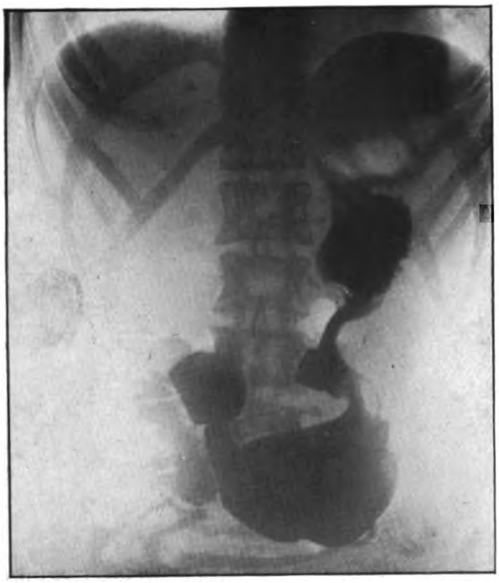


Fig. 130.—Skiagram taken in the erect posture after a bismuth meal in a case of malignant disease of the middle of the stomach. The pyloric end was normal. The diagnosis was verified by operation.

(*Skiagram by Dr. C. Thurstan Holland.*)

Gastrostaxis (see ANÆMIA, p. 36).

Hæmorrhagic erosions are probably the earliest stage of gastric ulcer, though they may not develop beyond the phase of minute erosions. They may be the actual cause of gastrostaxis, and perhaps the distinctions between gastrostaxis, hæmorrhagic erosions, and multiple small gastric ulcers are differences of degree and not of kind. There are certain conditions, however, especially acute malignant fevers, purpura, infective endocarditis, and similar septic states, in which a general tendency to subcutaneous and submucous hæmorrhages leads to multiple small gastric erosions, which produce hæmatemesis without being directly related to ordinary gastric ulcer.

Carcinoma.—Hæmatemesis is a less frequent and important sign of carcinoma of the stomach than of gastric ulcer, for it occurs in but a little over 20 per cent of the cases, and even then is generally slight. Bright-red blood is rarely seen in the vomit, for the slow ooze from the ulcerated surface of the growth allows the blood to remain in contact with the gastric juice and develop the 'coffee-grounds' appearance. About 60 per cent of the cases occur between the ages of forty and sixty. The chief symptoms and signs of the disease are: pain in the epigastric region, nausea, vomiting, anorexia, loss of weight and strength, pyrexia, anæmia, cachexia, and the presence of an abdominal tumour. Pain is one of the earliest symptoms, but it varies considerably in degree and position. It is referred most frequently to the epigastrium, but is not as a rule so severe as in gastric ulcer. Vomiting is another early symptom which varies in frequency and character according to the position of the growth. When the pylorus is involved and stenosed, the stomach dilates and a large quantity of frothy, brownish vomit is evacuated every two or three days; in cases of diffuse carcinoma the capacity of the stomach is diminished, and a small quantity may be vomited two or three times a day; when the growth is situated at the cardiac orifice, the symptoms resemble those of epithelioma of the œsophagus, and the food immediately after swallowing is regurgitated rather than vomited; whilst in cases of growth which involve neither of the orifices of the stomach, there may be no vomiting, or if present it may have no special characteristics, the symptoms being mainly those of dyspepsia or gastritis. A chemical analysis of the vomit may show deficiency of hydrochloric acid and the presence of lactic acid; but the value of this test is limited: first because there are a great many other conditions besides carcinoma of the stomach in which there is deficiency or absence of free HCl in the gastric contents—cachexia of any kind, cirrhosis of the liver, heart disease with failing compensation, enteric, pneumonia and other fevers, achylia gastrica, pernicious anæmia, and many other conditions under which the patient is ill enough for all his secretions to suffer, amongst them the gastric juice; and secondly because insufficient care is sometimes taken to recover the gastric contents at the right time after a meal. All persons fail to show free hydrochloric acid in the gastric juice for some while, generally three-quarters of an hour at least, after a meal containing proteid. The more the proteid the longer it takes for the stomach to accumulate sufficient HCl, first to combine with all the molecules of proteid as combined HCl, and later to permit of a surplus of uncombined or free HCl. Merely to test a vomit obtained at hazard is of little use therefore; one must know what food was taken previously, and how long before; it is on this account that test meals of known composition are employed, followed by lavage at such an interval afterwards that free HCl is to be expected unless there is some disease interfering with the proper formation of the gastric juice. A growth in the stomach may be seen with the aid of the gastroscope. The loss of weight and strength

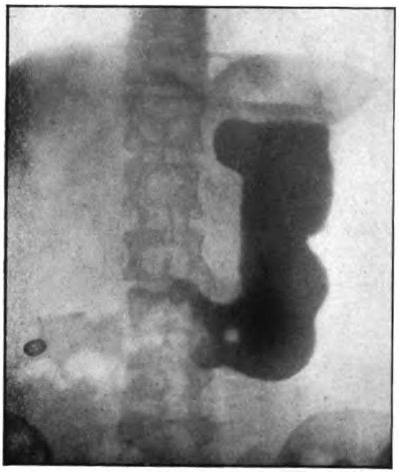


Fig. 131.—Skiagram after a bismuth meal in a case of carcinoma of pylorus and pyloric end of stomach.

(*Skiagram by Dr. C. Thurstan Holland.*)

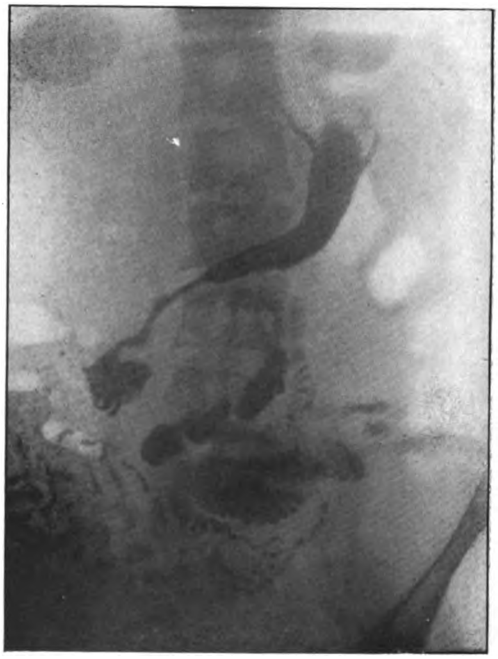


Fig. 132.—Showing bismuth in a 'leather-bottle' stomach —diffuse carcinoma of the stomach—taken after four tablespoonfuls of food (all the patient could take) had been swallowed. Food 'ran through' the stomach and duodenum into the small bowel.

(*Skiagram by Dr. C. Thurstan Holland.*)

are usually progressive, and they are amongst the most constant and characteristic signs of the disease. Anæmia of the secondary chlorotic type, with a low colour-index, may be so prominent a symptom that a primary anæmia may be suspected until a careful blood examination has been made. A very careful investigation of the abdomen must be made, for in about 70 per cent of the cases a tumour may be felt, though it is to be hoped that the gastroscope will lead to the diagnosis of carcinoma ventriculi before this stage is reached, and when surgical cure is still possible. The position and character of the tumour vary according to the part of the stomach which is involved. Pyloric growth may cause the abdomen to be distended as a result of gastric dilatation, and a movable tumour may be felt above the umbilicus, near the middle line and to the right of it. When the cardiac orifice is involved, there may be no tumour to be felt, and the same applies to the small 'indiarubber-bottle' stomach of diffuse carcinoma ventriculi (*Fig.* 132). Tumours of the body of the stomach may be felt in the epigastrium, or below the left costal margin. It may be necessary to examine under a general anæsthetic in some cases; *x*-ray examination after a bismuth or barium meal may assist in some cases (*Fig.* 131); and even laparotomy may be advisable as a diagnostic measure under some circumstances. If the patient is weighed carefully twice a week, and is proved to increase in weight steadily under treatment, carcinoma is improbable, provided this increase does not prove transient after a week or two.

Injuries.—Hæmatemesis may follow blows, stabs, or gunshot wounds in the epigastric region, or the passage of instruments or foreign bodies, such as a broken thermometer, into the stomach. The history and the evidence of any such occurrence would make the diagnosis sufficiently obvious.

Atheroma in association with arteriosclerosis or granular kidney and high blood-pressure may lead to hæmatemesis in very exceptional cases by causing weakness and rupture of small gastric vessels. Such a diagnosis should be made with extreme caution, even when other symptoms and signs of atheroma are present, for hæmatemesis as the result is exceedingly rare. It is, however, not uncommon in *splenomegalic polycythæmia* (p. 534), in which malady the blood-pressure is generally high.

Abdominal Aneurysm opening into the Stomach.—Aneurysm of the abdominal aorta is uncommon. The sac may rupture into the stomach, however, and lead to a sudden, profuse, and fatal attack of hæmatemesis. The chief diagnostic signs are: an epigastric tumour, with distinct expansile pulsation and severe pain both in the abdomen and in the back over the site of the bulge, in a patient who has suffered from syphilis and has been accustomed to repeated and violent muscular exertion.

D. Diseases of the Duodenum.

Duodenal Ulcer.—Hæmatemesis is caused in the same manner in this disease as in gastric ulcer, viz., by the erosion of small duodenal blood-vessels or by the ulcerative process spreading to and opening larger and deeper blood-vessels outside. The ulcers are in the first part of the duodenum in a very large proportion of cases. They are about eight times as common in men as in women. Some of the symptoms are similar to those of gastric ulcer, viz., hæmatemesis, melæna, abdominal pain and tenderness, anæmia, and vomiting. Hæmatemesis, however, is not so frequent as it is in gastric ulcer; it is generally less marked than is the melæna, and the latter may occur independently of hæmatemesis or before it. In the acute form of the disease there may be a copious intestinal hæmorrhage in an apparently healthy person, accompanied by acute pallor and followed by the evacuation of a mixture of black altered blood and bright arterial blood from the rectum. The more the bleeding, the greater the tendency for the blood passed to be still bright red. There may be no pain at all, but more often it is considerable; there is hardly any part of the abdomen to which it may not be referred, but generally it is deep-seated in the upper part, about an inch below the tip of the ninth right rib, more to the right of the middle line than is that of gastric ulcer, and usually its onset is two or three hours after the ingestion of food. One point about this pain that is almost pathognomonic is the way in which, coming on when the patient is beginning to get hungry—'hunger pain'—it is often relieved entirely by taking food. Vomiting is another important symptom which may be very troublesome, though in some cases it is entirely absent. One characteristic of some cases of duodenal ulcer is the very rapid way in which, under the *x*-rays, bismuth may be seen emptying out of the stomach into the duodenum.

Carcinoma of the Duodenum is very rare, and would only be diagnosed if there were general symptoms of malignant disease together with a fixed tumour in the situation of the duodenum.

Gall-stones ulcerating through from the Gall-bladder into the Duodenum may cause hæmatemesis and melæna. Previous attacks of pain occasioned by the gall-stone might lead to a diagnosis of gastric or duodenal ulcer ; but if the pain was colicky in character, and was associated with tenderness and enlargement of the liver, pain over the gall-bladder, and jaundice, it would point to a gall-stone. The diagnosis might be confirmed by the discovery of the stone in the fæces, or, in the case of a larger calculus, by the occurrence of acute intestinal obstruction from its impaction in the small intestine. As a cause of hæmatemesis this condition is naturally very rare.

E. Portal Obstruction.

As a result of obstruction to the flow of blood through the portal vein, passive congestion and hæmorrhagic erosion of the mucous membrane of the stomach, and varicose gastric and œsophageal veins, may be produced. Hæmatemesis may then arise through oozing of blood from the congested mucous membrane, or from an actual escape of blood in the case of hæmorrhagic erosion or the rupture of one of the varicose veins. The signs which are common to portal obstruction in addition to hæmatemesis are : nausea, vomiting, ascites, œdema of the legs, albuminuria, and the presence of dilated and tortuous superficial abdominal veins ; the chief cause is :—

Cirrhosis of the Liver.—This disease is one of the commonest and most important causes of profuse hæmatemesis, and it is often difficult to diagnose from gastric ulcer or carcinoma of the stomach. Hæmatemesis may be one of the earliest symptoms, and it is frequently profuse, and very liable to recur, though it is seldom fatal. There may be a history and the signs and symptoms of chronic alcoholism (p. 726). The liver may be enlarged, its surface rough and hard, and its edges irregular and beaded. The spleen may be enlarged as a result of the portal obstruction, but in adults rarely attains to such an enormous size as it does in the splenomegalic variety of cirrhosis in children and young adults. In distinguishing this disease from carcinoma of the stomach, it is very important to determine, if possible, the absence of a stomach tumour and of large nodules projecting from the surface of the liver.

Adhesive Pylephlebitis.—Non-suppurative thrombosis of the portal vein is very rare, and difficult to diagnose. It may, however, give rise to sudden and profuse hæmatemesis. It is distinguished from other forms of portal obstruction by the relatively sudden onset of ascites, hæmatemesis, melæna, and enlargement of the spleen, and by an absence of signs and symptoms of cirrhosis of the liver and other causes of portal obstruction.

Pressure on the Portal Vein.—Hæmatemesis, when due to this cause, is generally associated with ascites and intense jaundice, since the common bile duct is liable to be compressed as well as the portal vein, on account of their close proximity to each other. (See JAUNDICE, p. 329.)

Some cases of Enlarged Spleen (see SPLEEN, ENLARGEMENT OF, p. 628).—Hæmatemesis is a fairly common symptom in cases of enlarged spleen, even when the enlargement is not associated with cirrhosis of the liver or leukæmia. Osler explains the occurrence as being due to the " intimate relation between the vasa brevia and the splenic circulation " ; sometimes, however, it results from actual thrombosis of the splenic vein, though the diagnosis of this is scarcely possible without laparotomy or autopsy.

F. Acute Febrile Diseases.

Malignant Variola.—Hæmatemesis occurs in about a third of the cases of hæmorrhagic small-pox. It is associated with cutaneous, subcutaneous, and submucous hæmorrhages, hæmaturia, epistaxis, melæna, and bleeding from the gums. The sudden initial rigor, intense backache and headache, severe vomiting, epigastric pain, cutaneous hæmorrhages, and the diffuse hyperæmic rash with small punctiform hæmorrhages which appears first on the groins and lower part of the abdomen, would point to a diagnosis of hæmorrhagic or black small-pox if such a case occurred during an epidemic of the disease.

Malignant Scarlet Fever.—In the hæmorrhagic form of scarlet fever, hæmatemesis may occur; but hæmaturia, epistaxis, and cutaneous hæmorrhages are more frequent. The sudden and severe onset, the very high temperature, the extremely rapid and feeble pulse, the headache and delirium, and the appearance of the characteristic rash on the second day would point to scarlet fever.

Yellow Fever.—'Black vomit' due to the presence of altered blood is one of the most characteristic features of this disease. Hyperæmia and catarrhal swelling of the mucous membrane is the only change which is found in the stomach. It is essentially a disease of tropical and sub-tropical countries. The onset is sudden, with a chill, headache, and severe pain in the back and limbs. The face is flushed, and very soon jaundice appears. After the first day the pulse-rate drops, so that with a temperature of 103° or 104° the pulse may be only 70 or 80. Albuminuria is another early symptom, which may appear on the third day. In addition to the black vomit, there may be cutaneous petechiæ and bleeding from the gums. It is often difficult to distinguish from malignant malaria, though in the early stages of malaria jaundice, albuminuria, and hæmaturia are extremely rare, whilst an examination of the blood may reveal the presence of malarial parasites.

Cholera may be associated with hæmatemesis sometimes. The sudden onset of acute gastro-intestinal symptoms, the rapidly repeated rice-water stools, and the epidemic nature of the malady, all point to the diagnosis, which may be confirmed by recovering the vibrio from the motions bacteriologically.

Acute Yellow Atrophy of the Liver.—Hæmatemesis is the commonest form of hæmorrhage in this rare disease. Women between twenty and thirty are affected more frequently than men, especially during and just after pregnancy. It sometimes follows fright and mental emotion. The first symptoms are indistinguishable from catarrhal jaundice—viz., malaise, loss of appetite, nausea, vomiting, and jaundice. The vomiting soon becomes intractable, the jaundice increases, and drowsiness, restlessness, and delirium supervene. The vomit is black, and may resemble treacle, its appearance being due to altered blood. Melæna, epistaxis, and subcutaneous petechiæ may be noticed. The tongue becomes dry and brown; the liver dullness diminishes; the urine shows characteristic changes in the marked diminution in the amount of urea and the presence of bile pigment, whilst leucin and tyrosin crystals in it (*Fig.* 148, p. 333) are an important diagnostic sign of this disease.

G. Blood Diseases.

Purpura Hæmorrhagica.—Hæmorrhage from the stomach is rare in this disease. Hæmatemesis may occur, however, as a result of blood derived from the mucous membrane of the nose or mouth being swallowed. As purpura is a symptom rather than a disease in the majority of cases, before making a diagnosis of purpura hæmorrhagica or idiopathica, those diseases which lead to symptomatic purpura must be excluded (see PURPURA, p. 552). An examination of the blood must also be made, to exclude pernicious anæmia and leukæmia; and blood cultures may be required.

Scurvy.—Hæmatemesis is uncommon, only occurring in severe and well-marked cases, so that the diagnosis is not difficult. The swollen and spongy gums, anæmia, cutaneous hæmorrhages around the hair sacs, and subcutaneous indurations, in a patient who is found to have been living on a diet deficient in quantity and in vegetables, would point to scurvy.

Hæmophilia.—Out of 334 cases analyzed by Grandidier, there were only fifteen examples of hæmorrhage from the stomach. Excessive bleeding from slight cuts or after tooth extraction, epistaxis, bleeding from the mouth, and hæmorrhage into the joints, are the earliest and the commonest manifestations of the disease. The association of hæmatemesis with hæmorrhage from other parts, and with hæmorrhage into joints in particular, in a patient whose near male relations show a tendency to bleed on the slightest provocation, would point to hæmophilia. There are no pathognomonic blood changes.

Leukæmia.—Hæmorrhages from and into various parts, especially epistaxis, are common in this disease. Hæmatemesis may be the actual cause of death. Its association with enormous enlargement of the spleen is by no means pathognomonic of leukæmia, for the two conditions may be present in chronic malaria, splenic anæmia, and splenomegalic cirrhosis. An accurate diagnosis cannot be made until the blood has been examined and a high degree of leucocytosis found (100,000 to 1,000,000 white blood corpuscles per c. mm.), with a large proportion of myelocytes in the case of splenomedullary leukæmia and a high percentage of lymphocytes (90 per cent) in lymphatic leukæmia.

Hodgkin's Disease.—In the late stages of this disease there is a tendency to hæmorrhage from and into various parts of the body, e.g., epistaxis, bleeding from the mouth, cerebral hæmorrhage, and rarely hæmatemesis. There should be little difficulty in making a diagnosis, as hæmatemesis would be a late symptom; the characteristic features of the disease are described on pages 37 and 635.

Chlorosis.—It is difficult to determine whether hæmatemesis occurring in an anæmic woman under thirty is due to gastric ulcer or to gastrostaxis (p. 36). That chlorosis has something to do with hæmatemesis, apart from the formation of macroscopic ulcers, is probable; it is also probable that chlorosis predisposes to gastric ulcer. The precise nature of the symptom in a chlorotic girl is therefore very difficult to determine, some observers diagnosing gastric ulcer where others prefer to label the condition gastrostaxis. The former withhold solid food longer than the latter, and are perhaps inclined to recommend operation more readily; these are the main grounds for striving to draw a clear distinction between the two conditions.

Young women suffering from chlorosis are usually well nourished. The skin may have a greenish tinge and the sclerotics a distinct bluish appearance. Œdema of the feet, dyspnœa, palpitation, and amenorrhœa are prominent symptoms; but the diagnosis cannot be made with any certainty until the blood has been examined. It is pale and thin; the red blood-corpuscles are reduced in number, but rarely are under 3,000,000 per c.mm.; the average size of the red blood-corpuscles is below normal; the hæmoglobin is reduced much more in proportion than are the red blood-corpuscles, so that the colour-index is low, being as a rule about 0·5 or under; the white blood-corpuscles are not increased, and the differential leucocyte-count is almost normal. Seeing that amenorrhœa and hæmatemesis are both liable to occur in anæmic girls, the gastric hæmorrhage has sometimes been regarded as vicarious menstruation; there is little evidence to support this view of its pathology, however, and when the bleedings recur, the attacks do not show any monthly regularity.

Pernicious Anæmia.—Hæmatemesis is a very rare symptom in pernicious anæmia; when it occurs, the difficulty in distinguishing between this disease and carcinoma of the stomach is much increased. A correct diagnosis cannot be made until the blood has been examined (p. 24). The urine contains pathological urobilin.

Malarial Cachexia.—Anæmia and enlargement of the spleen may follow repeated attacks of malaria, and severe hæmatemesis may be a prominent symptom. In making the diagnosis, the history of residence abroad, of attacks of ague, and the condition of the blood, must be relied on. A normal or a diminished number of leucocytes, with a relative increase in the large mononuclear cells beyond 15 per cent, is strong presumptive evidence of a previous malarial infection.

Splenic anæmia may run its course without any hæmatemesis; on the other hand, the latter is sometimes one of the most serious symptoms in the case, and may be the cause of death. The chief features of the malady are described on p. 37.

H. **Miscellaneous.**

Chronic Interstitial Nephritis.—Hæmatemesis occasionally, but very rarely, occurs in this disease. Its association with anæmia, thickened and tortuous superficial arteries, high-tension pulse, hypertrophy of the heart, albuminuric retinitis, polyuria, and urine of low specific gravity containing a variable quantity of albumin and renal tube-casts, would point to chronic interstitial nephritis as the cause. It is most important that the blood-pressure should be measured instrumentally, and not guessed at by palpation.

Following Abdominal Operations.—Hæmatemesis may occur after severe abdominal operations, independently of any injury to the stomach or duodenum. Should death occur, no obvious lesion can be found in the stomach to account for it in the majority of cases. The reason of the occurrence of such an alarming symptom remains a mystery in many cases, but in some infective conditions, such as appendicitis, multiple minute ulcers have been found.

Prolonged Jaundice.—The importance of this condition as a cause of almost any variety of bleeding lies chiefly in the added danger attending operations in such cases.

Syphilis of the Liver is sometimes cited as being itself a cause of hæmatemesis; the difficulty is to exclude the possibility of alcoholic cirrhosis in such a case; it is doubtful whether syphilis alone can cause the vomiting of blood. *Herbert French.*

HÆMATOPORPHYRINURIA.—(See Urine, Abnormal Coloration of, p. 744.)

HÆMATURIA.—Blood may appear in the urine as the result of injury, of disease in some portion of the urinary tract, or of other organs involving the urinary apparatus, or of a few general diseases of other parts of the body. The blood may be present in large, small, or microscopic amounts, it may continue for days or even weeks, or appearing suddenly and without apparent cause, may disappear completely for a variable period. Further, it may be present in the urine either as corpuscles or as hæmoglobin, and it is necessary to distinguish between the two conditions. In hæmoglobinuria the urine is dark brown from the presence of methæmoglobin, and any deposit is found to consist of brownish débris in which no red blood-corpuscles can be found (see Hæmoglobinuria, p. 284). Occasionally the colouring matter of the blood may escape from the corpuscles if the stained urine has been retained for any length of time in the bladder, when crenated or disintegrated corpuscles will be found on microscopic examination of the sediment. The following list gives the chief causes of hæmaturia:—

I.—HÆMATURIA FROM AFFECTION OF SOME PART OF THE URINARY ORGANS.

A. **Renal Causes.**

Profuse.

Malignant tumours of the kidney:
  Hypernephroma
  Embryoma
  Carcinoma
  Sarcoma
Innocent tumours—papilloma of pelvis, angioma
Injury of the kidney
Calculus
Tuberculosis
So-called 'essential' hæmaturia

Slight.

Calculus
Tuberculosis
Renal mobility
Hydronephrosis
Polycystic disease
Injury
Oxaluria
Nephritis, acute and subacute
Coli bacilluria
Drugs: turpentine, carbolic acid, cantharides

B. **Ureteric Causes.**

Calculus in the ureter.

C. **Vesical Causes.**

Profuse.

Villous tumour
Papilloma
Villus-covered carcinoma
Prostatic adenoma or carcinoma

Slight.

Epithelioma
Tuberculosis of bladder or prostate
Calculus
Acute cystitis
Bilharzia hæmatobia
Injury

D. **Urethral Causes.**

Acute urethritis, impaction of calculus, injury
Acute spermato-cystitis
Nævus

II.—HÆMATURIA FROM DISEASE OF THE NEIGHBOURING VISCERA INVOLVING THE URINARY ORGANS.

Carcinoma of the uterus, vagina, or rectum
Acute appendicitis
Acute salpingitis
Pelvic abscess
Dysenteric or tuberculous ulceration of the intestine.

III.—HÆMATURIA IN GENERAL DISEASES.

Renal infarction in endocarditis
Arteriosclerosis
Leukæmia
Purpura and scurvy
Hæmophilia
Acute fevers, malaria, small-pox, and yellow fever.

In considering the diagnosis of a case presenting hæmaturia as a symptom, it is seldom that there are not other symptoms present, such as pain, tumour, or increased frequency of micturition, which will point to one or other organ as the source of the bleeding; but in some cases hæmaturia may be the only symptom. The following points will often help in the differential diagnosis:—

The Colour of the Urine.—If the urine is stained a bright red colour, the hæmorrhage is most likely to arise from the bladder or lower urinary tract. Dark-coloured blood in the urine may, however, be due to the retention of blood in the bladder for some time, or from the large amount present in the urine.

The Distribution of the Blood in the Urine during Micturition.—If the urine during micturition is only tinged with blood during the final expulsive efforts, or if the terminal urine is stained more deeply than the rest, the source of the hæmaturia is almost certainly in the bladder. If the first urine passed is blood-stained and the remainder clear, the bleeding is probably from the urethra or prostate; whereas if the urine is evenly stained with blood throughout, it suggests that the source of hæmorrhage is in the kidneys, although a vesical lesion which causes more than a slight hæmorrhage may also give rise to a deeply blood-stained urine throughout micturition.

The Quantity of Blood Present in the Urine.—A large quantity of blood in the urine, in the absence of traumatism, suggests some form of growth in the bladder or kidney. Papillomata and villus-covered carcinomata in the bladder may cause sudden profuse hæmorrhage without pain or other symptom, whilst equally profuse hæmorrhage may arise from a malignant tumour in the kidney which has invaded the renal pelvis. Examination of any clots of blood passed may occasionally afford useful information in determining the seat of hæmorrhage. The urine should be poured into a large flat tray containing water, and the clots floated out, when some may show the triangular or pyramidal shape indicating their formation in the renal pelvis, or others the thin, worm-like form with tapering or decolorized ends from their formation in the ureter; their passage down the ureter is accompanied by the same acute renal colic that is caused by renal calculus. Clots formed in the bladder are flat, disc-like, but often broken up in their passage through the urethra.

If the quantity of blood is increased by movement or exercise, suspicion of renal stone or growth will arise. In a recent case, profuse hæmaturia occurred after three successive railway journeys, when the lesion found at operation was an early carcinoma of one kidney which had recently invaded the renal pelvis.

The Association of other Elements from the Urinary Organs with Blood in the Urine.—Microscopical examination of the deposit obtained by centrifuging the urine may reveal cellular elements distinctive of the renal pelvis or vesical mucous membrane or epithelial, granular, and blood-casts from the renal tubules (*Plate I.* p. 6), which may help in the diagnosis in a case of hæmaturia. The presence of a number of urinary crystals in a urine of acid reaction will point to renal calculus. Occasionally, small pieces of growth may be passed in the urine from the delicate villous papilloma or villus-covered carcinoma of the bladder, and more rarely plugs of muco-pus from a caseous tuberculous cavity in the kidney may be found. It is important to remember that the presence of a villous tuft in the urine gives no indication whether it is derived from an innocent papilloma or a villus-covered carcinoma, as it becomes detached only from the surface of the growth.

The association of pus with blood in the urine does not give much assistance in determining the seat of the bleeding. Both pus and blood will often be present with either calculus or tuberculosis of the kidney or bladder, and may both be present with vesical growth or with prostatic enlargement.

The Amount of Albumin.—If the amount of albumin in the urine is in excess of that which would be due to the amount of blood present, the bleeding is probably renal in origin.

The Reaction of the Urine is of very slight assistance in determining the source of bleeding. Generally speaking blood in an acid urine is more likely to be derived from the kidney than from the bladder; this, however, is no universal rule, for blood may be present in an acid urine in a case of vesical calculus or growth; whereas, on the other hand, there may be blood in alkaline urine in a case of renal calculus as well as in pathological conditions of the bladder.

The association of *unilateral lumbar pain*, situated in the angle between the last rib and the border of the erector spinæ muscle, passing forwards above the iliac crest into the groin, with occasional attacks of colic, would suggest a renal lesion; whilst hæmaturia, accompanied by *increased frequency of micturition*, or by *penile pain* immediately following micturition, would indicate vesical disease. This statement must of necessity be taken in a very general sense, for exceptions to it are frequent. Thus a vesical tumour causing hæmaturia may implicate an ureteric orifice sufficiently to cause increased intra-renal tension on that side with lumbar aching or even enlargement of the kidney; whilst on the other hand a tuberculous lesion in the kidney, with descending ureteritis, may cause increased frequency of micturition before there is any vesical infection. Equally important is it to take into consideration the *age of the patient*; thus, in a young adult, continued slight hæmaturia with increased frequency of micturition are highly suggestive of tuberculous disease of the kidney, whereas slight hæmaturia in a more elderly patient suggests vesical carcinoma or calculus. At any age, severe hæmaturia may be present with a villous tumour of the bladder, or in a patient more advanced in years with renal growth or prostatic enlargement.

Further evidence of the source of the hæmorrhage may be obtained upon the physical examination of the patient. This should be carried out systematically, and not only should the urinary organs be examined, but any evidence of disease elsewhere in the body, as in the heart, lungs, blood, liver, or pelvic organs, sought for also. Each kidney should be examined bimanually, one hand being placed in the angle made by the last rib and the margin of the erector spinæ muscle, and the other in front, immediately below the costal margin; the patient is then directed to breathe deeply whilst pressure is maintained by the two hands, when an enlarged or unduly mobile kidney may be felt to descend, or may be grasped on deep inspiration. Any pain or undue tenderness on either side should be noted, especially any sharp, pricking pain experienced by the patient if the anterior hand be depressed suddenly, a sign said to be indicative of renal stone.

Examination of the bladder by palpation in the suprapubic area may elicit pain in acute inflammatory conditions, or may give evidence of a distended bladder in a case of hæmaturia from prostatic obstruction; but much more knowledge may be gained by a thorough *rectal examination*. For this purpose the patient should assume the knee-elbow position, when the examining finger can explore not only the prostate, but the vesiculæ seminales, the lower end of each ureter, and the bladder base, as well as the lateral pelvic wall. The prostate may show adenomatous enlargement, or may be infiltrated with primary carcinoma—which has recently been shown to be far from uncommon,—when the gland will present marked, firm, rounded nodules, and will often be immovable. Search should be made for any nodules in the prostate or vesicles, or thickening of the lower end of the ureter, suggestive of tuberculous disease, or thickening or infiltration in the bladder base, which may often be felt in a case of vesical carcinoma. Examination in the lateral pelvic space may show infiltration of the pelvic lymphatics, or enlargement of the lymphatic glands in a case of carcinoma of the bladder or prostate. *Examination of the testes* should always be made. A nodule in either epididymis may indicate tuberculous disease which may have spread to the urinary organs, but care must be taken not to mistake a nodule dating from a gonorrhœal epididymitis for one due to tuberculous disease.

Great assistance may be obtained by the use of the *cystoscope* (*Plates XV*, p. 282, and *XVI*, p. 284). Needless to say, the greatest gentleness must be used in carrying out any instrumentation, to avoid any further hæmorrhage, which would obscure a view by the cystoscope, and if any bleeding is present, an attempt should be made to arrest it by irrigation of the bladder with silver nitrate 1–1000, or with adrenalin solution of the same strength. If the bleeding is profuse, it is probably impossible to obtain a satisfactory view of the interior of the bladder, but with even moderate hæmorrhage going on a rapid distention of the bladder may produce a medium clear enough to obtain a view which will show the seat of hæmorrhage. Thus in renal hæmaturia blood-stained urine may be seen to be emitted from one ureteric orifice (*Plate XV*, *Fig. A*, p. 282) and clear urine from the other before the medium is too obscured; or with vesical hæmorrhage a vesical tumour may be seen. Even slight hæmorrhage will, however, rapidly render the medium in the bladder too hazy for a satisfactory examination of any minute changes in the vesical wall to be obtained—tuberculous disease for example. Hence it is better, if possible, to undertake any cystoscopic examination in the interval between attacks of bleeding, when the bladder can be examined thoroughly and any pathological lesion found. If no evidence is obtained in this way, a further examination may be conducted during an attack of bleeding.

I. HÆMATURIA FROM AFFECTION OF SOME PART OF THE URINARY TRACT.

A. Renal Causes.

The Malignant Tumours of the Kidney, hypernephroma, embryoma, carcinoma, and sarcoma, are all associated with profuse hæmaturia at intervals. Hypernephromata are the most common ; they arise in the cortical portions of the kidney, and are of comparatively slow growth. The embryomata may occur in small children or in elderly persons, whilst the true carcinomata and sarcomata are much more uncommon. These tumours cause an aching in the loin, and may lead to considerable enlargement of the kidney before any hæmaturia occurs. In the progressive growth of the tumour, the renal pelvis is involved gradually and hæmaturia is evoked. This is usually severe in type, so that clots may be formed in the calices of the renal pelvis or in the ureter, and cause the typical pain of renal colic in their descent of the latter. The renal tumour usually maintains the shape of the kidney, but in some cases may present a nodular form. Hence profuse hæmaturia, with clots of pyramidal or worm-like shape, associated with renal enlargement, is strongly suggestive of a renal malignant growth.

The only common form of *innocent tumour* in the kidney is papilloma of the renal pelvis. This gives rise to profuse hæmaturia and to renal enlargement, which in this instance is due to hydro- or hæmato-nephrosis from the obstruction to the ureter by the papillary growth or by blood-clot. Thus the renal tumour may vary in size. Papillomata of the mucous membrane of the renal pelvis are accompanied occasionally by similar growths in the ureter, and may also show a similar growth at the ureteric orifice upon inspection of the bladder.

An *angioma* of the kidney, forming a distinct tumour in the renal tissues and causing profuse hæmaturia, has recently been described by the writer, whilst Hurry Fenwick has described cases of angioma of the apex of a renal papilla causing hæmaturia.

Injuries to the Kidney may cause hæmaturia ; the diagnosis is usually obvious. The history of the accident, a blow or squeeze to the lumbar region, associated with hæmaturia, would point to an injury to the kidney. There may be renal enlargement, but this must be diagnosed from an extravasation of blood in the perinephric tissues from the rupture of the renal cortex. Comparatively slight injury to the loin may produce hæmaturia from a small lesion in the renal tissues, whilst in some cases there is no sign or recollection of external violence. In any case of hæmaturia following traumatism, it is essential to diagnose an injury to the kidney from injury to the *urethra* or *bladder*. In urethral injury the canal may be merely contused, or partially or wholly ruptured ; blood may be found at the urethral meatus or may be marked in the first portion of any urine that may be passed, whilst if the urethra be entirely divided, signs of extravasation of urine, with inability to micturate, will appear.

If the bladder be injured, blood may be present in any urine drawn off ; or after rupture of the bladder involving the peritoneal coat fluid may be found in the abdominal cavity. The length of time between the last passage of urine and the occurrence of the accident should be ascertained, and a catheter passed ; very gentle irrigation of the bladder with sterile fluid should be carried out in any suspected rupture of the viscus, to see if the amount of fluid run into the bladder is duly returned. At the same time, a thorough examination of the bony pelvis should be made for any sign of fracture, which is frequently the cause of direct injury to the bladder or urethra.

In *Renal Calculus* the bleeding is seldom profuse, is usually associated with a small amount of pus, and frequently is increased after exertion or the jolting of a journey. The subject of a renal stone will usually complain of pain in one loin of a constant aching character, which will remain of this character so long as the stone remains embedded in the renal tissues, in which condition slight hæmaturia is often present. When, however, the calculus projects into or is free in the renal pelvis, the urine also contains a small quantity of pus, and attacks of renal colic come on, characterized by very acute pain in the loin, passing forwards and downwards to the groin, upper part of the thigh, and testicle of the same side, and accompanied by frequent desire to pass urine. The calculus may be passed into the bladder along the ureter, may become impacted in the course of the ureter, or may remain in the renal pelvis, in which case successive attacks of renal colic may occur. The previous passage of a small calculus per urethram, following an attack of renal colic, is an important point in the history of such a patient, but in any case an examination by skiagraphy should be carried out, when a calculus may be proved present in the kidney (*Fig.* 133). A calculus in the kidney may attain a size too large to become engaged in the upper end of the ureter, when renal colic will be absent, or it may cause hydronephrosis, renal abscess, or pyonephrosis, of which symptoms may be present.

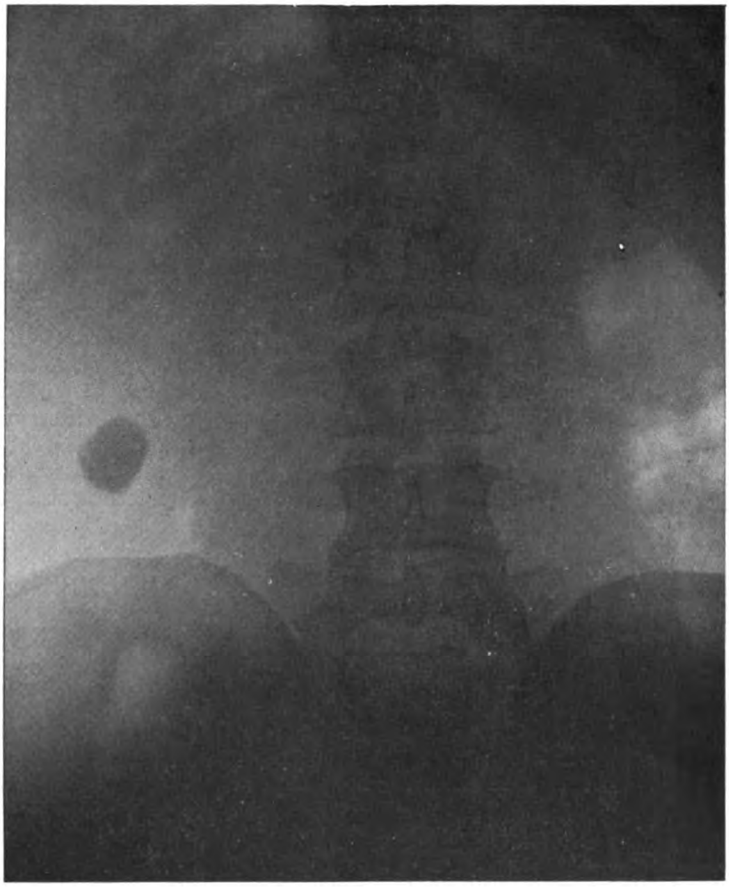


Fig. 133.—Skiagram of a calculus in a dropped left kidney.
(*By Dr. Alfred C. Jordan.*)

Renal Tuberculosis, apart from the miliary form of children, is not uncommon as a primary disease of one kidney. The patients affected are usually young adults, who complain of a constant aching in one loin, with occasional attacks of more acute pain resembling renal colic. At the onset of the disease, when the foci are limited to the renal tissues, there is no change in the urine beyond the occasional presence of albumin ; but as it advances the foci coalesce and form a softened area which opens into the renal pelvis, when there is a constant discharge of small quantities of pus and blood in the urine. The liberation of tuberculous material into the renal pelvis and ureter causes infection of the mucous lining of these passages, and is marked almost constantly by increased frequency of micturition during both day and night, even before any tuberculous infection has occurred in the bladder. These cases are often mistaken for renal calculus, but in any case of persistent slight hæmaturia or pyuria a careful search should be made for tubercle bacilli in the urine. It should be noted also that a skiagram may show a distinct shadow produced by a tuberculous focus in the kidney (*Fig.* 134), but its outline generally differs from that due to a calculus in its less definite border. In renal tuberculosis the hæmaturia is rarely increased by exertion on the part of the patient, as is frequently the case with calculus, and pain in the loin is less mitigated by rest in bed. In renal tuberculosis the lower end of the ureter of the affected side may often be felt to be thickened on examination per vaginam, or per rectum, whilst in the male tuberculous nodules may be felt in the prostate or vesicles.

In ***Renal Mobility***, hæmaturia is certainly uncommon, but occurs occasionally. In the case of a patient with markedly increased renal mobility, hæmaturia may follow any exertion, such as hunting or dancing. Renal mobility is so common, however, that the occurrence of hæmaturia should in any case arouse suspicion of some other lesion of the urinary tract, and a thorough examination both of the urine especially for tubercle bacilli, and of the bladder (by the cystoscope) and of the kidney, should be made before any attempt at fixation is undertaken. Movable kidney may be entirely painless and give rise to no symptoms whatever, or may cause lumbar aching or attacks of acute pain resembling renal colic (Dietl's crises). It frequently causes gastro-intestinal disturbance from the drag upon the duodenum in relation to it, and occasionally also polyuria and increased frequency of micturition. The kidney can be felt to be movable, but care must be taken not to mistake other abdominal swellings for a kidney (see KIDNEY, ENLARGEMENT OF, p. 352).

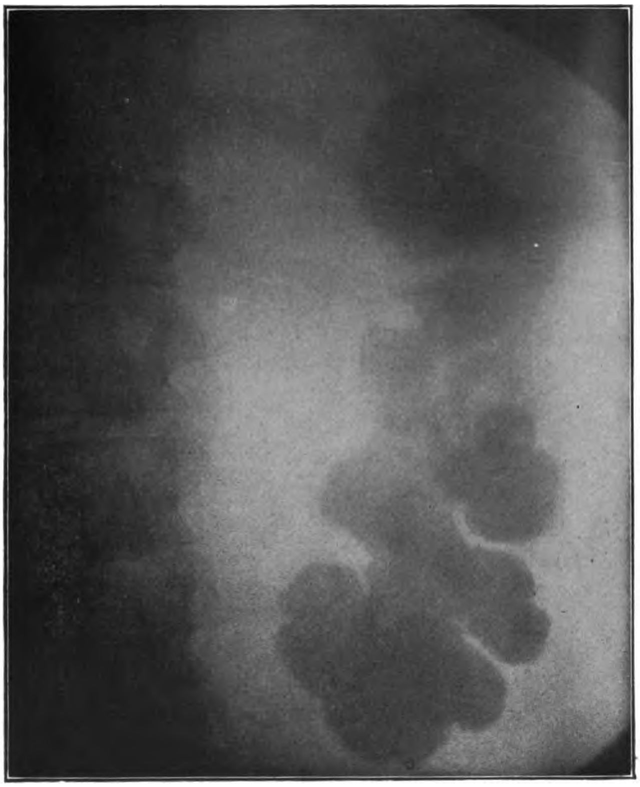


Fig. 134.—Skiagram in a case of tuberculous kidney with pyonephrosis ; the condition, due to marked deposit of calcium salts in the old tuberculous lesions, might be mistaken for a large antlered calculus.
(*Skiagram by Dr. C. Thurstan Holland.*)

Hydronephrosis occasionally gives rise to hæmaturia, and the combination of renal tumour and hæmaturia would suggest a growth in the kidney. The blood from a hydronephrotic kidney, however, is very rarely copious, and the other symptoms of hydronephrosis would distinguish the two, in particular intermittency with corresponding changes in the amount of the urine.

Polycystic disease of the kidneys is commonly accompanied by hæmaturia in the later stages of the disease. It occurs in early childhood or in adult life, and is most commonly bilateral, forming an enlargement of each kidney which may reach large dimensions, although on the other hand a tumour may only be felt on one side. In the early stages the diagnosis is difficult ; but later, pain, bilateral tumour, hæmaturia, and signs of renal inefficiency will be present. The renal tumour caused by polycystic disease is smooth and rounded, but differs from hydronephrosis in that fluctuation can seldom be obtained. Bilateral hydronephrosis will be diagnosed from polycystic disease by the finding of some lesion obstructing the normal urinary flow, such as stricture of the urethra, prostatic or vesical disease, or carcinoma of the pelvic organs invading the ureters.

Oxaluria (p. 423) may give rise to slight hæmaturia. The passage of large numbers of oxalate crystals in the urine occurs in some patients, especially after a diet containing rhubarb, gooseberries, or tomatoes, and is often accompanied by dyspepsia. An examination of the urine on successive days will demonstrate the condition. The aching in one loin, and the presence of envelope crystals in the urine, may simulate renal stone, but the absence of a shadow in a skiagram will disprove the latter.

Acute Nephritis is accompanied by hæmaturia, but is usually obvious by the sudden onset of the disease, by the history of some specific fever, or of a chill, and by the subcutaneous œdema. The urine is scanty and of high specific gravity, and contains, in addition to blood discs, hyaline and epithelial tube-casts, many renal epithelial cells, and abundant albumin. There are some cases of acute nephritis in which no œdema occurs, and then the abundance of renal tube-casts in the urine affords the main evidence as to the diagnosis.

Essential Renal Hæmaturia is the name given to a group of cases in which definite unilateral hæmaturia is present, but in which examination of the kidney on exploration has failed to show the cause of the hæmorrhage. The bleeding is profuse and comes on suddenly without any apparent cause ; it is intermittent and may be accompanied by lumbar aching, but there is no tenderness and enlargement of the kidney, and on cystoscopic examination it is proved to be unilateral. In the intervals of hæmaturia there may be no albuminuria. The kidney on exploration appears to be normal, but if a piece is removed for microscopic section, evidence of nephritis will usually be found. The evidence tends to show that these cases are probably due to a unilateral nephritis.

B. **Ureteric Calculus** may cause hæmaturia, either during the descent of the stone or when the latter becomes arrested in the duct without causing complete obstruction to the flow of urine. The diagnosis is usually easy from the history, and the character of the pain, accompanied by the increased desire to micturate ; but in some cases on the right side it may be mistaken for acute appendicitis. The previous history of the passage of a calculus or of renal symptoms of stone will usually be elicited. A skiagram should be obtained (*Fig.* 192, p. 455).

C. **Vesical Causes.**—The profuse hæmaturia of a *papilloma, villous tumour*, or of a *villus-covered carcinoma* of the bladder frequently occurs without any other symptom, coming on suddenly without any exciting cause ; it may last a variable time, and then disappear entirely, or continue as a slight hæmaturia for some days. With the carcinomatous form there may be some increased frequency of micturition in the absence of bleeding, but in either variety the clotting of blood in the bladder may cause urgent desire to micturate or even retention of urine. A rectal examination may give evidence of infiltration of the base of the bladder or of the pelvic lymphatics in the malignant form, but it is only rarely that an innocent tumour is large enough to be felt per rectum. In the intervals between hæmorrhages, a cystoscopic examination will demonstrate the presence of a vesical growth (*Plate XVI, Figs. F* and *G*). It should be noted that the common situation for a vesical tumour is at the base of the bladder, in close proximity to a ureteric orifice ; the latter may be obstructed, or dragged upon by the growth in such manner as to cause renal distention or hydronephrosis, so that a vesical tumour may give rise to renal pain and tumour, and in this way be mistaken for a renal growth. This difficulty will be overcome by a cystoscopic examination of the bladder.

Prostatic enlargement of the adenomatous, or more frequently of the carcinomatous variety, may cause hæmaturia. The age of the patient (54 or more), the increased frequency and difficulty in micturition, the evidence obtained by rectal examination and by catheterization, suffice to diagnose the disease. The hæmaturia of prostatic enlargement is often profuse, and may occur early in the disease ; but on careful inquiry it will usually be found that there has been for some months a gradually increasing frequency of micturition.

Vesical Epithelioma occurs in elderly patients, and causes slight but fairly constant hæmaturia. For hæmorrhage to take place from a vesical epithelioma there must be ulceration of the surface of the growth, and other symptoms will be present, namely, increased frequency of micturition both day and night, penile pain following the act of micturition, and pyuria. The blood often occurs as a few drops at the termination of urination, or may be mixed throughout the act. Usually a vesical epithelioma is situated on the base of the bladder, and may be felt as a distinct infiltration per rectum.

Vesical Tuberculosis gives rise to exactly the same symptoms as an epithelioma, but it occurs commonly in young adults. Persistent slight hæmaturia and pyuria in a young patient will always suggest tuberculous disease, and a very careful search should be made in the centrifugalized urine for tubercle bacilli, whilst other evidence of tuberculous disease, especially in the testes, vesiculæ seminales, and prostate, should be looked for. Difficulty may arise in the diagnosis between vesical and renal tubercle, for in the latter persistent hæmaturia and pyuria, together with increased frequency of micturition, may be present before the bladder becomes infected. With renal tubercle some renal enlargement and pain will usually be found, the ureter may be felt per rectum to be thickened, and the blood in the urine will not be more apparent at the end than during the rest of micturition, unless the bladder is also affected. When tuberculosis attacks the urinary organs, it is much more common as a primary disease in the kidney than in the bladder, but the infection finally spreads by the ureter to the bladder as soon as a renal focus discharges into the pelvis of the kidney, and may at the same time attack the prostate or seminal vesicles. When a tuberculous nodule in a vesicle or the prostate ulcerates into the bladder, a sharp attack of hæmaturia may result. Great assistance may be gained in the diagnosis of urinary tuberculosis by a careful cystoscopic examination (*Plate XV*, *Fig. E*), and by rectal examination.

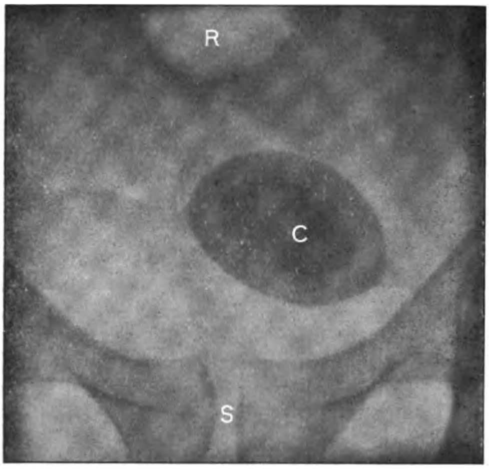


Fig. 135.—Skiagram of a composite (oxalate and phosphate) vesical calculus. C, calculus; R, rectum; S, symphysis pubis. (*By Dr. Alfred C. Jordan.*)

Vesical Calculus also causes slight hæmaturia, usually as a few drops in the terminal urine. The subject of a calculus in the bladder unaccompanied by cystitis will complain of increased frequency of micturition during the day or during exercise, but is usually free from micturition during the night. There is pain of a pricking character in the glans penis after micturition, and there may be a history of sudden stoppage of the stream during the act. The patients are usually men, and there may be a history of previous calculi in the bladder or of attacks of renal colic with the descent of a renal calculus which has not been passed per urethram, but which has increased in size since it entered the bladder. The stone may be felt with a sound, or better still, seen by a cystoscope, when small calculi which may be missed with a sound may be diagnosed with certainty (*Plate XVI*, *Fig. H*). The *x*-rays are also useful in detecting the stone in many cases (*Fig.* 135). If the calculus has caused cystitis, there will be in addition pyuria and nocturnal micturition.

Acute Cystitis is accompanied by hæmaturia ; but the other symptoms, such as vesical tenesmus, suprapubic pain, and pyrexia, together with pyuria and a cause for the condition, will point to the disease.

Bilharzia Hæmatobia causes slight hæmaturia, and gives rise to symptoms very similar to vesical tuberculosis. The discovery of the typical ova in the urine (see *Fig.* 26, p. 79), together with a history of residence in an affected district, notably Egypt or certain parts of South Africa, will make the diagnosis clear. The cystoscopic appearance in the bladder of small, glistening yellow nodules and small areas of raised granulation tissue, is distinctive of the disease (*Plate XVI*, *Fig. K*).

D. **Urethral Causes.**

Acute Urethritis, whether gonococcal or septic, may cause blood to appear in the urine from the acute congestion of the urethral mucous membrane. The history and the presence of an acute urethral discharge (p. 181) make the diagnosis evident.

The *Impaction of a Calculus* in the urethra causes some bleeding from direct injury to the urethral mucous membrane. There is usually retention of urine, so that true hæmaturia may not occur ; but the history of sudden stoppage of the stream of urine during micturition, with acute penile pain, together with the previous history of renal or vesical

PLATE XV

BLADDER APPEARANCES SEEN THROUGH THE CYSTOSCOPE

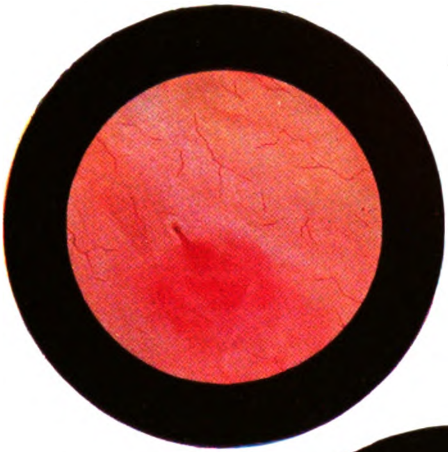


FIG. A.

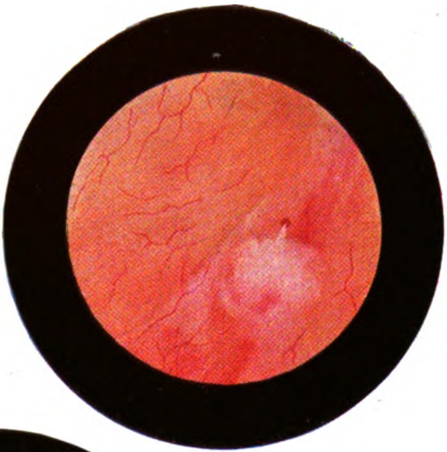


FIG. B.

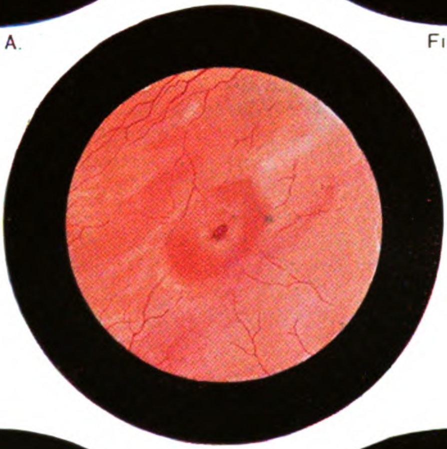


FIG. C.

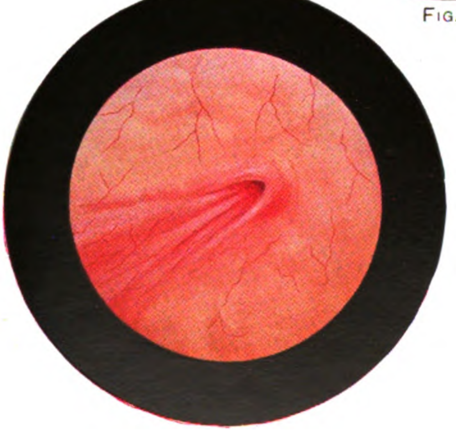


FIG. D

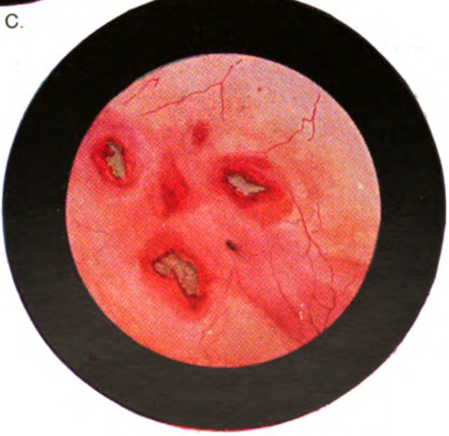


FIG. E.

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Fig. A.—Blood-stained urine issuing from the ureter.
Fig. B.—Purulent urine issuing from the ureter.
Fig. C.—Congestion around a ureteric orifice in calculous pyelitis.

Fig. D.—The retracted ureter common with descending renal tuberculosis.
Fig. E.—Tuberculous ulceration around the ureteric orifice in descending renal tuberculosis.

(*Figs. D and E are from sketches kindly supplied by Dr. C. F. Walters, of Clifton.*)

stone, will usually make the diagnosis clear. It is not uncommon in male children. The calculus may be felt from the outside in the course of the urethra, often at or near the meatus, or seen by an endoscopic examination.

Nævus of the urethral mucous membrane is a rare but important cause of severe and recurrent hæmaturia, the patient generally presenting no other symptoms beyond the spontaneous bleeding and serious anæmia resulting from it. The blood is passed both with and apart from micturition. There may or may not be bleeding nævi elsewhere ; but the condition is precisely analogous to the small bleeding nævi of the tongue and mouth that have been described in conjunction with nævi of the skin by Osler and others. The diagnosis of a urethral nævus could scarcely be made with certainty except by urethroscopy.

II. HÆMATURIA FROM DISEASE OF THE NEIGHBOURING VISCERA INVOLVING THE URINARY ORGANS.

The direct spread of *carcinoma* of the pelvic organs may in its progress involve the bladder, as is not uncommon in the later stages of carcinoma of the uterus, vagina, rectum, or pelvic colon. The infiltration of the bladder wall before actual ulceration has occurred is usually indicated by vesical irritability, followed by ulceration and hæmaturia, together with the passage of urine by the vagina or fæcal matter in the urine. Occurring as a late stage of carcinomatous disease, there is usually little difficulty in the diagnosis.

Hæmaturia may occur during an attack of *acute appendicitis* from the direct spread of the inflammatory process to the vesical wall. In some cases in which the inflamed appendix turns downwards over the pelvic brim, it may become adherent to the bladder, or an abscess may form in immediate relation to the bladder wall. The localized inflammation of the vesical mucous membrane causes hæmaturia, whilst the sudden appearance of a quantiy of pus in the urine has been noticed when an appendicular abscess has ruptured into the bladder. The history of acute pain low down in the right iliac fossa, the pyrexia, and general symptoms of peritoneal inflammation before any urinary symptom was noted, will point to the disease ; a rectal examination may reveal the inflammatory process in the right pelvic region.

Acute Salpingitis or *Pelvic Abscess* may similarly cause hæmaturia from direct inflammatory extension to the vesical wall, but this is rarer than in appendicitis.

Tuberculous and *Dysenteric Ulceration of the Intestine* have both caused hæmaturia by the adhesion of the bowel to the fundus of the bladder and the subsequent inflammatory condition of the mucous membrane. In a case of slight hæmaturia, a cystoscopic examination showed a localized area of intense congestion at the fundus of the bladder without any other vesical lesion, and on opening the abdomen, a coil of small intestine, obviously ulcerated by tubercle, was found adherent to the peritoneal aspect of the bladder. In most cases the symptoms due to the intestinal disease would be apparent.

III. HÆMATURIA IN GENERAL DISEASES.

The sudden plugging of a renal vessel by embolism (*renal infarction*) is not uncommon in cases of endocarditis, and may be accompanied by hæmaturia. The embolism is seen most commonly in infective endocarditis ; it is indicated by sudden pain in the loin, followed by hæmaturia. The occurrence of acute endocarditis in the course of acute septic processes, such as acute osteomyelitis, pneumonia, or acute rheumatism, is not uncommon, and will usually be diagnosed before there is any evidence of renal embolism. On the other hand, there are certain cases of chronic heart disease in which the first evidence of infected endocarditis having become superadded may be the occurrence of sudden hæmaturia ; and in some such cases there may be difficulty in excluding acute Bright's disease, because around each infarct there is local acute inflammation, and therefore the urine will contain tube-casts as well as blood ; the other signs of infective endocarditis (p. 34) should be watched for.

Leukæmia may be accompanied by hæmaturia ; but the enlargement of the spleen, general symptoms of anæmia, and the total and differential blood counts (p. 21) will point to the diagnosis.

Scurvy and the various forms of PURPURA (p. 552) may each be accompanied by hæmaturia, but the general symptoms of each disease are usually well marked before hæmaturia occurs. *R. H. Jocelyn Swan.*

HÆMOGLOBINURIA differs from hæmaturia in that the blood pigment is passed in solution in the urine apart from red corpuscles; small numbers of red corpuscles, or their ghosts, may be found microscopically, but these constitute hæmaturia in association with the hæmoglobinuria; the essential part of the latter is the passage of the blood pigment dissolved out of the red corpuscles. It gives the same chemical tests as ordinary blood; spectroscopically it is almost as common to find the bands of methæmoglobin (*Fig.* 35, p. 80) as those of oxyhæmoglobin (*Fig.* 30, p. 80); by the addition of ammonium sulphide the spectrum is changed to that of reduced hæmoglobin (*Fig.* 31, p. 80), and by the further addition of a few drops of concentrated caustic soda, that of alkaline hæmatin (*Fig.* 33, p. 80) is produced. The diagnosis depends upon the discovery of blood pigments in the urine, whilst the microscope shows no red corpuscles, or so few as to be out of all proportion to the pigment. It is important that the urine should be examined fresh, for otherwise, owing to the disintegration of red cells after they have been passed as such, it is possible to mistake for hæmoglobinuria that which is really hæmaturia. To the naked eye the urine may be only just tinged with a colour that suggests blood pigment, or it may be absolutely blood red, brown, murky, or even black, as in tropical blackwater fever. It is seldom clear; but clouded by mucus, casts, amorphous masses of pigment, and débris.

Hæmoglobinuria results from any condition which leads to hæmoglobinæmia by laking the red corpuscles within the living vessels. It has been produced in animals experimentally by the injection of various hæmolytic sera and other substances. It may occur in man as the result of the oral administration of certain chemical substances, such as potassium chlorate, phenylhydrazine, turpentine, ether, carbon bisulphide, pyrogallic acid, naphthol, carbolic, hydrochloric, sulphuric, nitric, oxalic and chromic acids, glycerin, chloroform, sulphonal, veronal, trional, tannin, saponin, strychnine, urotropine, and possibly quinine; after the inhalation of certain toxic gases, notably carbon monoxide, carbon bisulphide, naphtha vapour, arseniuretted, antimoniuretted or sulphuretted hydrogen; after the transfusion of certain foreign sera, or after the introduction of such poisons as those of snakes, or venomous toads or spiders; from ricin, abrin, robin, crotin, phallin; after eating poisonous mushrooms, toadstools, or truffles; after frostbite and extreme exposure to cold; after severe burns; after large internal extravasations of blood, especially those within the abdominal cavity; in a few cases in which pregnancy is associated with toxæmic symptoms; in some new-born infants, occasionally in an obscure epidemic form; in association with certain functional disorders of the vasomotor system, especially Raynaud's disease, factitious urticaria, and angioneurotic œdema; after very long-sustained excessive physical exertions and fatigue; in association with severe forms of microbial—or presumably microbial—toxæmia, especially malaria and blackwater fever, and to a much less extent in severe syphilis, typhoid fever, scarlet fever, acute pyogenic septicæmia, generalized anthrax, yellow fever; Henoch's purpura; in certain cases of nephritis; and in that remarkable affection known as paroxysmal hæmoglobinuria.

Although the above list may appear formidable, the differential diagnosis between the different diseases mentioned will seldom depend solely upon the presence or absence of hæmoglobinuria. The chief importance of the latter, indeed, lies first in the necessity of not mistaking it for hæmaturia, and secondly in that its occurrence is a sign that considerable hæmolysis is taking place and that the prognosis is proportionately less good. It is enough if the fact that it may be a complication of any of the above conditions is borne in mind.

The question of whether *blackwater fever* is due to the effects of quinine in a patient whose blood is already susceptible to hæmolysis on account of malaria, or whether the blackwater is due to a distinct and specific malady, has not yet been settled; the diagnosis is, however, generally obvious, the geographical circumstances under which the disease develops pointing to its nature.

Paroxysmal hæmoglobinuria is rare; but in Great Britain it is probably the commonest cause of considerable hæmoglobinuria without symptoms of extreme illness. It may affect adolescents or grown-up people, males or females; it has probably several different ultimate causes; amongst the latter, however, previous syphilis stands out pre-eminently, and probably heredity is also a factor. Males are affected rather than females. The remarkable feature of the malady is the way in which an attack can be brought on, almost at will, by certain immediate causes, of which the most potent is exposure to cold, others being

PLATE XVI

BLADDER APPEARANCES SEEN THROUGH THE CYSTOSCOPE

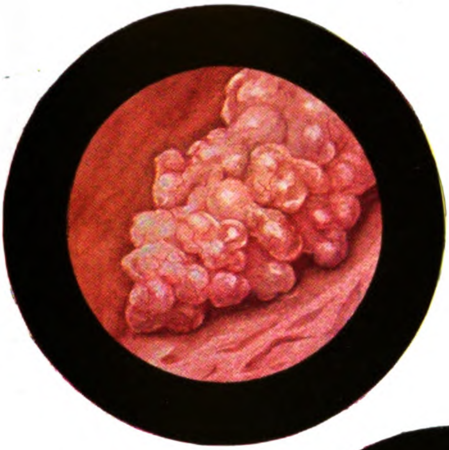

FIG. F.

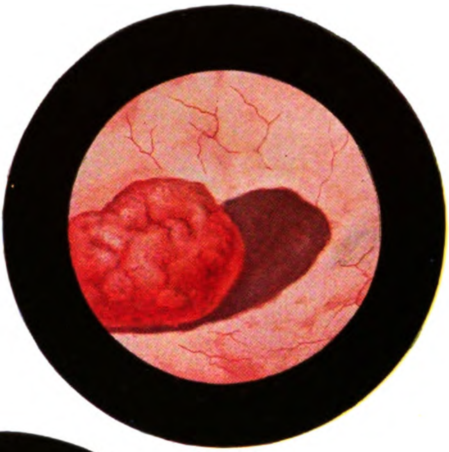

FIG. G

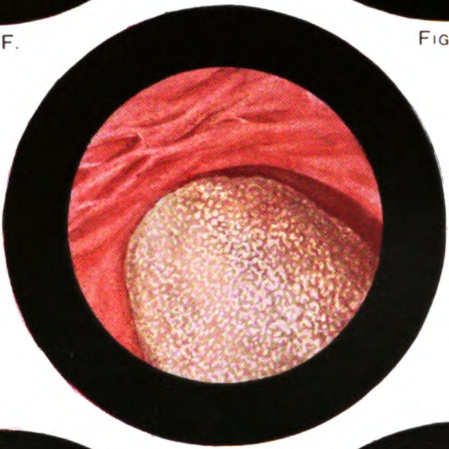

FIG. H.

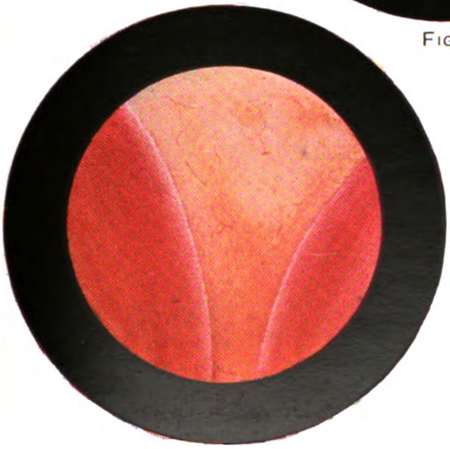

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FIG. I

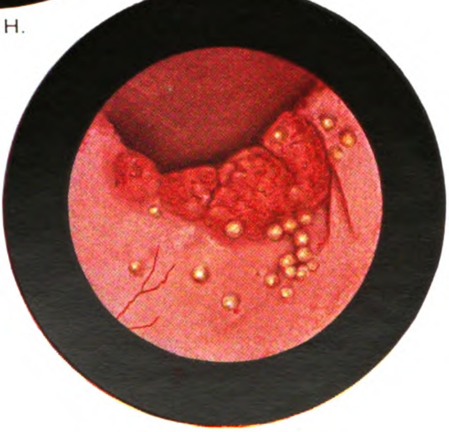


FIG. K.

W. Thornton Shiells, del.

Fig. F.—Pedunculated carcinoma of the bladder.
Fig. G.—Pedunculated bald carcinoma of the bladder.
Fig. H.—Uric acid calculus in the bladder.
Fig. I.—Appearance at the urethral orifice in bilateral adenomatous enlargement of the prostate.
Fig. K.—Bilharzia hæmatobia.

(*Fig. G is from a sketch by Dr. Walters, and Fig. K is reproduced by kind permission of Mr. H. A. Wilson.*)

excessive exercise or mental excitement. Sometimes the exposure has to be considerable before hæmoglobinuria results ; on the other hand, it may be impossible for the patient to keep his hands immersed in cold water for any length of time without an attack ensuing. The urine may look like blood, and the output of pigment, together with considerable albuminuria, persists for a day or two as a rule ; the attack may be unaccompanied by other symptoms, but sometimes there is a shivering attack or an actual rigor with rise of temperature, and a general feeling of illness, necessitating rest in bed. Sooner or later, if repeated attacks occur, the patient becomes severely anæmic, with all the symptoms that result from such anæmia. The diagnosis may be very difficult at the time of the first attack, but it is relatively easy when the attacks recur, especially when there is distinct relationship to some definite immediate cause, such as exposure to cold, to undue fatigue, or mental excitement. The main mistake to avoid is a diagnosis of hæmaturia, such as a villous tumour of the bladder might cause. The way to obviate this error is to employ both the microscopic and the spectroscopic tests for blood, much pigment and few corpuscles pointing to hæmoglobinuria. If there is still doubt as to whether the patient has hæmoglobinuria or hæmaturia, a little of his blood serum, obtained by venepuncture, may be examined in the laboratory for Eason's reaction, a complex serum test which is positive in essential hæmoglobinuric cases, negative in others ; the Wassermann reaction would probably be tested at the same time to determine whether the cause of the symptom was syphilis or not.

Herbert French.

HÆMOPTYSIS literally means blood-spitting, but clinically it is restricted to expectoration of blood derived from the lungs, bronchi, or trachea, to the exclusion of blood from the mouth, nose, or pharynx. Some include blood coming from ulceration of the larynx under the heading of hæmoptysis ; others do not, so that the meaning of the term is arbitrary ; for practical purposes it is simpler to include the larynx as a source for hæmoptysis.

The differential diagnosis resolves itself into two main portions, namely, (I) A determination of whether the symptom has really been hæmoptysis in the restricted sense, or whether the blood has been derived from the mouth, nose, or pharynx on the one hand, or the stomach on the other ; and (II) If true hæmoptysis has really occurred, a determination of its exact cause in the particular case.

I. THE DISTINCTION BETWEEN TRUE AND SPURIOUS HÆMOPTYSIS.

True hæmoptysis—that is to say, hæmorrhage from the lungs, bronchi, trachea, or larynx—can sometimes be distinguished at once from the spitting of blood derived from the nose, mouth, or pharynx. The occurrence of epistaxis, bleeding gums, sore throat, ulcerative stomatitis, epithelioma linguæ, injury to the mouth, gingivitis from a carious tooth, or from pyorrhœa alveolaris, pharyngitis, septic conditions of the antrum of Highmore, or frontal, ethmoidal, or sphenoidal air-cells, or rarer conditions, such as lupus of the palate or pharynx, sarcoma of the tonsil or of the basisphenoid, may generally be detected by a careful examination of the nose, mouth, gums, and pharynx ; moreover the blood in these conditions is usually mixed with saliva, and watery. It is important, however, to be decidedly guarded in concluding that blood comes from the mouth, nose, or throat, and not from the lungs ; and a careful examination for tubercle bacilli should be carried out in every such case, lest the early stage of phthisis be missed.

The distinction between hæmoptysis and hæmatemesis is often easy, but sometimes very difficult. The history may help, or the patient's own sensations may make him certain that he coughed up the blood, and did not vomit it. The following is a summary of the points of distinction :—

HÆMOPTYSIS.	HÆMATEMESIS.
1. The patient *coughs* the blood up	1. The blood is *vomited*
2. Part of the blood is often frothy	2. The blood is not frothy
3. The blood may occur by itself, but it is often mixed with sputa, recognizable microscopically	3. The blood may occur by itself, but it is often mixed with vomit, recognizable by the presence of food particles
4. The blood is alkaline in reaction	4. The blood may be alkaline if it is abundant, but it is often acid from admixture with gastric juice

Hæmoptysis, *continued.*	Hæmatemesis, *continued.*
5. Tubercle bacilli or elastic fibres may be detected	5. Tubercle bacilli will be absent
6. There may be a previous history of acute rheumatism, or of cough and night sweats, indicative of heart or lung disease, confirmed by abnormal cardiac or pulmonary physical signs	6. There may be a definite history, with or without physical signs, pointing to a gastric lesion or to cirrhosis of the liver
7. Before the blood is coughed up, there is often a sense of tickling or gurgling in the throat, always suggestive of true hæmoptysis	7. Before the blood is brought up there may be a feeling of sickness, nausea, oppression in the epigastrium, faintness, and giddiness
8. The motions are not altered afterwards unless the blood has been abundant and much of it has been swallowed, when they may be tarry as in hæmatemesis	8. The motions are often tarry afterwards
9. Blood-stained sputa may be expectorated for several days after a severe attack	9. There are usually no sputa
10. A history of cough	10. A history of abdominal pains after food

Notwithstanding all these points of distinction, however, one may be misled unless the patient can be kept under observation for a time; moreover, hæmatemesis may be caused by hæmoptysis, especially when the bleeding takes place in the night, the blood being swallowed as soon as it gets into the pharynx whilst the patient remains asleep and quite unconscious of the occurrence. The frequency with which hæmoptysis occurs during the night when the patient is at rest is remarkable; but in the majority of instances the incidence of bleeding excites coughing, and the patient wakes.

Malingering by the production of blood-spitting by gum-sucking is diagnosed upon circumstantial evidence. It has sometimes happened that a patient has produced the blood of fowls with the statement that this has been coughed up—a fallacy that can be detected by examining the red cells under the microscope.

Redness of the sputum is not always proof that the colour is due to blood; the presence of red cells should be verified under the microscope, and the guaiacum and ozonic ether test also applied; occasionally patients have been regarded as suffering from recurrent phthisical hæmoptysis when in reality the redness of the sputum has been due to infection of the respiratory passages by relatively unimportant pigment-producing micro-organisms, generally of the *Bacillus prodigiosus* type; this source of fallacy is to be avoided by having careful bacteriological examinations of the sputum made in all cases that are not perfectly straightforward.

II. DETERMINATION OF THE CAUSE OF THE HÆMOPTYSIS.

Having arrived at the conclusion that a patient has had hæmoptysis, the next point is to ascertain its cause. By far the commonest causes of hæmoptysis are *phthisis* and *mitral stenosis.* The heart and lungs need particular examination therefore, and the family and personal history, both as to acute rheumatism or chorea, and as to consumption, may assist. If there are no abnormal physical signs in the thorax, it does not follow that phthisis is absent—even phthisis with cavitation may exist without any definitely abnormal physical signs being detected; microscopical examination of the sputum, therefore, both for tubercle bacilli and for elastic fibres, should never be omitted, especially after the hæmoptysis has ceased; repeated examinations may be required if the first proves negative.

Although these are the commonest, there are a great many other causes, as the following tables indicate:—

A. **Hæmoptysis due to Changes in the Lungs**:—

1. Phthisis: (*a*) Early; (*b*) Later
2. Cirrhosis of lung: pneumonoconiosis:
    - (*a*) Knife-grinder's lung
    - (*b*) Stonemason's lung
3. Cardiac disease, especially mitral stenosis
4. Violent coughing efforts, as in whooping-cough or bronchitis
5. Injury to the chest:
    - (*a*) Blows upon the chest wall
    - (*b*) Fractured rib
    - (*c*) Exploratory needling
    - (*d*) At the end of paracentesis thoracis
6. Lobar pneumonia
7. Bronchopneumonia
8. Septic pneumonia, with or without abscess

A. Hæmoptysis due to Changes in the Lungs, *continued*:—

9. Gangrene of the lung
10. Infarction of the lung:
    (*a*) Embolic; (*b*) Thrombotic
11. Neoplasm of the lung, whether primary or secondary:
    (*a*) Sarcoma; (*b*) Carcinoma
12. Sporotrichosis of the lung:
    (*a*) Aspergillosis;
    (*b*) Actinomycosis
    (*c*) Other forms
13. Aortic aneurysm pressing on and opening into the lung
14. Empyema bursting through the lung
15. Hepatic abscess bursting through the diaphragm into the lung
16. Hydatid cyst
17. Primary atheroma of the pulmonary arterioles

B. **Hæmoptysis due to Changes in the Bronchioles, Bronchi, or Trachea**:—

1. Bronchitis:
    (*a*) Acute; (*b*) Chronic; (*c*) Plastic
2. Bronchorrhœa
3. Bronchiectasis
4. Aortic aneurysm opening into the trachea or a bronchus
5. Ulceration of the trachea or a bronchus:
    (*a*) Tertiary syphilitic
    (*b*) Malignant
        (i) Primary epithelioma of bronchus
        (ii) Invasion of a bronchus by a mediastinal sarcoma, lymphosarcoma, œsophageal epithelioma, or other neoplasm.
    (*c*) Secondary to a foreign body, such as a button, a fruit-stone, a tooth, etc.; or to a tracheotomy tube
    (*d*) Secondary to a caseous or calcareous bronchial gland
6. Parasitic infection by *Distoma pulmonale westermanni*

C. **Hæmoptysis due to Changes in the Larynx**:—

1. Acute laryngitis
2. Tuberculous ulceration
3. Syphilitic ulceration
4. Malignant ulceration:
    (*a*) Epitheliomatous
    (*b*) Sarcomatous
5. Post-typhoidal ulceration
6. Post-diphtheritic ulceration
7. Injury to the larynx, by a blow, a throat grip, a cut thoat, intubation, or operation
8. Lupus of the larynx
9. Variolous ulceration
10. Leprosy of the larynx
11. Angioma of the larynx

D. **Hæmoptysis due to Changes in the Blood**:—

1. Purpura and its various causes (p. 552)
2. Scurvy
3. Splenomedullary leukæmia
4. Lymphatic leukæmia
5. Pernicious anæmia
6. Lymphadenoma
7. Malignant types of specific fevers, such as variola or measles
8. Hæmophilia

E. **Doubtful Causes of Hæmoptysis**:—

1. Granular kidney
2. Arteriosclerosis
3. Vicarious menstruation
4. Recurrent hæmoptysis in arthritic subjects (Andrew Clark)
5. Hæmoptysis in apparently sound and healthy young subjects

Copious Hæmoptysis has only two causes, namely, *rupture of an aortic aneurysm* into trachea, bronchus, or lung; and *rupture of an aneurysm of a pulmonary arteriole in a lung cavity or phthisical vomica.* The former, when once it causes severe hæmoptysis, nearly always proves immediately fatal; the latter may also cause rapid death, but sometimes the severe bleeding stops, and recovery may ensue. In either case, however, there is often a stage of slight or premonitory bleeding for days, weeks, or even months before the final rupture occurs.

There are some causes of hæmoptysis in the above list about which little need be said. The whole of *Group E*, for instance, is open to much doubt; it is true that apparently sound young subjects may have transient hæmoptysis and never develop phthisis; on the other hand a certain proportion of such cases do become consumptive later, so that the presumption is that in all of them the hæmoptysis really has a tuberculous origin, cure resulting rapidly in some, but not in others. Particular care should be taken in the examination of the sputum and of the chest by the ordinary physical methods, and perhaps by the *x*-rays also, and even although the cause of the hæmoptysis may not be determined precisely, the patient would be well advised to live as healthily as possible, lest a further stage of phthisis develop. The same applies to so-called vicarious menstruation; and in not a few cases in which the hæmoptysis has been attributed to the arthritic diathesis, to arteriosclerosis, or to renal lesions, the cause may really be an intercurrent infection of the lung by tubercle bacilli even in middle-aged or elderly people.

Causes in *Group D* seldom give rise to extensive true hæmoptysis, though there may be much epistaxis, bleeding from the gums, and so on. The diagnosis between the different conditions in this group will be found elsewhere.

Phthisis is by far the commonest cause of hæmoptysis. It may be the very first sign of the disease, it may be the last, or it may occur at any intermediate stage. The amount of blood brought up is very variable ; the sputum may be only streaked, or a pint or more may stream from the mouth. In advanced stages the diagnosis is not difficult. There is the history of cough, loss of appetite and weight, night sweating, and expectoration ; there are the wasting and flattening of the chest wall, especially above and below the clavicles, often more on one side than the other ; the deficient movement on respiration, the unequal tactile vocal fremitus, the impairment of note, over one upper lobe more than over the other, with the bronchial breathing, consonating râles, bronchophony and pectoriloquy at one apex, with signs of similar but less advanced disease at the other. Detection of pus cells, tubercle bacilli, and perhaps elastic fibres in the sputum, is conclusive. Hæmoptysis may, however, be the earliest evidence of phthisis ; the diagnosis is then difficult, for the physical examination may not reveal any abnormal signs. Particular stress may be laid upon greater prominence of one clavicle than of the other, prolongation of the expiration, and the constant presence of one or more apical clicks, or râles, perhaps brought out only on coughing. In some cases the mottled shadows seen with the *x*-rays may assist the diagnosis (*Fig.* 41, p. 103), although, taken by themselves, they may be misleading ; tubercle bacilli may be found in the sputa quite early, so that a careful examination even of the most insignificant amount of sputum must always be made before a definite opinion as to the cause of the hæmoptysis can be given. In the early stages of phthisis hæmoptysis results from local inflammatory hyperæmia with rupture of capillaries ; the amount of blood expectorated is then usually small, and it may amount only to streaking of the sputum. A little later, small vessels may themselves become inflamed and softened, or directly invaded by the tuberculous process, consequently rupturing if any extra strain is suddenly put upon them, —for instance, during attacks of coughing. This may lead to a more profuse hæmoptysis even quite early in the disease. When the malady is more advanced, caseation and breaking down of lung tissue may lead to softening of the external wall of a considerable branch of the pulmonary artery, resulting in an aneurysmal bulge, which, if thrombosis does not occur within it, will sooner or later rupture, and cause a profuse and possibly fatal hæmorrhage.

Cirrhosis of the Lung—Pneumonoconiosis, Miners' Phthisis—is a particular variety of fibrosis due to the inhalation of irritating particles, especially amongst workers at certain occupations. Coal miners seldom get it ; although their lungs become packed with carbon —*anthracosis*—these particles do not seem to inflame the tissues. Knife-grinders suffer from it—*siderosis* ; so do workers in certain limestone quarries, rock-drilling gold mines, and diamond mines—*silicosis*. The chief point in the diagnosis is the history as to occupation ; there is much doubt as to whether these conditions are not really of a chronic tuberculous nature, and tubercle bacilli should be looked for in all these cases, whilst the blood should also be tested for Wassermann's reaction, because recent evidence points to syphilis being another important factor in many of these patients. The hæmoptysis is far less frequent and less abundant than it is in ordinary phthisis.

Mitral Stenosis is the second commonest cause of hæmoptysis. Other forms of heart disease seldom lead to it direct, though mitral regurgitation may do so occasionally, and so may aortic stenosis or regurgitation when they have caused secondary mitral regurgitation. *Congenital heart disease*, unlike the acquired forms, is so liable to lead to phthisis that any hæmoptysis associated with it would arouse suspicions of the latter. *Fungating endocarditis* may also cause hæmoptysis, but as the result rather of the septic state or of infarction than of the valvular lesion. Mitral stenosis is the chronic valvular heart disease *par excellence* to produce hæmoptysis, and it may do so either when there is complete compensation or when there is evidence of failure. When compensated, the right ventricle pumps blood into the lungs with vigour, and causes great rise of pressure in the pulmonary vessels because the blood cannot escape freely through the stenosed mitral orifice. This is indicated clinically by accentuation or reduplication of the second sound in the second left intercostal space close to the sternum. At the impulse, which is often not materially displaced, the first sound will have a slapping character, and it will generally be preceded by a shorter or longer presystolic rumbling bruit. The latter is so short sometimes that it may be overlooked, but there may be a history of chorea or rheumatism to assist the diagnosis, and the accentuated pulmonary second sound will arouse suspicion in other cases, particularly if the precordial impairment of resonance is increased upwards and to the right, but not much to the left. The result of the great rise of blood-pressure in the lungs is that capillaries rupture from time to time ; the resultant hæmoptysis alarms the patient, but it is really no sign of danger ; sometimes patients have this hæmoptysis whenever the heart is at its best, losing it again when failure threatens. Far different is it when blood-spitting occurs in failing cases of mitral stenosis ; it is then generally due to infarction or to pulmonary 'apoplexy.' The infarction is less often due to *embolism* from an ante-mortem clot in the right auricular appendix or other part of the right side of the heart than it is to *thrombosis,* which results as follows : atheromatous degeneration of the pulmonary arterioles is brought about by the greatly increased tension within them ; rupture of small branches of such degenerated pulmonary arterioles gives rise to 'apoplexies,' and the alteration in the tunica intima due to the atheroma, together with the deficient rate of blood-flow, strongly predispose to thrombosis and consequent infarction. An *embolic infarct* occurs suddenly, and causes acute pain in the corresponding part of the thorax, orthopnœa, increased cyanosis, dyspnœa, and hæmoptysis ; a *thrombotic infarct* arises gradually, and causes hæmoptysis without the other symptoms.

Violent Coughing efforts, as in whooping-cough, or emphysema and bronchitis, may cause such pressure of the frænum linguæ against the teeth as to abrade its surface and lead to the expectoration of blood-streaked salivary sputum—spurious hæmoptysis ; it is said that they can also produce true hæmoptysis ; this is possible, but before blood-spitting in any given case is attributed merely to violence of coughing, every care should first be taken to exclude both tubercle and heart disease.

Injury to the Chest is not an uncommon cause of blood-spitting. There need have been no fracture of a rib —a severe blow on the thorax sometimes suffices. The only difficulty in the diagnosis is to be sure that the injury is the sole cause, and that it has not merely been the final factor in producing hæmorrhage from a latent tuberculous focus or an aneurysm.

In **Lobar Pneumonia** the amount of blood expectorated is slight in the majority of cases; the sputum is thick, viscid, tenacious, and generally there is no more blood than will give it a rusty or russet-brown colour. It may, however, be bright red, and in a few cases copious enough to be in itself alarming. The difficulty then is to distinguish it from phthisis, or from lobar pneumonia superposed upon phthisis. The diagnosis is often obvious enough ; but sometimes, notwithstanding the acute onset, the continued fever, the high ratio of the respiration to the pulse-rate, the viscidity of the sputum, the presence of capsulated diplococci in it, the abnormal physical signs, and the absence of chlorides from the urine, serious doubt remains until the subsequent course of the case has been watched. When the x-rays can be utilized at the bedside, a skiagram may sometimes serve to differentiate between phthisis (*Fig.* 41, p. 103) and lobar pneumonia (*Fig.* 136).

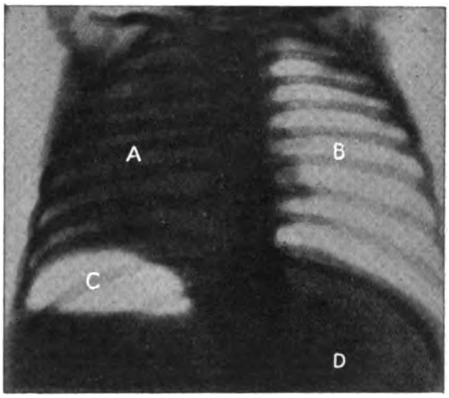


Fig. 136.—Skiagram of lobar pneumonic consolidation of left lung (A); normal right lung (B); gastric gas bubble (C); liver (D). (*By Dr. Alfred C. Jordan.*)

Bronchopneumonia is a rare cause of hæmoptysis, because the disease mainly affects children at an age when no spitting occurs. In older patients bronchopneumonia is generally either influenzal, or else due to the inhalation of septic particles from the mouth after operations under anæsthetics, or in association with such diseases as epithelioma of the tongue, or otitis media with lateral sinus thrombosis. Septic bronchopneumonia is diagnosed by reason of its being a lung complication of some other malady likely to give rise to it. Influenzal bronchopneumonia is apt to cause characteristic sticky râles at the bases, with less pyrexia but more asthenia than does lobar pneumonia ; and the minute *Bacilli influenzæ* may be found in the sputum in large numbers. If the signs are apical rather than basal, it will be difficult to be sure that the condition is not tuberculous, except by watching the case, and finding that rapid and compltee resolution and recovery ensue.

Gangrene of the Lung, due to whatever cause (p. 259), is characterized by the extreme stench of the breath and sputa. The only conditions which produce similar stench are fœtid decomposition of the retained sputum in bronchiectatic cavities or old phthisical vomicæ, or similar decomposition in the pus of an empyema which has ruptured through the lung, and which empties out its contents periodically. Gangrene of the lung can be differentiated from all these by the pulmonary elastic fibres to be found in the sputum, and by the history being shorter than would probably be the case with the others.

Infarction of the Lung, embolic and thrombotic, has already been mentioned in connection with heart disease, its most frequent cause. It only remains to add that it may also occur as the result of embolism secondary to thrombosis of systemic veins, infective endocarditis of the pulmonary or tricuspid valves, or from primary thrombosis in some blood diseases, such as leucocythæmia. A large embolus causes sudden death without hæmoptysis ; a smaller one may give rise to sudden acute pain in some part of the chest, and a local patch of crepitant râles with a pleuritic rub, and perhaps impairment of percussion note with bronchial breathing. Hæmoptysis associated with such physical signs and accompanied by evidence of endocarditis or venous thrombosis would suggest an infarct ; difficulty arises mainly when there is no obvious phlebitis in the case, when the vein affected is deep-seated—in the pelvis, for instance, after childbirth or some operation. The diagnosis is not so difficult when there have been repeated sudden acute pains in different parts of the chest, each followed by a little pyrexia and sometimes by hæmoptysis, due to repeated small emboli.

Carcinoma and **Sarcoma of the Lung** (*Fig.* 42, p. 105) are usually secondary. The diagnosis is sometimes obvious, sometimes very obscure indeed. The primary seat of the growth may be near the lung, for instance in a bronchus, the œsophagus, breast, or mediastinal glands ; or it may be distant, in the stomach, or a bone, and so on. The sputum may be merely blood-tinged, or it may be dark like red-currant jelly ; occasionally the hæmorrhage is profuse. A large number of cases of malignant disease in the lung are accompanied by pleuritic effusion, and unless the existence of a primary neoplasm elsewhere is known, growth may not at first be suspected. If aspiration is performed, the fluid is generally found to contain blood ; indeed, the discovery of blood-stained pleural fluid at a first tapping of a case that is not absolutely acute, is always very suggestive of neoplasm ; microscopically large cancer cells, with atypical mitosis, or even fragments of new growth, may be found either in the sputum or in the pleuritic exudate to clinch the diagnosis. Increasing varicosity of the veins on the chest wall, with reversal of the blood current in them, also points to intrathoracic growth obstructing the superior vena cava. The neoplasm may also stenose a bronchus, leading to unilateral deficiency of movement and tactile vocal fremitus, impairment of note, and deficient or absent breath-sounds, with or without faint bronchial breathing and crackling râles ; whilst, accompanying these physical signs, no tubercle bacilli would be found in the sputum, and yet the weakness and emaciation would be progressive.

Sporotrichoses of the Lung are being recognized with increasing frequency. Hitherto they have generally been mistaken for phthisis. They are due to various moulds of the nature of *Actinomyces*, *Aspergillus niger*, and others, and the diagnosis depends upon bacteriological investigations of the sputum by cultural methods. When no tubercle bacilli can be detected on repeated examination in the ordinary way, the possibility of sporotrichosis should be borne in mind, particularly if the patient's occupation leads to contact with vegetable products such as hay or straw, grain, bird foods, or even cotton, as in the case of seamstresses and tailors.

Aortic Aneurysm far less often opens into the lung itself than it does into a bronchus ; the symptoms are similar in either case, and if the history is long the diagnosis will already have been made on account of some other symptom than hæmoptysis, especially pain in the chest or in the back. The *x*-rays are a valuable means of deciding the diagnosis (*Fig.* 100, p. 209). Two points are worthy of particular attention, and these are : first, that the rupturing of an aortic aneurysm into a bronchus, with copious and rapidly fatal hæmoptysis, may be the very first sign that anything is wrong ; and secondly, that in not a few cases there may have been slight hæmoptysis and blood-streaking of the sputum for weeks or months before the fatal rupture ensues ; these preliminary slight attacks of hæmoptysis are probably due to erosion of small vessels in the wall of the bronchus, and if the aneurysm is partially obstructing, say, the left upper bronchus, so as to produce impairment of note over the left apex, with a few râles there, and hæmoptysis, it is clear that a mistaken diagnosis of phthisis might readily be made. Tubercle bacilli will be persistently absent from the sputum, there will very likely be a previous history of syphilis, hard manual work, and perhaps drinking ; without the *x*-rays to show the pulsating shadow of the aneurysm, however, the correct diagnosis may be missed, and even when the fatal rupture occurs, the condition may still be erroneously attributed to phthisis, unless a post-mortem examination is made.

Empyema bursting through the Lung may or may not cause hæmoptysis ; the main features of the case will generally be an obscure febrile illness subsequent to pneumonia, followed by a sudden eruption of pus from the respiratory passages, and a repetition of a similar copious expectoration of pus at intervals ; in many cases there are comparatively few abnormal physical signs, for had the empyema not been hidden away deeply in the thorax, its existence would have been diagnosed earlier, and it would have been relieved by operation before it burst into the lung.

A Hepatic Abscess that has burst through the lung is apt to give rise to anchovy-sauce-coloured sputum which is characteristic ; no amœbæ may be discovered, and the pus will very likely be sterile ; the diagnosis is generally based upon the history of residence in the tropics, possibly of an attack of amœbic dysentery, and of hepatic symptoms, pyrexia, and rigors previous to the expectoration of the blood-stained pus. The abscess occurs on the right side more often than on the left, and there may be the typical dome-shaped dullness at the base of the right lung.

Hydatid Cysts are much rarer in Europe than in Australia and New Zealand ; those of the lung are, as a rule, secondary to hydatid of the liver. They may give rise to neither signs nor symptoms ; on the other hand, they may cause hæmoptysis, and phthisis may be simulated. The *x*-rays are very efficient in detecting their spherical shadows (*Fig.* 137). The blood may exhibit eosinophilia or the specific hydatid serum reaction.

Primary Atheroma of the Pulmonary Arterioles is so rare as to be undiagnosable. There is no relationship between systemic and pulmonary atheroma, and the commonest cause of the latter is mitral stenosis, as described above.

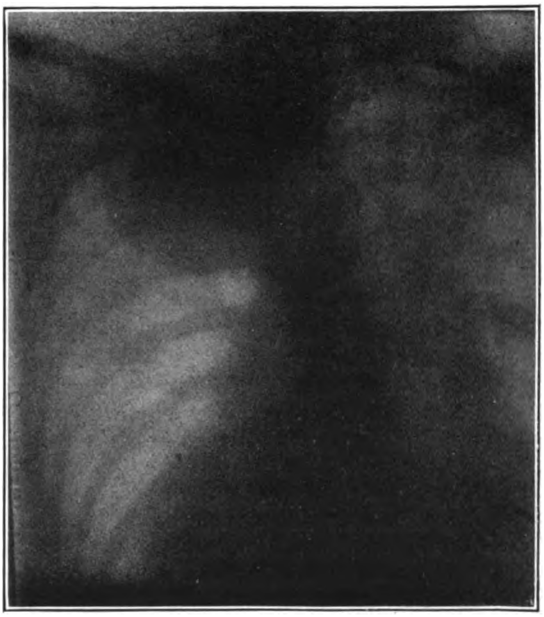


Fig. 137.—Skiagram of a hydatid cyst of the thorax, occupying the position of the upper lobe of the left lung.

Hæmoptysis due to changes in the Bronchioles, Bronchi, and **Trachea,** as distinct from changes in the lung, have to some extent been incidentally considered with the latter.

Bronchitis should never be diagnosed as the cause of hæmoptysis until phthisis and mitral stenosis have been thoroughly excluded.

Bronchorrhœa is, in most respects, only a variety of bronchitis.

Bronchiectasis may be associated with recurrent slight or even severe hæmoptysis sometimes, or when the bronchiectasis is due to obstruction of a bronchus by a thoracic aneurysm there may be copious and fatal hæmoptysis, as described above. Bronchiectasis seldom occurs apart from fibrosis of the lung ; indeed, fibroid lung is commoner than bronchiectasis ; when fibrosis and bronchiectasis occur together and affect one lung in particular, the diagnosis is relatively easy, for there is deficiency of bulk, movement, and resonance, tactile vocal fremitus, vesicular murmur, and voice sounds over the affected lung; the heart is materially displaced towards that side: numerous loud crackling râles, with or without bronchial breathing, bronchophony, and pectoriloquy, will be heard over scattered patches of the affected lung, whilst in the intervening areas there will be little to be heard at all ; the râles will be brought out best when the patient coughs ; the lung on the other side may give relatively normal signs. Clubbing of the fingers may be extreme. The diagnosis of fibroid lung and bronchiectasis itself is not complete, however, until the precise cause of the latter has been ascertained ; sometimes so complete a diagnosis is not possible. The following is a list of the chief causes of the condition :—

1. *Causes in the lung :—*
   - Congenital atelectasis
   - Recurrent attacks of bronchopneumonia
   - Delayed resolution of lobar pneumonia
   - Pneumonoconiosis
   - Chronic tuberculosis
   - Sporotrichosis
   - Recurrent bronchitis (doubtful)
2. *Causes which act by partially stenosing a bronchus :—*
   - (*a*). Causes within the bronchus :
     - A foreign body
     - Inspissated bronchitic mucus.
   - (*b*). Causes in the wall of the bronchus :
     - Syphilitic stenosis
     - Primary epithelioma.
   - (*c*). Invasion of the bronchus from without :
     - Aortic aneurysm
     - Mediastinal new growth
     - Hodgkin's or lymphadenomatous glands
     - Caseous bronchial glands
     - A hypertrophied left auricle in some cases of mitral stenosis.
3. *Causes which have long compressed the lung from the pleural side :—*
   - Pleuritic effusion
   - Pleural effusion
   - Thick pneumonic lymph
   - Empyema
   - A large heart
   - Pericardial effusion
   - Ascites
   - Subdiaphragmatic abscess
   - Hepatic tumour
   - Splenic tumour.

There will be no need to discuss each of these here ; if the different possibilities are kept in mind, a probable diagnosis can be made fairly easily in most cases. Amongst modern methods of diagnosis one must not forget the bronchoscope, through which, in skilled hands, it is often possible to get visual proof of the nature of a tracheal or bronchial obstruction. The only bronchial causes of hæmoptysis that need be dealt with further are syphilitic ulceration and infection by the *Distoma pulmonale.*

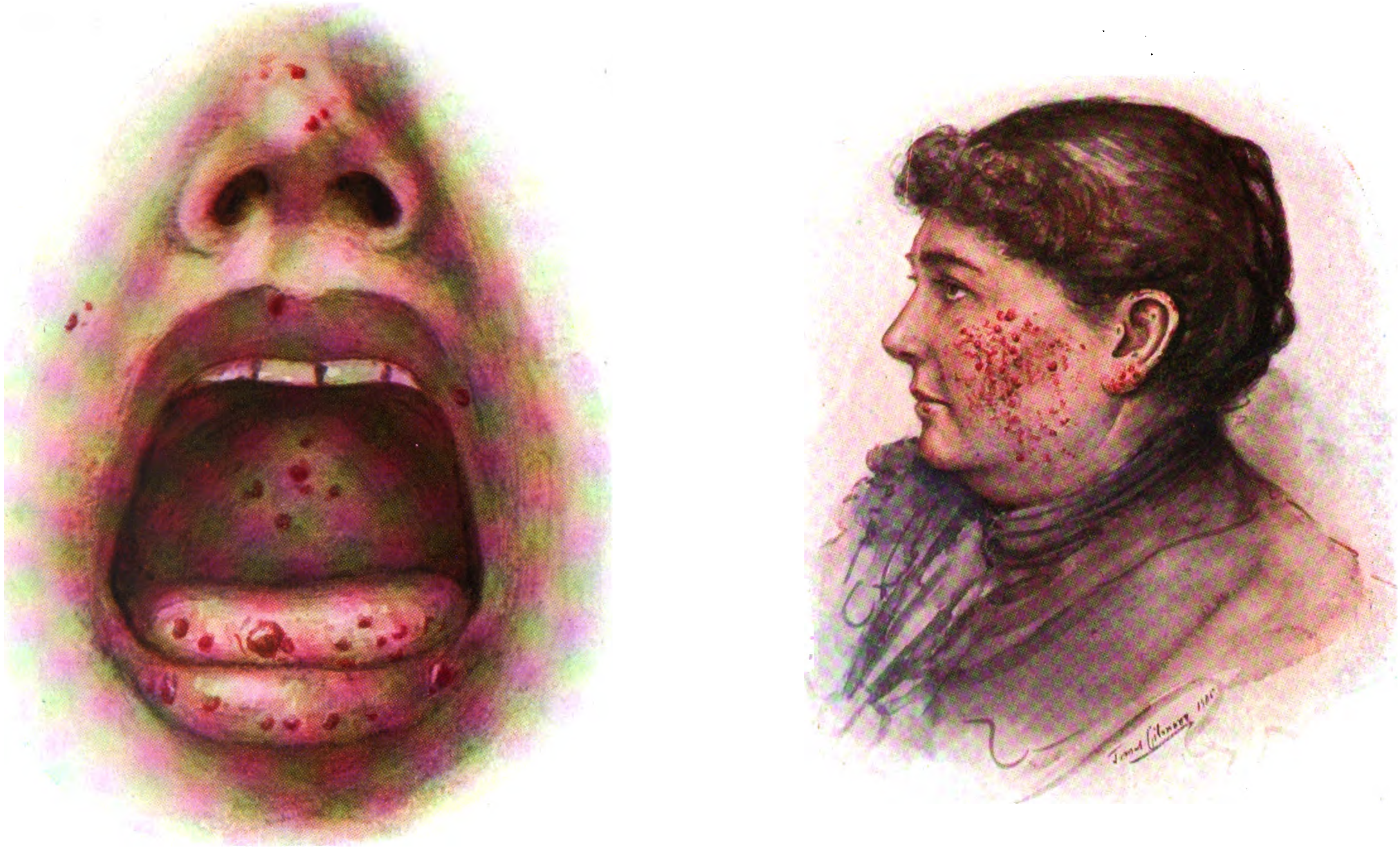
Syphilitic Disease of a Bronchus is a tertiary lesion of gummatous nature, and as it heals it causes bronchial stenosis and consequent fibrosis of the lung, with or without bronchiectasis. It will hardly be diagnosed unless there are other very definite means of knowing that the patient has had syphilis, or is still suffering from its tertiary effects, or has a positive Wassermann reaction ; and even then care must be taken to exclude the possibility of the luetic patient having developed phthisis. The influence of iodide of potassium in such a case does not afford conclusive evidence one way or the other, for even though the syphilitic lesion heals, it leaves behind it the fibrous stenosis of the bronchus.

The **Distoma Pulmonale Westermanni** is very unlikely to be the cause of hæmoptysis in any patient who has not been resident in China, Japan, or Formosa. History of residence in those countries, on the other hand, would suggest the diagnosis, confirmation of which would be afforded by examination of the sputum for the parasites or their ova.

The differential diagnosis of **Hæmoptysis due to changes in the Larynx** depends mainly on two things : the history of the case, and the condition seen locally with the laryngoscope. The history and course are the chief factors in diagnosing acute simple laryngitis, post-typhoidal, post-diphtheritic, or variolous ulceration of the larynx, or conditions due to injury of the larynx by a blow, a hand-grip, a cut throat, or intubation or other operation. Leprous ulceration of the larynx seldom, if ever, occurs in any patient who has not lived in leprous lands, and who has not for a long time exhibited subcutaneous and cutaneous evidence of his disease. Of the remaining five conditions given in the list, namely,

PLATE XVII

BLEEDING NÆVI



Multiple bleeding nævi of the tongue, mouth, and cheeks.

tuberculous, syphilitic, and malignant ulcerations, lupus, and angioma of the larynx, the last two are very rare indeed, though both may be diagnosable by their laryngoscopic appearance, particularly if there is also lupus of the face on the one hand, or a tendency to cutaneous or buccal blood-oozing nævi on the other (*Plate XVII*). Between the remaining three conditions there may be some doubt for a time, but if it can be seen that the ulceration is extensive and yet unilateral, it is probably epitheliomatous ; if tubercle bacilli are present in the sputum, if there are apical lung signs, and if multiple shallow ulcers can be seen along the epiglottis, as well as in the larynx, tuberculous ulceration is probable—it practically never occurs except secondary to pulmonary tubercle, though the latter may be slight and may remain in abeyance whilst the laryngeal tubercle advances rapidly ; syphilitic laryngitis may be diagnosed by exclusion, but if there is a tendency to healing, with marked deformity, after extensive bilateral destruction of the laryngeal and neighbouring tissues, and if there is decided collateral evidence of tertiary syphilis, including, perhaps, a positive serum reaction, the diagnosis may often be made directly. The chief difficulty arises in cases in which there may be both syphilis and tubercle at the same time. This brings us back once more to the fact that, once it has been decided that true hæmoptysis has occurred, the next step is to examine the sputum and the chest carefully for signs of tubercle, and not to diagnose any other condition until both tubercle and mitral stenosis have been excluded. *Herbert French.*

HÆMORRHAGE, GASTRIC.—(See HÆMATEMESIS, p. 265.)

HÆMORRHAGE FROM GUMS.—(See BLEEDING GUMS, p. 72.)

HÆMORRHAGE, INTESTINAL.—(See BLOOD PER ANUM, p. 75 ; and MELÆNA, p. 385.)

HÆMORRHAGE FROM LUNG.—(See HÆMOPTYSIS, p. 285.)

HÆMORRHAGE, NASAL.—(See EPISTAXIS, p. 220.)

HÆMORRHAGE, RETINAL.—(See OPHTHALMOSCOPIC APPEARANCES, p. 415.)

HÆMORRHAGE, SUBCUTANEOUS.—(See PURPURA, p. 552.)

HÆMORRHAGE, URINARY.—(See HÆMATURIA, p. 275.)

HÆMORRHAGE, UTERINE AND VAGINAL.—(See MENORRHAGIA, p. 385 ; METRORRHAGIA, p. 390 ; and METROSTAXIS, p. 392.)

HÆMOTHORAX.—(See CHEST, BLOODY EFFUSION IN, p. 102.)

HALTING.—(See GAIT, ABNORMALITIES OF, p. 251.)

HAND, CLAW.—(See CLAW-HAND, p. 109.)

HEAD, RETRACTION OF.—(See RETRACTION OF THE HEAD, p. 589.)

HEADACHE is one of the commonest symptoms met with in medical practice, and the various conditions with which it is associated are numerous, as the list given below demonstrates.

Headache may be the first symptom calling attention to the existence of grave organic disease, and the correct diagnosis of the cause of this symptom is obviously of the greatest importance. Too often, unfortunately, treatment of a headache precedes a careful investigation as to its cause, and an increased risk may thereby be incurred by the patient through the delay in recognizing some one of its more serious causes.

The explanation of the mode of production of the pain known as *headache* is not easy, seeing that the brain substance itself is insensible to mechanical stimulation. The meninges are supplied with sensory nerves, and abnormal stimuli received therefrom reach the cortex and give rise to the impression of pain. Abnormal states of the intracranial blood-vessels may cause pain, which is more difficult of explanation, as it is uncertain that they have any sensory nerve-supply. It seems probable that the headache produced by increased vascular tension is a pressure effect acting on the brain as a whole, or on its coverings the meninges. The scope of this article does not allow further discussion of this part of the subject. Certain general lines of diagnosis may be laid down. The closest attention should be paid to the character, situation, and time of occurrence of the pain, and also to accompanying symptoms.

Character.—Whether throbbing, paroxysmal, or affected by movement or position. Headaches associated with alimentary disturbance, and raised blood-pressure, are often throbbing in character, are relieved by rest in a recumbent position, and are increased on movement. Severe paroxysmal attacks would suggest a neuralgia.

Situation.—This may be frontal, vertical, occipital, or unilateral, and in cases of organic disease of the cerebrum may be an important indication and an aid in localizing the situation of the lesion. In renal disease, the headache associated with chronic uræmia is usually frontal, but may be occipital. It is vertical in constipation, the 'bilious' headache. It may be unilateral in migraine, tumour, abscess, middle-ear disease ; or occipital in cerebellar disease. Occipital headache may also be simulated by myalgia in the muscles and tendons of the nape of the neck.

Time of Occurrence.—Headache associated with organic disease of the brain or its meninges often persists or becomes worse at night, and may wake the patient from his sleep, whereas that due to toxic and functional causes is relieved by rest in a horizontal position. Grave suspicion of the organic nature of the headache should, therefore, attend a case in which pain in the head disturbs the patient's sleep at night. A headache experienced on rising in the morning may be due to a stuffy, ill-ventilated room, or to the slighter degrees of combined astigmatism and hypermetropia, or to faulty adjustment of the pillows. Pillows piled too high may cause interference with the cerebral circulation and result in headache. Persistent morning headache may be associated with chronic nephritis, and careful observation should therefore be made of the patient's urine. *Evening* headaches are most commonly due to mental overwork, or eyestrain, especially where some visual defect exists.

For the purposes of classification it is convenient to divide the causes of headache into three main groups :—(*A*) *Organic disease* (brain, intracranial vessels, meninges, skull, special sense organs) ; (*B*) *Toxic states* ; (*C*) *Functional conditions.*

A. Causes due to Organic Disease.

These may be classified anatomically as follows :—

1. *Diseases of the brain :—*

Concussion
Tumours
Abscess
Gumma
Cysts
Hydrocephaly
Disseminated sclerosis
General paralysis of the insane.

2. *Diseases of intracranial vessels :—*

Hæmorrhage (rupture)
Thrombosis
Embolism
Aneurysm
Syphilitic endarteritis
Arteriosclerosis.

3. *Diseases of the meninges :—*

Meningitis, various forms —localized or diffuse
Pachymeningitis
Syphilis—meningeal type
Tumours.

4. *Diseases of the skull :—*

Tumours { Innocent
Malignant { Primary
Secondary.
Tertiary syphilis.
Suppuration or new growth in frontal, antral, or mastoid sinuses
Suppuration or tumour in the orbit
Dental diseases.

5. *Diseases of special sense organs :—*

Eye—errors of refraction, iritis, glaucoma, etc.
Ear—middle-ear disease.
Nose—adenoids, polypi, nasopharyngeal catarrh. Inflammation of one of the accessory air sinuses—frontal, ethmoidal, sphenoidal ; empyema of a frontal sinus.

Headache in Organic Cerebral Disease.

Time of Occurrence.—Organic cerebral disease should be suspected if a history of recurrent nocturnal headache be obtained.

Severity.—The pain is often intense, and sometimes paroxysmal in character.

Situation.—This may give some clue as to the existence of an organic lesion. In cases of cerebral tumour the pain may be unilateral or frontal, or occipital with a cerebellar lesion. In middle-ear and mastoid disease with unilateral headache and localized tenderness, occipital headache may be one of the earliest symptoms of meningitis.

Associated Signs and Symptoms.—One or more of the following signs and symptoms may present themselves at an early period in cases of headache due to organic cerebral disease, and their early recognition is of great importance :—

Vomiting—that is of the cerebral type' (see VOMITING, p. 763): it usually bears no relation to food, and is not preceded by nausea
Inequality of the pupils
Squint
Optic neuritis (*Plate XIX*, p. 416)
Irregularity in force and frequency of the pulse
The onset of drowsiness
Fits.

Tapping the skull over the site of the pain may reveal local tenderness.

The onset of any of these signs associated with headache would point to the existence of some organic lesion, such as are enumerated above. As in many of these conditions the diagnosis is unattended with difficulty, it will suffice to direct attention to a few of them.

The headache occasionally met with in *disseminated sclerosis* is sometimes paroxysmal and accompanied by vomiting, and is situated most frequently in the back of the head and neck. The absence of optic neuritis and the presence of the special signs of disseminated sclerosis should lead to a correct diagnosis.

Cerebral hæmorrhage, thrombosis, and *embolism* are often followed by headache of varying severity. In *cerebral aneurysm* a rhythmic beating or pulsation is sometimes felt and rushing noises are heard, more particularly when the internal carotid is involved. Rhythmic beatings or noises in the head, however, do not by themselves suggest an intracranial aneurysm, for they are complained of commonly by many anæmic persons and by sufferers from vascular degeneration, especially atheroma of the cerebral arteries.

Advanced *arteriosclerosis* is sometimes attended by severe headache accompanied by vomiting; and cases have been described presenting features closely resembling those of cerebral tumour. In arriving at the diagnosis instrumental measurement of the blood-pressure is all-important.

In *meningitis*, especially in the epidemic cerebrospinal and the post-basal varieties, the character of the headache is significant. It is usually very intense, is occipital, and even at an early stage may be attended by stiffness of the neck and retraction of the head. Examination of the cerebrospinal fluid (p. 304) obtained by lumbar puncture is of great importance in determining the presence of meningitis.

Special Sense Organs.—Eye. Headaches due to errors of refraction, glaucoma, iritis, etc., are generally frontal or temporal. A slight error of refraction may cause what appears to be a disproportionately severe headache, particularly in children. This headache is frontal, occurs mostly in the evening or after school hours, and is often attended by a burning, pricking, or watering of the eyes. Correction of the defect by suitable glasses settles the diagnosis by curing the headache.

B. Toxic Causes.

These may be subdivided into two groups, one in which the toxic influence is acquired from without, or is *exogenous*; the other in which the disturbing element or toxin is produced within the body, and is of *endogenous* origin.

1. *Of exogenous origin* :—

Foul air, as in close, ill-ventilated rooms
Poisonous gases, CO_2, CO, chloroform, ether, etc.
Drugs, e.g., quinine, iron in some individuals, salicylates, opium.
Alcohol | Tobacco | Lead poisoning.

2. *Of endogenous origin* :—

Uræmia
Cholæmia
Gout
Diabetes
Gastro-intestinal disturbances : dyspepsia, constipation
Toxæmias : specific fevers, pyrexial phthisis, suppuration, etc.

As regards the *toxic causes* of headache little further need be said as to the diagnosis of the exogenous poisons.

Uræmia is classed for purposes of convenience as a condition due to endogenous bodies, or substances produced within the body. It stands out as one of the most important causes of headache, and special attention must always be given to ensure its recognition. Uræmic headaches may be met with in all degrees of severity, from a slight frontal headache felt on rising in the morning to an intense vertical or general cephalalgia. Other uræmic manifestations may be present, such as vomiting, drowsiness, dyspnœa, affections of vision, and retinal changes. Examination of the urine in all cases of headache should never be neglected, as regards its specific gravity, the presence of albumin, blood, and casts.

C. **Functional Causes :**

Abnormal blood-pressure { high, in arteriosclerosis and renal disease
{ low, in anæmia, morbus cordis, Addison's disease.

Venous congestion
Excessive mental strain
Pressure on the head—heavy hats, carrying weights on the head
Persistent noises—' gun headache '
Sea-sickness—movement of boat, train
Menstruation
Hysteria—(' clavus hystericus ')
Migraine
Epilepsy
Eye strain, ' academy headache '
Sunstroke.

High blood-pressure is often a cause of headache, usually of a throbbing character, accompanied by a sense of fullness of the head. The headache tends to come on towards evening and after meals. The vascular condition should be ascertained by means of some suitable apparatus for estimating blood-pressure.

Headache associated with *low blood-pressure* (cerebral anæmia), as in some forms of morbus cordis and anæmia with feeble cardiac action, is relieved by rest in the horizontal position and cardiac tonics such as digitalis and iron.

Venous congestion may cause headache. This is also met with in heart disease with failing compensation. It may also account for the headache felt on rising in the morning as the result of sleeping with pillows too high or too low.

The ' *clavus hystericus* ' is a boring pain felt in the vertex and in hysterical states.

Headache in *migraine* is often unilateral, though quite commonly bilateral, and frequently it is accompanied by vomiting. Transitory visual disturbance usually precedes the headache.

In *epilepsy* headache is of frequent occurrence in the post-epileptic state, and it should be borne in mind that it may also follow the slight manifestations of *petit mal.*

After *sunstroke*, chronic headache, usually vertical, may persist for months, and the same applies to many head injuries.

It is sometimes difficult to distinguish between *headache*, which implies pain inside the skull, and *neuralgia*, which is pain felt in the peripheral course of a nerve trunk (see PAIN IN THE FACE, p. 446). Neuralgia, if of wide distribution, may simulate headache. Careful examination may be necessary to decide whether the supposed headache may not in reality be a neuralgia. The local distribution, the often intense and paroxysmal character of the pain, the presence of ' tender spots,' the existence of some definite exciting cause such as dental caries, should point to the diagnosis of neuralgia. *H. Morley Fletcher.*

HEARTBURN is a common symptom, yet difficult to define. Apparently it is due to regurgitation into the lower end of the œsophagus from the stomach of acid products of digestion, and it is thus related to pyrosis or waterbrash, in which similar acid products regurgitate suddenly as far up as the upper end of the pharynx and the back of the mouth, causing a local sense of burning acidity in the throat, and often a temporary huskiness of the voice. Neither waterbrash nor heartburn is distinctive of any particular malady. Either may occur in a perfectly healthy individual who for some transient digestive cause has rather more gas in his stomach than the latter can hold comfortably ; with the escape of some of this gas a drachm or two of the liquid gastric contents may be shot up into the lower end of the œsophagus to cause heartburn, or further up still to cause waterbrash. On the other hand, if the symptoms are persistently troublesome, either may indicate more serious lesions, such as flatulent dyspepsia (see FLATULENCE, p. 240) or pyloric stenosis from healed ulcer or gastric carcinoma (see DILATATION OF THE STOMACH, p. 173). If the symptoms are obviously not transient, analyses of test meals (pp. 270 and 319) or x-ray examination of the stomach (*Fig.* 122, p. 245) may be required before the diagnosis of their cause can be established. Duodenal ulcer, gall-stone dyspepsia, appendix dyspepsia, and ileocæcal kinking will also need to be borne in mind as possibilities.

The actual symptoms of heartburn take two forms. The first consists in a more or less acute pain, sometimes of a really burning character, more often of a severe aching, boring, or even lancinating type, referred to the mid-line of the lower end of the gladiolus, especially between the two fifth costal cartilages, coming on as a rule an hour or more after food, when digestion is at its height, beginning either gradually or suddenly, often increasing in severity for half an hour or an hour, and lasting sometimes for several hours or a whole day ; this is generally referable to its cause—flatulence—with ease ; especially if simple treatment with bicarbonate of soda and a little rhubarb relieves or cures the pain. The other type consists in attacks of acute gripping pain in the precordial region, especially near the apex of the heart ; this pain comes on quite suddenly, and it often lasts less than a minute and seldom more than a few minutes. While it is there the patient may find himself unable to take an ordinary breath without increasing the pain to an unbearable extent ; he therefore holds his breath entirely for as long as he can, generally presses a hand over the precordial region, and when he is compelled to inspire again he finds that he gets checked before he has breathed in as much air as he would like ; he therefore contents himself for a minute or two with a minimum depth of breathing, by which time the acute stitch-like precordial pain passes off and he is able to breathe normally again. The attack may be repeated after an interval of minutes or hours ; there is often no sense of palpitation, but in some cases severe palpitations accompany or follow the pain, and the main difficulty is to exclude organic heart disease which the patient fears his symptoms indicate. When the attacks are only very occasional, and there is no shortness of breath, gastric disorder is more probable than heart trouble ; when, however, the attacks are frequent and the patient is out of condition, it may require very skilful judgement to decide that the attacks of precordial colic are of gastric and not cardiac origin. Fatty or fibroid or tobacco hearts are the most difficult to exclude ; the relief the patient receives from carminatives such as bicarbonate of soda, gentian, cajaput oil, ginger, rhubarb, peppermint, does not necessarily indicate that the trouble is primarily gastric ; and even when the fullest examinations have been made, including the use of the electrocardiograph, there are many instances in which it remains very much a matter of opinion whether the attacks are due to myocardial changes or to the much less serious heartburn. *Herbert French.*

HEART, ENLARGEMENT OF.—(See ENLARGEMENT OF THE HEART, p. 206.)

HEART IMPULSE, DISPLACED.—The apex beat, which is the lowest and outermost point at which the cardiac impulse can be felt, is situated in the normal adult chest in the fifth left intercostal space, one-half to one inch internal to the mammary line. It may be impossible to define the position of the apex beat even in health, on account of increased thickness of the chest wall either from muscular development or excess of fat ; or in the female on account of a large mamma. A similar difficulty arises when the cardiac impulse is feeble ; when the heart is overlapped by the left lung, as in pulmonary emphysema ; or when pericardial effusion is present. In children, the apex beat is situated further to the left and a little higher than in adults. Speaking generally, it is outside the mammary line during the first three years of life, in the mammary line from the fourth to the tenth years, and it gradually reaches the adult position by the age of fifteen.

The conditions which produce displacement of the cardiac impulse are :—

A. **When the Bulk of the Heart is in the Normal Position.**

1. *Diseases of the heart* :

   (*a*). Valvular ; (*b*). Myocardial ; (*c*). Pericardial.

2. *Changes in the heart secondary to* :
    - (*a*). Diseases of the lungs, such as emphysema, fibrosis, etc.
    - (*b*). Arterial sclerosis and chronic renal disease
    - (*c*). Anæmia and debilitating conditions, affecting chiefly the right ventricle
    - (*d*). Toxic conditions producing myocardial changes, as in infective diseases
    - (*e*). Muscular exertion.

B. **When the Whole Heart is Displaced.**

1. *Changes in the lungs* :
    (*a*). Contraction of one lung or a portion of it ; (*b*). New growth of lung.

2. *Changes in the pleuræ* :
    (*a*). Pleurisy with effusion, empyema, pneumothorax ; (*b*). New growth of pleura.

3. *Other thoracic tumours* :—New growth, aneurysm.

4. *Deformities of the chest wall* :—The result of scoliosis.

5. *Changes in the abdomen* :—Ascites ; tympanites ; abdominal tumour ; pregnancy.

6. *Transposition of the heart.*

To distinguish between the two groups is usually not difficult, for when the bulk of the heart is in the normal position and the apex beat is displaced beyond the left mammary line, the area of cardiac dullness is increased, not only to the left, but also to the right of the sternum, and upward. If both lungs are emphysematous and the cardiac apex is displaced outwards, although the size of the heart cannot be estimated by percussion, yet the bulk of the heart may confidently be presumed to be in the normal position.

The presence of a cardiac lesion, arterial sclerosis, or chronic renal disease helps to confirm the view that the abnormal position of the apex beat is due to an increase in the bulk of the heart, and not to a displacement of the organ as a whole. The examination of the pulse gives valuable information ; if it be of high tension and is sustained, or if the pulse is of the ‘ water-hammer ’ type, it indicates that the displaced apex beat is due to enlargement of the left ventricle, and that probably there is no displacement of the heart. The blood-pressure is increased in arterial sclerosis and in renal disease, and must therefore be estimated, for it gives additional evidence that the displaced apex beat is part of a general enlargement of the heart. Examination of the urine must never be omitted ; if there be polyuria, with a small trace of albumin, low specific gravity, and hyaline and granular casts, the presence of chronic interstitial nephritis is ascertained, and this will account for the enlargement of the heart, and any displacement of the apex beat down and to the left.

The presence of a cardiac bruit is of great value in determining that the displaced apex is due to morbid changes in the heart ; but the absence of a bruit does not necessarily mean that the displaced apex beat is unassociated with cardiac disease. The enlargement of the left ventricle, due to arterial degeneration or chronic interstitial nephritis, may not be accompanied by any bruit unless dilatation becomes so great that mitral regurgitation supervenes. The characters of any of the cardiac sounds are frequently altered in dilatation and hypertrophy of the left ventricle. Thus, the aortic second sound may be accentuated on account of the increased arterial tension, and the second sound over the base of the heart may be reduplicated on account of the aortic and pulmonary valves not closing synchronously. The first sound is frequently louder and more prolonged in hypertrophy of the ventricles, due to an increase in the muscular element of the sound, and the greater force with which the auriculo-ventricular valves are closed. In simple dilatation of the ventricles the first sound is often slightly accentuated, but is usually sharper and shorter.

In the second class of cases, in which the whole heart is displaced, the cause of the displacement is usually easy to ascertain. The chest is frequently asymmetrical, for there will be either bulging of the chest wall on the side from which the heart is displaced, or some shrinking on the side to which it is drawn. Percussion may show that resonance is present where normally there is cardiac dullness ; thus, when the right lung is emphysematous and the heart is pulled over to the left, the resonance of the right lung may be found extending to the left of the sternum. It is not always so easy to determine the boundaries of the heart when the displacement is due to the presence of a *pleural effusion*, as there is dullness over the effusion which may be continuous with the cardiac dullness. In such a case, however, the dullness over the base of the lung is not only in front, but is likely to be found behind as well. There are also signs of compression of the lung by the effusion, such as absence of breath and voice-sounds, tubular breathing, or skodaic resonance; ægophony may be heard at the upper level of the fluid, and it is always very suggestive of pleuritic effusion. When the heart is drawn over to one side by the *contraction of the lung*, as in fibrosis, there is impaired resonance upon percussion over the fibrosed lung, continuous with the cardiac dullness. The boundaries of the heart may be difficult to define by percussion, but over the fibrosed lung, breath-sounds are present, tubular in character, vocal fremitus and resonance are decreased, and crackling râles and other adventitious sounds may be heard. When the heart is drawn over in this manner by fibrosis of one lung, the resonance over the healthy lung will be found to extend across the middle line, and thus invade the normal position of cardiac dullness. Examination of the chest by means of the x-rays usually helps to determine the position of the heart; but large tumours, pleuritic effusions, etc., produce shadows which may be continuous with that of the heart. The pulsations of the heart are generally well seen, especially in children, and indicate its position.

The *changes in the abdomen* causing displacement of the heart upwards are not likely to be overlooked, because there must be a considerable amount of abdominal enlargement before the heart can be raised by it; and therefore if displacements of the apex-beat are due to ascites, tympanites, abdominal tumours, or pregnancy, the causes are all of such a marked degree or in such an advanced stage, that they are easily recognizable. When the heart is displaced as the result of marked changes in the lungs and pleuræ, intrathoracic tumours, or abdominal enlargements, the causes of the displacement are usually found first on account of the symptoms and physical signs they produce; the alteration in the position of the apex beat is then a confirmatory sign.

In the first group, in which the bulk of the heart is in the normal position, the direction in which the apex beat is displaced is of some diagnostic value. It is displaced downwards and to the left in *hypertrophy of the heart*, especially when it affects chiefly the left ventricle. In *mitral regurgitation* the apex beat is displaced outwards and to the left, whereas in *lesions of the aortic valves* the displacement is to the left and downwards, so that the apex beat is commonly situated in the sixth intercostal space. In both these conditions the left ventricle is enlarged, but with mitral regurgitation the right side of the heart becomes enlarged early in the disease, and the apex is displaced much more outwards than downwards. When the *right ventricle is alone enlarged*, as in pulmonary emphysema, the displacement of the apex beat is directly to the left, and not downwards at all.

The varying enlargement of the two ventricles may be fairly gauged by watching the position of the apex. It is raised and displaced slightly to the left by any cause which increases the height of the diaphragm, such as ascites, tympanites, abdominal tumours, and pregnancy. The cardiac impulse is also raised by pericardial effusion. There are, however, a considerable number of cases of displaced apex beat, with the bulk of the heart in the normal position, in which it is difficult to ascertain the cause of the displacement. This is especially so in young adults, in whom the apex beat may be found to be displaced slightly outwards without any apparent cause. If the subject be a young and muscular man who otherwise seems in good health, the condition is probably due to hypertrophy of the left ventricle as the result of *excessive athletic exercise* or of some arduous muscular work. The history would confirm this view. If, on the other hand, the patient is not a muscular individual, the displacement may still be due to strain, but some other cause should always be looked for, and in the absence of any obvious cardiac lesion, *pericardial adhesions* must not be forgotten: these may produce few symptoms except slight enlargement of the heart.

In young girls the apex beat is often situated in the mammary line, and this displacement is associated with *chlorosis* and other debilitating conditions which produce dilatation of the conus arteriosus. The apex beat is not only displaced outwards, but also raised. The diagnosis is confirmed by finding that the cardiac dullness is increased in an upward direction, and by the presence of a functional systolic bruit in the pulmonary area and a *bruit de diable* in the neck.

In elderly people, in whom there is no valvular disease of the heart, the apex may be displaced, not only as the result of hypertrophy of the left ventricle secondary to renal disease and arterial sclerosis, and as the result of enlargement of the right ventricle secondary to pulmonary emphysema; but also as the result of *myocardial degeneration*.

The last condition may be difficult of diagnosis, but when there is no evidence of valvular disease, emphysema, chronic renal disease, arterial sclerosis, or anything causing displacement of the heart as a whole, it must always be suspected. Symptoms which frequently accompany it are dyspnœa upon exertion, attacks of syncope, palpitation, and œdema of the legs. The pulse is often feeble and irregular, and in failing cases the impulse is feeble, a gallop rhythm may be present, and a soft systolic bruit at the apex.

Another form of uniform cardiac hypertrophy that needs special mention is that which results from long-continued drinking of large quantities of fluid, particularly beer—the *beer-drinker's heart.* *J. E. H. Sawyer.*

HEART SOUNDS, ACCENTUATION OF.—(See Accentuation of Heart Sounds, p. 1.)

HEART SOUNDS, REDUPLICATION OF.—(See Reduplication of Heart Sounds, p. 587.)

HEMERALOPIA.—(See Vision, Defects of, p. 763.)

HEMIANÆSTHESIA.—(See Sensation, Abnormalities of, p. 610.)

HEMIANOPSIA—or, as it is sometimes called, *hemiopia* or *hemianopia*—means inability to see objects in one half of the field of vision. It is generally, but arbitrarily, restricted to cases in which this defect is due to changes elsewhere than in the retina or disc. It is not a common condition, but it sometimes escapes recognition because, whichever half of the visual field has become blind, good vision remains at the central part, and even the patient himself may not always be conscious of his defect until some accident,

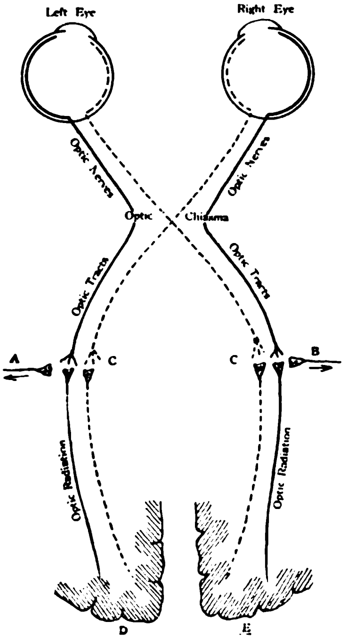


Fig. 138.—A diagram illustrating the connections of the optic nerves and tracts, the 3rd cranial nerves, and the occipital cortex. A, 3rd nerve going to left eye; B, ditto to right eye; C, relay of cells in optic thalamus and superior corpus quadrigeminum; D, left occipital cortex, which sees objects in the right half of the field of vision; E, right occipital cortex, which sees objects in the left half of the field of vision.

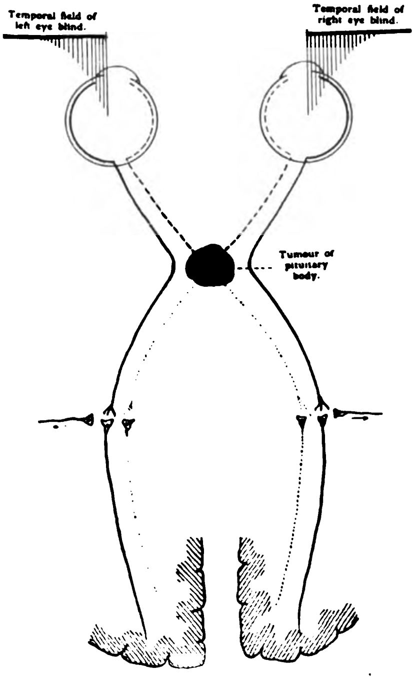

Temporal field of left eye blind.
Temporal field of right eye blind.
Tumour of pituitary body.

Fig. 139.—A diagram showing how a tumour of the pituitary body affecting the decussating fibres at the optic chiasma prevents impulses passing from the nasal half of either retina to the corresponding cortex or to the corresponding 3rd nucleus. Hence bilateral temporal hemianopsia and absence of pupil reaction to light thrown on the nasal half of either retina.

such as running into objects in broad daylight, draws his attention to it. To map out the blind area with accuracy an instrument known as the perimeter is required.

It is possible to get hemianopsia in one eye only, but this is very rare apart from functional conditions or migraine.

When both eyes are affected, the blindness may affect : (1) Corresponding halves of the field of vision—*bilateral homonymous hemianopsia*—spoken of as *right* if neither eye can see objects in the patient's right-hand half of the field of vision (*Figs.* 140 and 141), and as *left* if in the left half ; or (2) Opposite halves of the field of vision—almost invariably the temporal halves, and referred to as *bilateral temporal hemianopsia* (*Fig.* 139).

These are the only two varieties that are of clinical importance. They are generally not the only symptoms in the case, but they sometimes serve to localize certain cranial lesions with accuracy.

Bilateral Homonymous Hemianopsia has a variety of causes, affecting one or other of three main sites, namely : (*a*) One optic tract ; (*b*) The posterior limb of one internal capsule ; or (*c*) The optic radiations or one occipital region at or near the cuneus. In any of these sites the pathological lesion may be either vascular—thrombosis, hæmorrhage, embolism, or intermittent closure ; or a neoplasm, such as a gumma, a tuberculous nodule, an inflammatory swelling, or a gliomatous, carcinomatous, or sarcomatous nodule. The first step is to locate the site of the lesion ; its nature will then be determined more easily, because in the internal capsule a hæmorrhage, thrombosis, or embolism of the middle cerebral artery is the commonest cause of the symptom ; a neoplasm, or an abscess, is probably its commonest cause in the occipital cortex, though an abscess, or rupture or occlusion of the posterior cerebral artery would also be thought of ; in the optic tract it is as often as not gummatous, or in some other way syphilitic.

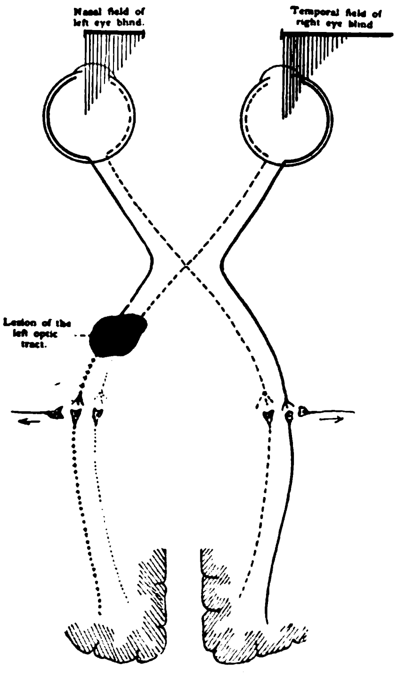

Nasal field of left eye blind.
Temporal field of right eye blind
Lesion of the left optic tract.

Fig. 140.—A diagram showing how a lesion of the left optic tract causes blindness of the right half of the field of vision of each eye, and also prevents the pupils from reacting in response to a ray of light falling on the blind half of either retina.

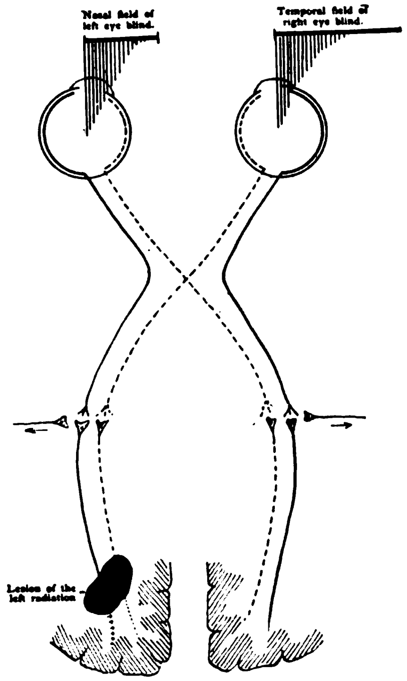

Nasal field of left eye blind.
Temporal field of right eye blind.
Lesion of the left radiation

Fig. 141.—A diagram showing how a lesion of the left optic radiations or of the visual portion of the left occipital cortex causes blindness of the right half of the field of vision of each eye, but does not prevent the pupils from reacting in response to a ray of light falling on the blind half of either retina.

Bilateral Temporal Hemianopsia.—There is only one spot at which a single lesion can produce this condition ; this is at the central part of the optic chiasma, where the fibres from the nasal half of each eye are decussating. The three commonest causes of this rare lesion are: (*a*) Hypertrophy of the pituitary body, a condition which also leads to acromegaly, so that it is important to test for bilateral temporal hemianopsia in every case of acromegaly, and it will be found in a certain number; (*b*) Callus, resulting from a fracture of the base of the skull through the basi-sphenoid bone; (*c*) A gumma or other tumour in this region. The differential diagnosis between these three groups will generally be obvious enough when the variety of hæmianopsia has been established.

In order to decide the locality of the lesion, it is essential in the first place to determine whether a pencil of light falling upon that part of the retina which cannot see is able to evoke a reflex contraction of the pupil. This requires careful testing in a dark room, with a small pencil of light directed towards different portions of the eye at the observer's will by a suitable mirror or lens. Anatomical considerations make it obvious that if the optic tract is destroyed there is no path by which the light impulses from the non-seeing portions of retina can reach the oculomotor nucleus, so that there will be no reflex movement of the pupil in response to light (***Fig.*** 140). If, on the other hand, the optic tract is intact, the lesion being in the posterior limb of the internal capsule, or in the optic radiations or the cuneus, the same hemianopsia results, but the pupils react to light stimuli falling upon the blind halves of the retinæ (***Fig.*** 141).

If the light reflex is lost the lesion is at once located to the optic tract, provided there is no obvious trouble, such as cataract, or locomotor ataxy, or iritic adhesions, to prevent the reaction. If, on the other hand, the light reflex remains, the lesion must be in one of the three other places mentioned, and in determining this the history may help considerably. If there has been an apoplectic seizure in an elderly person, hæmorrhage in the region of the internal capsule is likely, and there will often be both hemiparesis and hemiparæsthesia at the same time. In a younger person suffering from heart disease, a somewhat similar history would point to embolism involving the posterior limb of the internal capsule. If, on the other hand, there has been a slow onset, with increasing headache, vomiting, and giddiness, then a neoplasm or gumma affecting the optic radiations or one occipital pole will be not unlikely.

If the patient is unable to see things in the right halves of his fields of vision, the lesion will be in his left optic tract, left internal capsule, left optic radiations, or left cuneus, as the case may be, and vice versa.

Hemianopsia due to migraine or to intermittent closure of cerebral vessels will be distinguished from that due to the other causes by its presence on some occasions and its absence on others.

Irregular or partial forms of hemianopsia result from irregular or partial lesions in the optic tract or other regions mentioned above. The differential diagnosis is then more difficult, though it is made upon the same lines as those described above. From a diagnostic point of view it is fortunate perhaps that hemianopsia, when it occurs at all, is generally definite, and either bilateral temporal or bilateral homonymous.

Herbert French.

HEMIPLEGIA signifies loss of motor power in the limbs of one side; the face, especially its lower half, being affected frequently at the same time. In the great majority of cases the face is paretic on the same side as the affected arm and leg, but there is one important exception, namely, when the lesion is in one side of the pons Varolii, when there is paralysis of the face upon the same side as the lesion, and of the arm and leg upon the opposite side—a condition known as *crossed hemiplegia.* The lesion in most cases, however, is in or near the internal capsule, less often in the motor cortex, of the opposite side to that which is hemiplegic. There may or may not be hemianæsthesia (p. 610); and in rare cases, when the lesion is far back in the internal capsule, there may also be HEMIANOPSIA (p. 300). When the cause lies in the internal capsule, the paralyzed muscles may be either flaccid or spastic, but they do not as a rule exhibit the athetotic and other involuntary movements that cortical lesions may give rise to (see CONTRACTIONS, p. 131). When a patient has difficulty in speech associated with hemiplegia, it is important to distinguish dysarthria from aphasia (see SPEECH, ABNORMALITIES OF, p. 626). Lesions of the internal capsule often produce difficulty in using the tongue, which renders speech mechanically difficult (dysarthria)—a very different thing from the aphasia or difficulty in uttering the correct words when the mechanism for the movements of the tongue is unaffected. True aphasia associated with hemiplegia suggests a lesion at, or close to Broca's area of the cortex on the left side, and is therefore far less common with left-sided than with right-sided hemiplegia.

The fact of hemiplegia is generally not difficult to determine, though in some cases there may be so slight a weakness that doubts arise as to whether there is any hemiplegia at all. Routine examination of such a patient will generally detect a little inequality in the degree to which the eyes can be closed firmly, a slight difference in the depth of the two naso-labial folds when the patient opens his lips with his teeth clenched, a greater difference than previously between the two hand-grips, as measured by the dynamometer, slightly brisker radial and ulnar wrist-jerks, or tricipital and bicipital elbow-jerks upon the affected side, inequality of the knee-jerks with a tendency to exaggeration upon the paretic side, with corresponding extensor plantar reflex and increased Achillis jerk or even ankle-clonus. All these changes will be pronounced in cases where the hemiplegia is more definite, though if the patient be seen within a short time of the onset of hemiplegia from cerebral hæmorrhage, the tendon and other reflexes—which will presently be exaggerated should the patient survive—may for the time being be decreased or even unobtainable upon the affected side.

Stress is often laid upon the presence or absence of rigidity in connection with hemiplegia, particularly according as the rigidity comes on early or late in the case. This helps less, however, in the diagnosis than it does in the prognosis. A few cases of hemiplegia are flaccid throughout, though this is uncommon if the patient survives and the hemiplegia persists; in cases of hemiplegia due to cerebral hæmorrhage, early rigidity generally suggests a smaller hæmorrhage than does early flaccidity followed by rigidity; so variable is this, however, that the point is of less value than has sometimes been supposed.

It is difficult to classify the causes of hemiplegia satisfactorily, but the following is a summary of those discussed:—

A.—THE COMMONER CAUSES OF HEMIPLEGIA.

1. **Hemiplegia of Moderately Rapid Onset.**

Cerebral hæmorrhage
Thrombosis of a middle cerebral artery
Syphilitic endarteritis of a middle cerebral artery.

2. **Hemiplegia of Sudden Onset.**

Embolism of the middle cerebral artery, generally due to mitral stenosis, or to fungating endocarditis.

3. **Hemiplegia dating from Birth,** or from infancy, and resulting from:—

Injury
Congenital malformation
Acute encephalitis
Sinus thrombosis
Meningococcal meningitis.

B.—LESS USUAL CAUSES OF HEMIPLEGIA.

General paralysis of the insane
Borderland sufficiency of the cerebral circulation in old people (intermittent claudication)
Cerebral tumour, with or without hæmorrhage into it
Cerebral abscess
Hemichorea
Stab or bullet wound injuring the spinal cord in the cervical region
Meningitis, whether tuberculous, suppurative, posterior-basal, or epidemic cerebrospinal
Disseminated sclerosis
Caisson disease
Hysteria.

Granted that a patient is suffering definitely from hemiplegia, the exact cause of the symptom has to be determined. One may say at once that the diagnosis is easy in a very large proportion of cases. Hemiplegia of moderately rapid onset in a patient over fifty years of age is almost certainly due to *cerebral hæmorrhage*, particularly when it is associated with coma (p. 117) of rapid but not instantaneous onset, when there is a high blood-pressure and enlargement of the heart, with a ringing aortic second sound, with or without albuminuria or other evidence of granular kidney or arteriosclerosis. If the hemiplegia has been of gradual onset in a young adult, particularly if one limb is very much more affected than the rest of that half of the body, if there had been premonitory symptoms for some hours, or even days, before the paresis became marked, and if there has been no loss of consciousness, the great probability is that the patient is suffering from *syphilitic endarteritis* of the middle cerebral artery, with or without secondary *thrombosis*. The diagnosis may be confirmed by a history of syphilis, by the occurrence of cutaneous ulcers or other syphilitic lesions, or by a positive Wassermann's serum reaction.

If the patient is young, if the hemiplegia has been of absolutely sudden onset, generally without, but sometimes with, loss of consciousness, the probability of *embolism of the middle cerebral artery*, secondary to *mitral stenosis* or to *fungating endocarditis*, will be considerable, and the diagnosis will generally be confirmed by physical examination of the heart, and by inquiry into the history as regards acute rheumatism, chorea, or other rheumatic affections. In cases of fungating endocarditis one would look for the signs described on p. 34.

If the patient has been hemiplegic from birth or from early infancy, the probability is that there has either been *an injury* to the opposite side of the brain at birth, or *congenital malformation* of that side, or acute inflammation of it after birth—the result perhaps of *acute encephalitis*, *sinus thrombosis*, or even *meningococcal meningitis* which has recovered. It is particularly in these infantile cases that hemiathetosis is liable to be associated with the hemiplegia.

Although the above are by far the commonest causes of hemiplegia at the different age-periods, it is possible for them to overlap as regards age incidence; and one occasionally sees fatal cerebral hæmorrhage, apparently of the senile type, in persons not much over twenty; similarly, syphilitic thrombosis of the middle cerebral artery may not occur until after fifty; fungating endocarditis followed by cerebral embolism may occur at any age, though it is commonest in young persons; the same applies to cerebrospinal meningitis. The diagnosis will be indicated, if at all, by other symptoms than the hemiplegia. In doubtful cases assistance may be derived from lumbar puncture and analyses of the cerebrospinal fluid: the following are some of the main points in which the latter may differ from the normal under various pathological conditions:—

Appearance.—Cerebrospinal fluid is normally quite clear and free from colour, so that in a test-tube it may be difficult to distinguish it from water; when there are inflammatory changes in the central nervous system, particularly in all the acute forms of meningitis, the fluid becomes opalescent, turbid, purulent, or even fibrinous; and, instead of being colourless, it may develop a yellow or reddish-brown colour—when coagulable proteid is also present the combination has been termed the *xantho-proteic reaction*.

Specific Gravity.—Its normal specific gravity is low, lying, as a rule, between 1·004 and 1·007. It may retain a normal specific gravity even in diseased conditions, for instance, in cases of general paralysis of the insane; but with inflammatory changes, such as meningitis, the specific gravity is liable to increase.

Tension.—Normally the fluid drops out through the lumbar-puncture needle at the rate of 60 drops per minute. If it exudes at a lower rate than this no definite deduction can be drawn; but if the rate of outflow is higher than one drop per second, it indicates a condition of hypertension due to disease such as meningitis, cerebral tumour, hæmorrhage, or abscess.

Reaction.—Cerebrospinal fluid, normal or abnormal, is always alkaline.

Cryoscopy.—The normal freezing point of the cerebrospinal fluid is $-0{\cdot}55°$ C.; in disease it may be either above or below this; generally speaking, the greater the diminution in the freezing point the more likely is acute organic disease to be present in the central nervous system.

Sugar.—The amount of reducing substance in normal cerebrospinal fluid, estimated by the reduction of Fehling's solution, is approximately 1·5 parts per 1000; in diabetes mellitus this is more or less increased; what the figures are in other conditions has not yet been established fully, but there is some evidence to show that the sugar is materially decreased in dementia præcox.

Urea.—Urea in cerebrospinal fluid amounts normally to 0·15 parts per 1000; the disease in which there is any material increase in this is uræmia, and the excess of urea in cerebrospinal fluid in this condition is sometimes an important point in the differential diagnosis in cases of coma.

Proteids.—There is little if any coagulable proteid in normal cerebrospinal fluid; careful analyses have shown that no albumin is present, but that there are traces of globulin ; in diseased conditions, particularly those associated with inflammation within the cranium or spinal canal, there are albumin, more globulin than normal, and often some nucleo-proteid.

Choline.—Some stress was laid at one time upon the supposed fact that choline platino-chloride crystals were obtainable from the cerebrospinal fluid when acute nervous degeneration was taking place, and not in health : but the tests employed were unreliable, and the general opinion now is that deductions drawn from analyses for choline are erroneous, even when the choline periodide crystals are tested for instead of the platino-chloride.

Cytological Examination.—The normal fluid is practically free from cells, although, owing to the impossibility of avoiding slight injury to vessels by the introduction of the lumbar-puncture needle, a few red corpuscles are generally found in the centrifugalized deposit, and a few leucocytes corresponding to the numbers that would be expected in the blood represented by the red cells. It is probable that cerebrospinal fluid obtained quite free from blood contamination would be free from leucocytes. Quite otherwise is it in certain diseases—not only in acute lesions, such as meningitis, but also in chronic degenerations, such as general paralysis of the insane. It is important to examine the centrifugalized deposit, not merely for the presence or absence of leucocytes, but also for the different relative proportions of polymorphonuclear cells and of lymphocytes. A considerable number of polymorphonuclear cells generally indicates bacterial infection of the subarachnoid space by some organism other than the tubercle bacillus, especially streptococci, staphylococci, pneumococci, and meningococci. Some degree of polymorphonuclear excess may, however, accompany the characteristic lymphocytosis of a few cases of tuberculous meningitis. Mononuclear proliferation—lymphocytosis—indicates, as a rule, a subacute or chronic inflammatory or degenerative condition ; it almost invariably accompanies syphilitic lesions of the central nervous system, particularly general paralysis and tabes dorsalis ; it is also to be expected in tuberculous meningitis, and in sleeping sickness. It is not, however, pathognomonic of any of these, for it has been observed also in entirely different conditions, such as herpes zoster, acute anterior poliomyelitis, some cases of cerebral tumour, lymphatic leukæmia, chloroma, and even mumps. Although lymphocytosis generally indicates chronic mischief, and polymorphonuclear leucocytosis acute infection, in the later stages even of acute microbial infections mononuclear cells may be more numerous in the cerebrospinal fluid than are the polymorphonuclears. In a few cases of new growth, especially sarcoma, affecting the spinal cord or its meninges the diagnosis has been suggested by the discovery of large atypical cells in the fluid obtained by lumbar puncture.

Bacteriological Examination.—Normal cerebrospinal fluid is absolutely sterile. In pathological conditions it may be examined bacteriologically in various ways, including direct staining of films made from the centrifugalized deposit, cultural methods, and inoculation into animals. The most important organisms that have been found are the pneumococcus, streptococcus, bacillus tuberculosis, meningococcus (Weichselbaum's *Diplococcus intracellularis meningitidis*), pneumobacillus, staphylococcus, bacillus typhosus, bacillus influenzæ, spirochæta pallida, and, probably as a terminal infection only, the *Bacillus coli communis*. The cerebrospinal fluid may be used for testing for Wassermann's reaction for syphilis in the same way as is blood serum ; the test is not necessarily positive in the former when it is in the latter, but when the cerebrospinal fluid itself gives a positive reaction, there is almost certainly active syphilitic disease of the nervous system. It is important to know that a negative Wassermann reaction in the blood does not exclude syphilis of the nervous system—the blood is negative in not a few cases of tabes dorsalis, for example, even when no antisyphilitic remedies have been employed. The *Treponema pallidum* (*Spirochœta pallida*) has been found in the cerebrospinal fluid, but it is more likely to be detected in the local syphilitic lesions. The only protozoon at all constantly met with in the cerebrospinal fluid in disease is the *Trypanosoma gambiense* in cases in which the trypanosomiasis has reached the stage of sleeping sickness.

Amongst the less usual causes of hemiplegia it is worthy of particular mention that *general paralysis of the insane* sometimes attracts little or no attention until a seizure of some kind occurs, this seizure not infrequently being epileptiform, and sometimes producing a hemiplegia closely simulating that due to cerebral hæmorrhage. The diagnosis may remain uncertain until the course of the case can be followed, but Wassermann's serum reaction, and the lymphocytosis in the cerebrospinal fluid, may each serve to point to the true nature of the case. Another feature is the very rapid rate of temporary recovery exhibited by some patients: deeply comatose and hemiplegic when seen upon the day of seizure, nearly all the symptoms may have disappeared by the next morning in a way that would be unusual were they due to a hæmorrhage of sufficient size to cause so deep a coma.

In elderly people, incomplete hemiplegia may occur rapidly but transiently over a period of years, in such a way as to suggest during the first attack or two that there has been an actual extravasation of blood within the brain. The rapidity with which the hemiplegic symptoms may disappear, and the way in which they may recur and yet disappear again each time, render it probable that these patients are not suffering from the effects of recurrent small hæmorrhages, but from a condition of partial occlusion of their cerebral vessels by atheroma to such an extent that, whereas the circulation is just sufficient for the needs of the brain at one time, it is just insufficient at other times; the result being that when the insufficiency of cerebral circulation is most in evidence, weakness of a hemiplegic type ensues, to disappear when rest in bed restores the cerebral circulation to a sufficiency again. Cases of this kind have been spoken of as suffering from *intermittent claudication*, as though the vessels could alternately dilate and close up spontaneously; but there is evidence to show that there are no efficient vasomotor nerves in the cranial vessels, so that the theory of *borderland sufficiency of circulation* through atheromatous vessels is more probable than that of intermittent claudication.

Cerebral tumour or *cerebral abscess* may produce hemiplegia by infiltrating either the cerebral cortex or the pyramidal tract directly, or by these becoming involved in the softening around the tumour or the abscess; in most cases there will be a history of weeks or months of headache, giddiness, and effortless vomiting, with or without signs of irritation previous to the paralysis; ophthalmoscopic examination will frequently reveal optic neuritis of the choked disc type (*Plate XIX*, *Fig. k*, p. 416), and in the abscess cases there will generally be a predisposing cause, particularly otitis media. It is well known, however, that either a tumour or an abscess within the cranium may be latent for months, and in some such cases symptoms may come on acutely, especially if there has been hæmorrhage into a softening tumour. Ordinary cerebral hæmorrhage may be simulated in this way, but if well-marked optic neuritis is found in both eyes, it is probably not a hæmorrhage only. The existence of pyrexia is not by itself evidence of abscess, for hæmorrhage near the internal capsule, or in the motor cortex, often leads to some rise of temperature for the time being, whilst pontine hæmorrhage is not infrequently associated with hyperpyrexia, and in not a few cases of intracranial abscess pyrexia is conspicuously absent.

Injury to the Spinal Cord in the Cervical Region is a very rare cause of paralysis of the arm and leg upon the same side; first, because trauma here is extremely liable to damage more than half the cord; and, secondly, because the injury must involve the lower part of the cervical enlargement if the arm is to be paralyzed, and it is, therefore, very liable indeed to interfere with the subsidiary respiratory centres, and thus prove rapidly fatal. Occasionally, however, either a knife stab or a bullet wound on one side of the neck produces hemiplegia with evidence of unilateral paralysis of the diaphragm as observed when the patient's abdominal respiratory movements are watched in a good light. It has sometimes been asserted that the patient will have anæsthesia, not of the same, but of the opposite side of the body: in practice this is not generally the case, the hemiplegia and the hemianæsthesia being on the same side as the lesion in at least some instances.

Children of a rheumatic tendency, who are subject to *chorea*, sometimes present the movements of the latter upon one side of the body only—*hemichorea*; both before the actual movements appear and after they have ceased there is apt to be considerable, and occasionally extreme, weakness of the affected side; so much so that some intracranial lesion may be suspected, unless there has been clear evidence of the existence of chorea.

Occasionally, weakness of a hemiplegic nature may be the first symptom of *meningitis*, whether tuberculous, suppurative, posterior basal, or epidemic cerebrospinal; sometimes, upon post-mortem examination a definite unilateral softening, or a tuberculous nodule affecting the pyramidal fibres, may be found to account for this; but more often the appearances seen after death fail to explain why there should have been unilateral paretic symptoms. In the earlier stages the diagnosis may be quite obscure, but sooner or later the paresis becomes bilateral, and the course of the disease indicates meningitis beyond doubt, especially if there are convulsions, vomiting, and optic neuritis. Choroidal tubercles may be detected in some cases (*Plate XX*, *Fig. w*, p. 418), and the cerebrospinal fluid may be examined cytologically and bacteriologically.

Disseminated Sclerosis is a very slowly progressive disease, in which during the earlier stages the foci of sclerosis are few and quite irregularly distributed, so that whereas in the later stages ataxy, intention tremor, more or less spasticity with increased knee-jerks, extensor plantar reflexes, ankle-clonus, and either slurred or staccato speech, are to be expected, these are only present when, in the course of years, numbers of sclerotic foci have accumulated in the spinal cord and brain ; long previous to this there have been irregular symptoms, amongst which may be hemiplegia ; the diagnosis at this stage is often a matter of opinion only, though if the patient can be watched over a sufficient length of time the nature of the case ultimately becomes obvious.

The symptoms of *caisson disease* are due to the liberation of air bubbles in the nervous system, and what the symptoms will be depends on where these bubbles are ; in most instances they are widely scattered, so that bilateral paralyses are more common than unilateral ; it is possible, however, for caisson disease to produce hemiplegia if a relatively large air bubble becomes liberated in or near the internal capsule. The diagnosis depends on the history and occupation.

Hysteria may be responsible for almost any form of nerve symptom, hemiplegia being not an uncommon variety. There is no wasting, except that which may be due to disuse ; the knee-jerks may be exaggerated, but the plantar reflexes will remain flexor, and there is no ankle-clonus ; the face, as a rule, is unaffected ; if the patient, lying flat upon her back, is asked to raise her legs from the bed, she will raise the sound leg, but not that which is paretic ; whereas, in a case in which there is incomplete paralysis of one leg due to organic lesions of the upper neuron upon one side, an attempt to raise the leg in this way often leads to the paretic leg being lifted as well as the other. The sex and age of the patient, her previous history, and the presence possibly of other functional nerve symptoms (p. 465), would indicate the diagnosis. *Herbert French.*

HICCOUGH is a symptom which more often than not has no clinical significance, resulting, as it does even in the healthiest people, from excessive laughter, from stimulation of certain reflex spots, especially about the chin, from tickling, or even coming on spontaneously without any obvious cause at all.

Occasionally, however, hiccoughing may be so persistent, or may reach so alarming a degree, that it becomes of clinical importance. The patients may be divided into two groups, namely, those in whom there is already severe illness, and those who are not obviously ill. Of the former group there are two main types—the **Alcoholic** and the **Peritonitic.** No difficulty of diagnosis arises between these two ; the drunken person's hiccough has a character of its own. The patient who has an acute abdominal condition associated with hiccough will have presented grave symptoms long before hiccough sets in, the diagnosis often having been arrived at by urgent laparotomy. Hiccough in these cases does not serve to distinguish between acute peritonitis due to whatever cause, acute hæmorrhagic pancreatitis, acute intestinal obstruction from any cause, or acute post-operative dilatation of the stomach ; its occurrence and persistence, however, indicate a very grave prognosis.

When persistent or recurrent hiccough is a troublesome symptom in a patient who is not obviously ill—so troublesome that something more than a simple hiccough has to be thought of—three main types of malady will suggest themselves, namely :—

1. Hysteria or neurosis.
2. Mediastinal irritation of vagi or phrenic nerves, e.g., by caseous glands.
3. Degenerative changes in the medulla oblongata.

None of these three types is at all common, and their diagnosis during life is often a matter of opinion.

Functional Hiccough is a remarkable malady hardly to be mistaken. The patient is generally a girl between 15 and 25 years of age, and she may hiccough persistently throughout her waking hours for weeks, at the rate of two or three times a minute. She will sleep well, and the hiccough stops during sleep. She will eat well, but may hiccough during meals in a most distressing way. She will have exaggerated knee-jerks, brisk flexor plantar reflexes, and she will be amenable to treatment by suggestion. Whether treated or not, the hiccough will cease in time, though it may persist on and off for weeks; often it will be noticed to have come in the place of some other neurosis (p. 465), and when it goes it may be replaced by other functional nerve symptoms.

Irritation of a Vagus or **Phrenic Nerve** by something in the mediastinum causes recurrent attacks of intractable hiccough only in rare cases. In a child, the least uncommon cause is *tuberculous caseation of bronchial* and *mediastinal glands*; these seldom obstruct a bronchus or in other mechanical ways afford evidence of their presence; but they may be associated with periodic attacks of febrile illness in a patient who looks delicate, and who has been in the habit of drinking much milk; and there may be evidence of chronic enlargement of the glands elsewhere, particularly those in the neck or in the abdomen. It may be possible to see the shadow of the gland with the *x*-rays (*Fig.* 61, p. 149). *Adherent pericardium*, or any cause of great *enlargement of the heart*, may also produce hiccough. In an adult the least uncommon causes are either *malignant* or *lymphadenomatous deposits* in the mediastinum, or else *fibrous mediastinitis*. The former may be indicated by reason of there being symptoms of a primary growth in the œsophagus or elsewhere, or by progressive varicosity of the veins of the chest wall, or signs of recent and increasing obstruction to a bronchus; chronic mediastinitis has generally been preceded by repeated attacks of pleurisy and pericarditis, especially in those subject to acute rheumatism. Hiccough is an exceptional symptom in these cases.

Uræmic Hiccough is rare, but it may be persistent and of grave omen. A few patients suffering from serious but not urgent abdominal disease develop distressing hiccough in some reflex way that is not understood; in a few instances it may be that the diaphragm is being irritated, for example by secondary deposits of *cancer in the liver*, a *gumma* or *abscess in the liver*, an *infarct in the spleen*, or a *carcinoma of the stomach*; but sometimes the mischief seems far removed from the diaphragm—a *carcinoma of the sigmoid colon*, for instance, or *cancer of the womb*, even when there are no secondary deposits.

Finally, if hiccough is due to *degeneration* or *softening of the medullary centres*, it will almost certainly be associated with other symptoms of cerebral or spinal mischief; in a young adult there might be a suggestive history of syphilis or chronic alcoholism, whilst in an older person there would be thickened and tortuous arteries, a high-tension pulse, an enlarged heart, arcus senilis, possibly albuminuria in an abundant urine of low specific gravity—signs of senile degenerative changes.

Herbert French.

HIPPUS.—(See Pupil, Abnormalities of the, p. 551.)

HUSKINESS.—(See Speech, Abnormalities of, p. 623).

HYPERACUSIS, or undue sensitiveness to ordinary noises or sounds, is seldom a symptom pointing to disease of the ear itself; aural lesions such as otitis media nearly always cause impaired hearing or actual Deafness (p. 163), and not hyperacusis. The latter is complained of rather by those whose brain centres are in an unstable, irritable, over-strained, or excitable state as the result of either local or general conditions which include the following:—

1. **Local Causes**:—

(*a*). *With gross lesions of the brain itself.*

Tuberculous meningitis }
Meningococcal meningitis } Early stages
Suppurative meningitis }
Pachymeningitis

Gumma of the brain
Abscess of the brain
Tumour of the brain.

(*b*). *Without gross lesions of the brain itself, but affecting the latter reflexly from the severity of local pain*:—

Tic douloureux
Glaucoma
Iritis
Inflammatory conditions of the scalp

Severe headache from any cause (p. 293), especially sick headache or migraine.

(*c*.) *After injury to the head—concussion.*

2. **General Causes:—**

(*a*) During convalescence from any severe illness, especially fevers.

(*b*) Strychnine poisoning
Tetanus
Malaria
Secondary syphilis
High blood-pressure conditions
Neurasthenia
Hysteria
Hypochondriasis
Graves's disease.

The circumstances of the case will nearly always indicate the nature of the cause. Probably the most marked instances of hyperacusis are met with in association with tic douloureux; it is not so much perhaps that the patient hears more acutely than usual, as that she dreads the onset of a paroxysm of facial pain which may be brought on suddenly and acutely by almost anything, and often by hearing a door bang, or somebody talking loudly; she therefore complains of the least noise, and keeps herself shut up in her room, from which all sounds are excluded by all kinds of special devices.

The neurasthenic patient who suffers from hyperacusis is to be pitied greatly; for though suffering from no serious organic disease, he dreads all sounds so much that he becomes an almost useless member of society, a misery to himself and his friends. His functional sufferings may be so bad as to drive him to desperation and to suicide.

The desire for perfectly silent surroundings during an attack of sick headache or of migraine is familiar to all. The remaining affections in the list above need not be discussed in detail here, for they will be associated with other symptoms that will point to the differential diagnosis—Convulsions (p. 143) for example; or Eye, Acute Inflammation of (p. 231), or Vomiting (p. 763), and so on. One would add, however, that before diagnosing a case as purely functional, the blood-pressure should be measured instrumentally, and the urine tested for albumin, in case the cause is arteriosclerosis or chronic nephritis with hyperpiesis; and in not a few cases it is advisable to have the blood tested for Wassermann's reaction, for it is surprising how often obscure nerve symptoms that at first sight appear purely functional really have a syphilitic basis. *Herbert French.*

HYPERIDROSIS.—(See Sweating, Abnormalities of, p. 654.)

HYPERPYREXIA.—The point at which pyrexia becomes hyperpyrexia is arbitrary; by some it is fixed at 105° F., by others at 106° F. It may occur occasionally in many different diseases, but it is seldom itself of diagnostic significance. The patient will nearly always have exhibited other symptoms or signs pointing to the diagnosis; therefore the following list of maladies in which hyperpyrexia may occur needs little discussion:—

A. **Fevers of Microbial, or probably of Microbial, Origin:—**

Lobar pneumonia
Bronchopneumonia
Scarlatina
Pyæmia
Septicæmia
Erysipelas
Typhoid fever
Typhus fever
Tetanus
Malignant endocarditis
General tuberculosis
Tuberculous meningitis.
Posterior basal meningitis
Epidemic cerebrospinal meningitis
Suppurative meningitis
Malaria
Relapsing fever
Cholera
Dysentery
Yellow fever
Rheumatic fever
Chorea insaniens
Uræmia due to ascending nephritis
Pyelitis.

B. **Lesions of the Central Nervous System:—**

Cerebral hæmorrhage, especially pontine, or into one optic thalamus
Fractured skull, with contusion of the brain
Cerebral softening
Cerebral tumour or abscess, especially tumour of the pons Varolii
Fractured spine, especially in the lower cervical or upper dorsal regions
Acute myelitis after injury.

C. **Affections that are less easy to classify:—**

After burns or scalds
Heat stroke or sunstroke
Infantile convulsions
Delirium tremens
Uræmia other than that due to septic nephritis
Acute yellow atrophy of the liver.

D. **Hysteria.**

There are, however, certain small points about hyperpyrexia that may be important in diagnosis.

Acute rheumatism is often stated to be a prominent cause of it; as a matter of fact, hyperpyrexia is excessively rare in acute rheumatism, so that should it occur in a case that has been regarded as rheumatic fever, the diagnosis should be very carefully revised, lest it really be septicæmia.

In children the physical signs alone may leave one in doubt as to whether there is bronchitis only, or bronchopneumonia, or even general tuberculosis of the lungs; the occurrence of hyperpyrexia generally indicates that there is more than bronchitis; if the patient is not particularly livid, bronchopneumonia is more likely than general tuberculosis; the latter becomes the more probable the more ill the patient is out of proportion to the physical signs. Occasionally hyperpyrexia occurs in an infant or child after a fit, without any definite cause being assignable either for the convulsion or for the high temperature, and without any serious consequence resulting.

In *tuberculous meningitis* hyperpyrexia is generally terminal; in *posterior-basal meningitis*, on the other hand, it sometimes occurs periodically and transiently, producing acute upward 'spikes' upon a temperature curve that is not otherwise very high; these pyrexial 'crises' (*Fig.* 253, p. 591), as they have been called, point to posterior-basal rather than to the more serious tuberculous meningitis.

Hyperpyrexia may sometimes serve as the chief point in distinguishing *pontine hæmorrhage* or *heat-stroke* from other forms of coma, such as acute alcoholism or opium poisoning; in the latter, the temperature is below normal. The circumstances of the case, such as climatic conditions or occupations, will generally serve to distinguish between heat-stroke and pontine hæmorrhage.

After an injury to the back—for instance, by a fall in the hunting field—the occurrence of hyperpyrexia sometimes serves to exclude the diagnosis of a mere bruising, and to point to the gravity of the conditions—a fractured or dislocated spine near the cervical region, or acute traumatic myelitis or softening of the upper part of the spinal cord.

The diagnosis of the other diseases mentioned in the above list is not much assisted by the occurrence of hyperpyrexia.

It only remains to add a word or two about *hysteria* and high temperatures. There can be no doubt that, in exceptional cases, nearly all of which are of the female sex, the mercury in the clinical thermometer does actually rise to a very high figure without there being any corresponding illness in the patient. Malingerers have sometimes learned a trick, such as compressing the bulb of the thermometer enough to send the mercury up; but quite apart from malingering, there are females in whom, for some reason that is not yet understood, the mercury really does record temperatures that are not those of the internal tissues. Readings have been taken simultaneously in the mouth, armpits, and rectum, all possibility of malingering being excluded by special precautions; all the thermometers registered hyperpyrexia. The diagnosis is generally made by the fact that the readings are so high that they must be unreal; the following have been recorded in various cases: 107°, 108°, 111°, 113°, 115·8°, 116·4°, 117°, 120·8°, 122°, 127°, 128°, and even 131° F. In most of these patients the symptoms were slight, though sometimes there have been flushings, headache, restlessness, and various functional nerve symptoms, or even delirium and convulsions. Unless it is at once obvious that the patient is not really ill, there must always be difficulty, danger, and anxiety in arriving correctly at the diagnosis of hysterical hyperpyrexia; the nature of the case may remain in doubt until the course and result have been watched. *Herbert French.*

HYPERTROPHY OF THE HEART.—(See ENLARGEMENT OF THE HEART, p. 206.)

HYPOTHERMIA signifies a condition of subnormal temperature, and generally speaking it is assumed to refer to the temperatures registered by the thermometer in the mouth. Rectal temperatures do not always coincide with those of the mouth, but the clinical significance of variations in rectal temperatures is not yet fully understood. From a diagnostic point of view hypothermia is not often a symptom of great importance, but there are at least two points about it that require special mention. In the first place, coma due to *opium poisoning* may be closely simulated by coma due to pontine hæmorrhage; in both there are bilateral loss of movement, pinpoint pupils, and few other symptoms; with opium poisoning, however, the temperature becomes subnormal, whilst with pontine hæmorrhage it tends to rise to the level of hyperpyrexia, so that the thermometer may be the means of diagnosing between them. In the second, patients suffering from *chronic valvular heart disease*, with symptoms of impending or actual failure of compensation, very commonly suffer from hypothermia. This is a point not always emphasized sufficiently; not a few cases of heart disease having for their normal temperature base-line not 98·4° F., but 97° F., or even 96° F. (*Fig.* 142). It follows that a patient whose normal temperature is 96° F. really has over two degrees of fever when his temperature reaches 98·4° F.; he may develop fungating endocarditis on the top of his chronic valve lesion, and yet his temperature may not rise materially above 98·4° F. The fact, therefore, that hypothermia is a common feature in heart cases has great importance, for it indicates the necessity for regarding even slight rises above 98·4° F. with greater seriousness in them than in other cases.

For the rest it will suffice to indicate the chief causes of hypothermia, which are as follows:—

Chronic Debilitating Maladies, such as:—

Chronic valvular heart disease
Addison's disease
Diabetes mellitus
Cretinism
Arteriosclerosis
Myxœdema
Chronic nephritis, with or without uræmia
Inanition, malignant or otherwise (*Fig.* 143).

Coma, due to poisons, particularly:—

Opium
Alcohol
Chloral
Anæsthetics
Carbolic acid
Oxalic acid.

Increased Intracranial Pressure in certain cases of:—

Cerebral abscess
Cerebral tumour
Cerebellar abscess
Cerebellar tumour
Cerebral hæmorrhage.

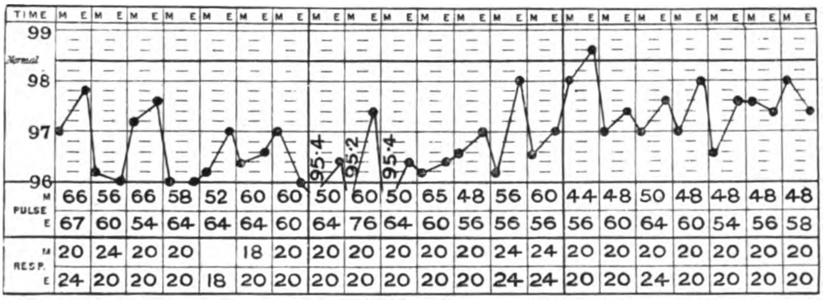


Fig. 142.—Temperature chart of case of chronic mitral stenosis, showing an ordinary degree of hypothermia.

These same lesions, especially if they involve either the pontine or the subthalamic regions, or if they affect the corpus striatum, may produce pyrexia or even HYPERPYREXIA (p. 309) instead of hypothermia, so that the inconstancy of the latter symptom detracts considerably from its value in differential diagnosis in these cases.

Convalescence after certain fevers; for instance:—

Pneumonia
Typhoid fever
Relapsing fever.

Shock after **severe injury** or after a serious **operation.**

Collapse due to loss of fluid from the tissues from such conditions as:—

Severe vomiting, whatever the cause
Severe diarrhœa, choleraic or otherwise
Peritonitis
Intestinal obstruction
Hæmorrhage.

Exposure, especially in the case of a child.

In the morning, in cases of Intermittent Pyrexias of the septic or hectic types. It is important that the temperature should have been taken both night and morning before the low figures for the morning are assumed to indicate continued hypothermia; very low readings in the earlier part of the day in a patient whose malady is not at once obvious may arouse suspicion of a tuberculous lesion which further investigation may confirm.

It is a point of clinical interest that, although the human normal temperature is upon the whole remarkably constant all over the world, *Anglo-Indians* and others who have resided long in the Tropics are frequently found to have persistently low normal mouth temperatures when they return to England on retirement; thus it is quite common for them to have a normal temperature as low as 96° F., or in a few instances even 94° F.

Herbert French.

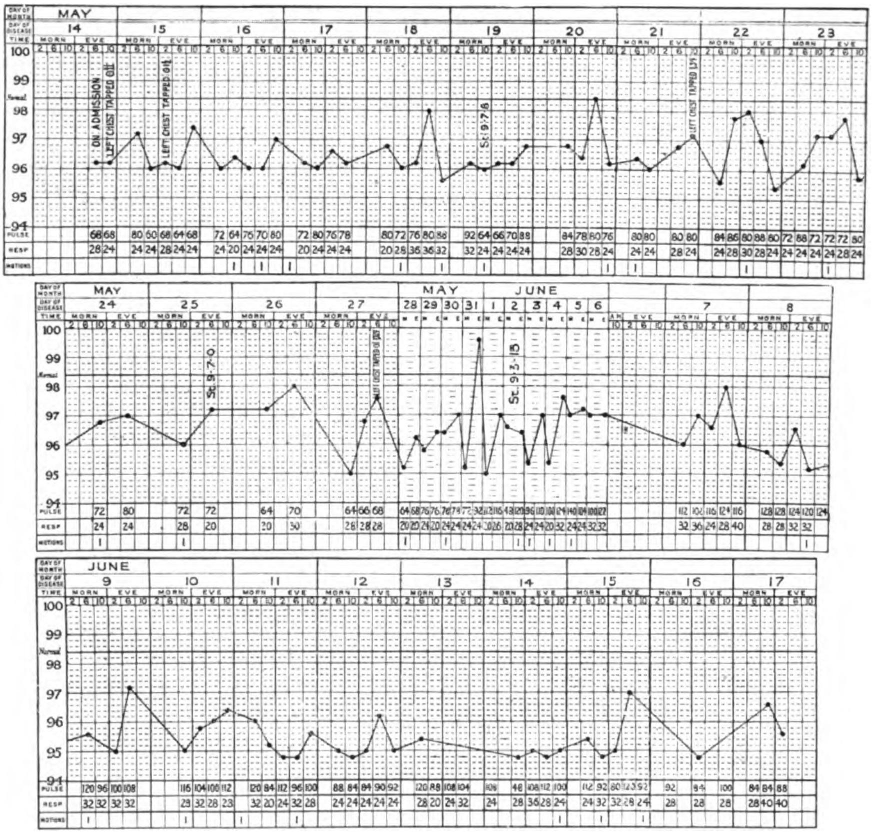


Fig. 143.—Temperature chart illustrating hypothermia in cachexia from malignant disease. The patient, a man aged 74, had a mediastinal sarcoma, with recurrent effusion into left pleural cavity.

ICTERUS.—(See JAUNDICE, p. 324.)

IMPOTENCE, strictly speaking, includes any condition, whether in the male or in the female, that prevents the performance of coitus; by common consent it has come to be restricted to inability on the part of the male. It is not synonymous with sterility; the latter, in the male, implies absence of the spermatozoa necessary to fecundation; a man may be sterile without being impotent, or impotent without being sterile, or both impotent and sterile. There are three main groups of conditions which lead to impotence, namely :—

1. **Mechanical Defect,** such as congenital or acquired malformation of the penis, absence of the penis, carcinoma, elephantiasis, and so on. These need not detain us, for their diagnosis is generally obvious on inspection; one need only say that even considerable deformities of the genital organs are by no means necessarily associated with impotence.

2. **Entire Absence of Penile Erections:** as the result either of some organic disease

of the nervous system, or of some general constitutional condition; one may mention particularly the following:—

Tabes dorsalis
Ataxic paraplegia
General paralysis of the insane
Primary spastic paraplegia
Disseminated sclerosis
Amyotrophic lateral sclerosis
Transverse softening of the cord
Plumbism
Compression of the lower part of the cord
Dementia
Diabetes mellitus
Atrophy of the testicles from injury or from severe orchitis, gonorrhœal or otherwise
Senility
Pernicious anæmia
Malarial cachexia
Syphilitic cachexia
Cancerous cachexia
Phthisical cachexia
Exhaustion from excesses.

There is little need to discuss these further here, for, providing they are borne in mind, they will be diagnosed readily as the result of a careful routine examination of the nervous system, urine, lungs, and so on. One need only add that impotence may be an early symptom in ataxic paraplegia, disseminated sclerosis, and phthisis, and that the diagnosis may seem to be neurasthenia only until the case has been watched.

3. Impotence due to Inability to obtain Erections at the right time.—This is a very common form of the symptom; the patients are generally told they are suffering from neurasthenia; and so they are, of a particular sort. This is psychical or nervous impotence: strong erections may be present at inopportune times, there may be emissions during sleep, and yet at the very moment when sexual intercourse is intended the erection is either quite absent or imperfect. Sometimes, owing to extreme irritability, emission occurs on so little excitation that it takes place before insertion is complete, the rigidity of the penis relaxing almost at once, so that completion of coitus becomes impossible. Temporary impotence of this kind is not at all uncommon during the first few days or weeks of married life, especially if the wedding has been preceded immediately by particularly hard business strain or mental overwork preparatory to the honeymoon. The diagnosis is arrived at partly by the history, partly by the negative result of careful physical examination of all the systems, especially the nervous and pulmonary; early phthisis is often accompanied by inability to obtain penile erection; but the final criterion is the effect of time. Where there is no organic cause for the symptom, normal coitus will occur presently if the patient ceases to be over-anxious about it. *Herbert French.*

IMPULSE, DISPLACED CARDIAC.—(See HEART IMPULSE, DISPLACED, p. 297.)

INCONTINENCE OF FÆCES.—Evacuation of the contents of the rectum without voluntary control or initiation may occur under several distinct conditions, the investigation of which may yield results of great diagnostic importance.

In healthy persons the reflex relaxation of the sphincter ani which is necessary for defæcation takes place only at the bidding of the will. Some healthy persons are better able to resist an imperative call to stool than others, and it happens occasionally that an individual who is poorly endowed with the power of inhibiting the reflex may suffer from an incontinence of fæces when the stimulus evoked by irritating contents of the bowel is overpoweringly strong. The individual would be conscious of the accident, which would be of rare occurrence, and examination would reveal no abnormality. Children often suffer in this way. Somewhat similar 'explosive diarrhœa' is also a prominent feature of certain cases of carcinomatous stricture of the sigmoid colon.

Mechanical incontinence of fæces results from injuries or diseases of the rectum or perineum, such as carcinoma, in which the outlet of the bowel is no longer guarded by an efficient sphincter. Local inspection and digital examination of the parts will suffice to discover the cause of such incontinence.

In conditions of coma or partial unconsciousness, from whatever cause arising, reflex emptying of the bowel may occur at intervals, particularly if aperients are administered. Digital examination of the rectum in such cases will reveal a normal sphincter which closes on the observer's finger.

Injuries or diseases of the central nervous system above the sacral region of the cord, if they interfere with impulses passing from the cortex to the lumbo-sacral enlargement, but do not cause serious sensory disturbance in the perineal area, lead to an unstable condition to which the term 'precipitancy of defæcation' is applied. In these circumstances the patient is usually constipated, but the call to stool, when it comes, spontaneously or as the result of aperient medicine, is imperative, and finds the patient powerless to resist or delay the act. The examination of such a person discloses a normal sphincter but, in all probability, some degree of spastic paraplegia, with brisk tendon jerks and extensor plantar responses, and inquiry will elicit the history of precipitate micturition. Moreover, the patient will be conscious of the acts of defæcation and micturition. This association of signs and symptoms is common enough in cases of partial injury to the spinal cord, in cases of old dorsal myelitis, of disseminated sclerosis, of syringomyelia, etc.

With more serious disease of the central nervous system above the sacral region, the impulses conveying the need for defæcation do not reach the brain, and the act takes place in a reflex manner without the knowledge of the patient. Under these circumstances, paraplegia with sensory disturbance over the sacral segmental areas will help to localize the site of the lesion. The tone of the sphincter ani may be little below normal or quite unimpaired.

Disease or injury leading to destruction of the sacral cord or of the cauda equina is distinguished by the fact that incontinence of fæces is associated with an *insensitive relaxed sphincter* and with serious motor, sensory, trophic, and reflex disturbances in the lower extremities. When the fæces are small and fluid they escape, more or less continuously, through the gaping anal aperture. On the other hand, they sometimes tend to accumulate in dry masses too large to pass the portal without assistance. The patient is unconscious of the accumulation, unconscious of soiling, and insensitive to the exploring finger.

It will be understood from the above statements that for the purpose of diagnosis it is necessary, in all cases of fæcal incontinence, not only to inquire into the exact features of the incontinence, the presence or absence of a call to stool, the tendency to constipation or precipitancy, the ability to feel the passage of motions, etc., but to supplement the knowledge gained in this way by a local examination, especially of the sphincter ani, and an investigation of the motor, sensory, and reflex conditions in the lower extremities.

E. Farquhar Buzzard.

INCONTINENCE OF URINE.—(See MICTURITION, ABNORMALITIES OF, p. 393.)

INCO-ORDINATION.—(See ATAXY, p. 55.)

INDICANURIA.—Indican in the urine is mainly due to the formation of indol in the intestine as the result of putrefactive changes in the products of tryptic digestion of proteids. The indol so formed is absorbed from the bowel, and converted in the liver into relatively innocuous potassium indoxyl sulphate, or indican. This is tested for by oxidizing it to indigo, the blue colour of which is characteristic. Almost any oxidizing agent could be utilized for the test, but the difficulty is that even slight excess of the reagent destroys the indigo. A brown ring appearing at the junction of the urine and the acid, when testing for albumin with nitric acid that is slightly fuming, generally indicates a considerable degree of indicanuria. To be certain of this, Jaffe's bleaching-powder test is usually employed. There are several ways of using it. Fresh solution of calcium hypochlorite is essential. To about 20 c.c. of urine add 3 c.c. of chloroform and 3 c.c. of hydrochloric acid of medium strength ; the colourless chloroform sinks to the bottom of the mixture ; a drop of the calcium hypochlorite solution is now added and the test tube deliberately inverted once or twice ; a second drop is added, and so on, the colour of the chloroform being watched the while. If indican is present, it becomes oxidized to indigo, which is dissolved out by the chloroform so that the latter changes from colourless to blue, and the depth of indigo-blue colour in the chloroform affords a rough measure of the amount of indican in the urine. The main precaution to be taken is, not to add the hypochlorite solution too rapidly, for excess of it discharges the colour.

Indican being an ethereal sulphate, it is present in excess under the same circumstances as an excess of ethereal sulphates. At one time it was thought that much useful clinical information as to the condition of the intestines could be learned from its occurrence. It is true that any circumstances that are likely to increase the putrefactive changes in the proteid in the bowel are also likely to increase the amount of indican in the urine ; marked indicanuria is generally found in cases of chronic constipation, intestinal obstruction, diarrhœa, typhoid fever, dilated stomach, peritonitis, acute enteritis or colitis, appendicitis, membranous, tuberculous or ulcerative colitis, acute and chronic dysentery, cholera, intussusception, and carcinoma coli. It affords no assistance in diagnosing between one and another of these various affections, however. Moreover, it may occur when there is decomposition of albumin elsewhere in the body than in the bowel; for instance, in gangrene of the lung, gangrenous empyema, putrid bronchitis, bronchiectasis, or advanced pulmonary tuberculosis. Another difficulty is that a considerable number of perfectly healthy individuals pass quite large quantities of indican in their urine. There are some who contend that even these healthy persons are really suffering from intestinal putrefactive changes without knowing it; this is possible, but from the patient's point of view it is tantamount to saying—what is indeed almost true—that indicanuria has no real diagnostic or clinical significance. *Herbert French.*

INDIGESTION.—It is important to remember that 'indigestion' is a symptom, and not a disease; and if a patient complains of this, one should inquire more particularly as to the exact nature of the abnormal sensations present, e.g., pain, fullness, flatulence, vomiting, etc. The diagnostic indications furnished by each of these symptoms is considered separately (see VOMITING, p. 763; PAIN IN THE EPIGASTRIUM, p. 436; FULLNESS, p. 243; FLATULENCE, p. 240, etc.), but it may be convenient here to offer some general guidance as to the methods of arriving at a diagnosis in cases in which 'indigestion,' in one or other of its aspects, is the chief complaint.

I. SIMULATION OF DYSPEPSIA BY OTHER CONDITIONS.

At the outset one should never forget that a patient may describe his case as one of indigestion, although he is not really suffering from any primary affection of the stomach at all; and mistakes can only be avoided by subjecting every such case to a thorough physical examination of all the organs, and not confining it to the abdomen. If the possibility of error is borne in mind, it is not usually difficult to avoid; and, accordingly, it will be sufficient to enumerate briefly the chief conditions to be thought of. These are:—

The Vomiting of Pregnancy.—The possibility of pregnancy should always be present to the mind when one is consulted by a young woman who complains of vomiting and indigestion, and the other signs and symptoms of pregnancy (p. 19) looked for.

Cerebral Vomiting.—In children, particularly, vomiting of cerebral origin may be mistaken for dyspepsia. Incipient meningitis and tumour are the commonest causes of such vomiting. The former, in its earliest stage, may be very difficult to diagnose with certainty, but the presence of signs of cerebral irritation (e.g. photophobia, squint, irritability, headache, Kernig's sign, etc.) should make one suspicious; paralyses, headache, and optic neuritis point to tumour. Examination of the cerebrospinal fluid obtained by lumbar puncture may be required (p. 304).

Uræmia may masquerade as 'indigestion,' characterized by loss of appetite and vomiting (uræmic gastritis). The 'uræmic odour' in the breath, high arterial tension, and albuminuria and albuminuric retinitis, should be looked for. It must be remarked, however, that albumin may be absent from the urine in undoubted cases of uræmia.

Phthisis.—In cases of early phthisis, indigestion may be the chief symptom of which the patient complains, nausea and vomiting being often present. This can be excluded by a careful examination of the chest and of the sputum, which should never be omitted, especially in young subjects.

The Gastric Crises of Tabes are apt to be mistaken for dyspepsia. Paroxysmal vomiting of great violence is the usual form they assume, and they may simulate gastric ulcer or other organic affections of the stomach. If the knee-jerks be absent and the pupils immobile to light, the diagnosis is easy, but it must be remembered that gastric crises may occur early in a case of tabes before the usual signs of disease of the cord have manifested themselves. One should inquire in such a case for a history of lightning pains, and for any trouble with the bladder. It is said also that the blood-pressure is raised during a gastric crisis, whereas it is lowered in all other cases of acute vomiting. (See also PAIN IN THE LOWER EXTREMITIES, p. 438.)

Nervous or **Hysterical Vomiting** may also simulate dyspepsia. The diagnosis here must be made largely by the method of exclusion. The patient is usually a woman, and there may be other signs of hysteria present (p. 465).

In **Chronic Intestinal Obstruction** the abdominal pains, and the vomiting which often accompany them, may be described by the patients as 'indigestion.' In such a case there will be distention of the abdomen, often with visible peristalsis, and a history of gradually increasing constipation. A tumour may be felt, or examination with bismuth and the x-rays, or with the sigmoidoscope, may clear up the case.

Cholecystitis is very apt to be diagnosed as 'indigestion.' In the case of middle-aged or elderly women, particularly, who complain of 'wind' and 'spasms,' the possibility of the presence of gall-stones should always be thought of.

Chronic Appendicitis may manifest itself chiefly by symptoms which point to the stomach rather than to the vermiform appendix as the seat of the disease. The pain in such a case may have the character of a typical 'hunger-pain,' and be relieved by alkalies. In children who are brought to one for 'indigestion,' with vague abdominal pains, the possibility of appendicitis should be specially remembered.

Angina Pectoris in one of its forms may be accompanied by much flatulence, which leads the patient to consult his doctor for 'indigestion.' The occurrence of the symptoms upon exertion, the characteristic tendency of the pain to spread into the left arm, and the frequent presence of a high blood-pressure are all of diagnostic value. *Abdominal angina*, in which the pain is seated in the large abdominal blood-vessels, may be more difficult to differentiate. Flatulence is again a pronounced feature ; but there may also be vomiting, and even hæmatemesis. Thickening of the peripheral blood-vessels is usually present ; and the therapeutic test is of help, the pain being relieved by vasodilators, and especially by diuretin.

A patient who suffers from **Migraine** may describe his case as one of 'indigestion.' The chief diagnostic point is the occurrence of severe headache with or preceding the gastric symptoms, and the marked periodicity of the attacks.

Extra-abdominal causes of Pain are often put down by patients to indigestion. Examples of these are pleurisy, spinal caries, and aneurysm.

Eructatio Nervosa, due to air-swallowing, is also usually described as indigestion. For the method of diagnosing it, see FLATULENCE (p. 240).

II. FUNCTIONAL VERSUS ORGANIC DYSPEPSIA.

Having excluded all these possible causes of error one may conclude that one has to deal with a case of either organic or functional disease in the stomach itself. If vomiting, loss of flesh, or *severe* pain be prominent symptoms, the disease is probably *organic* ; if these be absent, and the affection has persisted for some time, one has most likely to do with a *functional* disorder.

III. DIFFERENTIAL DIAGNOSIS OF ORGANIC DYSPEPSIAS.

The chief organic diseases which have to be thought of are : (1) *Cancer*, (2) *Ulcer*, (3) *Gastritis*, (4) *Obstructive dilatation*.

Cancer.—A malignant growth in the stomach may be situated either at the *cardiac orifice*, in the *body*, or at the *pylorus*. In the first of these situations it will produce difficulty in swallowing. If at the pylorus, it will result in dilatation of the stomach (see below). Growths in the body are those which are most difficult to diagnose.

(*a*). A history of 'indigestion' beginning abruptly in a patient (oftenest a man) above the age of forty, and not yielding speedily to simple treatment, is very suspicious. On the other hand, it must be remembered that in a considerable number of cases the growth starts in an old ulcer, so that a history pointing to this may also be in favour of carcinoma.

(*b*). Steady loss of weight, and the early appearance of anæmia, point to malignant growth ; but, on the other hand, the absence of these signs, and even a temporary gain in weight under treatment, by no means exclude it.

(*c*). Loss of appetite, and especially a disinclination for meat, are usually early symptoms. Nausea and vomiting supervene later but are rarely absent altogether. Pain may be present early, and is often more or less constant. (See PAIN IN THE EPIGASTRIUM, p. 436.)

(*d*). A steady diminution in the amount of hydrochloric acid in the gastric juice, with the presence of lactic acid and of Oppler-Boas bacilli in the gastric contents, is a combination pointing strongly to carcinoma.

It is therefore upon a combination of these symptoms and signs that the diagnosis must be based in the early stage when it is most important to make it. Later, a tumour may be felt below the left costal margin, or in the epigastrium; enlarged glands may appear above the left clavicle, although they are exceptional; and there may be signs of secondary growths in the liver, or at the umbilicus. When ulceration has supervened, traces of blood may be found in the gastric contents, and occult blood in the stools (p. 81).

In some cases of carcinoma of the body of the stomach, pronounced anæmia is one of the earliest and most striking symptoms. Such cases have to be diagnosed from pernicious anæmia. A blood-count will usually suffice to distinguish them, for in gastric carcinoma the red cells are rarely below 2,000,000 per cubic mm., whereas in pernicious anæmia they go much lower than that; in pernicious anæmia, also, the colour-index is about 1 or above it, in carcinoma it is less than 1. Megaloblasts are found in the film in pernicious anæmia (p. 24), but not in carcinoma. In spite of all that has been said above, the early diagnosis of carcinoma of the stomach is a matter of great difficulty; and it may be justifiable to resort to an exploratory operation in a suspicious case which does not clear up after a few weeks' treatment.

Ulcer.—The characteristic symptom of gastric ulcer is *pain* which comes on after food and is relieved by vomiting, which is usually though not invariably present. Hæmatemesis is strongly confirmatory, but is often absent. The symptoms will be found on p. 268. Duodenal ulcer is also often associated with symptoms that the patient describes vaguely as 'indigestion'; the symptoms are given on p. 271.

The chief *sign* of ulcer is a *localized* spot of tenderness on deep pressure.

Gastritis.—Chronic 'gastric catarrh' is certainly diagnosed oftener than it should be, the majority of cases so described being really examples of functional dyspepsia. The symptoms are loss of appetite, fullness and weight in the epigastrium, *depending greatly upon the kind of food taken*; pain is not a feature of gastritis; nausea is common, and vomiting may occur, but is not usually a prominent symptom. There is no characteristic physical sign, and a diagnosis cannot be made with certainty without the use of the stomach tube. This shows: (*a*) Diminished total acidity, or even complete absence of gastric juice; (*b*) Excess of mucus in the contents, or the presence of mucus on washing out the fasting stomach. Having determined the presence of gastritis, one has to settle whether it is primary or secondary. Secondary gastritis may occur: (*a*) Where there is disease of the heart, causing back-pressure; (*b*) In cirrhosis of the liver; (*c*) In chronic renal disease. If all of these can be excluded, primary gastritis may be diagnosed, and the chief causes of the latter looked for. These are: (*a*) Defective or carious teeth, and oral sepsis; (*b*) Abuse of alcohol, tea, or tobacco, or the taking of irritating articles of food.

Dilatation.—*The presence of dilatation* is determined by: (*a*) Showing that the stomach is enlarged; and (*b*) Proving the occurrence of stagnation of the contents.

(*a*). Enlargement of the stomach may be inferred when, by percussion, the greater curvature is found to reach below the level of the umbilicus, the lesser curvature being in its normal position. In order to facilitate percussion it may be necessary to inflate the stomach by making the patient swallow 90 gr. of tartaric acid followed by 120 gr. of bicarbonate of soda. Examination by the *x*-rays after a bismuth meal is of help in obscure cases (*Fig.* 144). The presence of splashing is not a certain sign of dilatation, unless it be present some hours after a meal.

(*b*). The occurrence of stagnation of contents is proved by giving the patient an evening meal, preferably containing some easily recognizable food, e.g., currants, and washing out next morning. If food residues are present in the washings, stagnation may be inferred. Examination with the *x*-rays is a still more certain method of diagnosing delay in emptying of the stomach; normally the bismuth or barium meal should have left the stomach in six, or at most eight, hours.

The cause of dilatation may be either: (*a*) Some obstruction at the pylorus; or (*b*) Primary atony of the stomach wall.

In distinguishing between these, the history may help. Thus the occurrence in the past of symptoms of ulcer points to a cicatricial stenosis of the pylorus. If visible peristaltic waves are seen, one may be sure of the existence of an obstruction. These can sometimes be elicited by massaging the stomach, or by flicking the surface of the abdomen gently with a wet towel. The presence of actual stagnation of the contents is also strongly in favour of obstruction, as this rarely, if ever, occurs in cases of atonic dilatation. Copious vomiting also points to obstruction, as it is exceptional to meet with this symptom in atony.

Assuming that obstructive dilatation has been diagnosed, one has next to determine its cause. Here one has to distinguish between *benign* and *malignant obstruction.* A history or signs and symptoms of ulcer (see above), point to the former ; the general symptoms of carcinoma, to the latter. A tumour may be felt in either case. Examination of the stomach contents also helps in the differential diagnosis. The presence of abundance of free HCl, with sarcinæ and yeasts, points to benign stenosis ; diminution or absence of HCl with the presence of lactic acid and Oppler-Boas bacilli, to malignancy.

One has further to distinguish dilatation from : (*a*) Gastroptosis ; and (*b*) Hour-glass stomach.

(*a*). In *gastroptosis*, percussion (if necessary after inflation) will show that the lesser curvature is displaced downwards, as well as the greater ; but the normal distance between the two curvatures—about four inches—is preserved. In most cases of gastroptosis the right kidney is more or less freely movable, and this affords confirmatory evidence. The x-rays may also be of help (*Fig.* 144).

(*b*). *Hour-glass stomach* may be diagnosed by the following signs :—

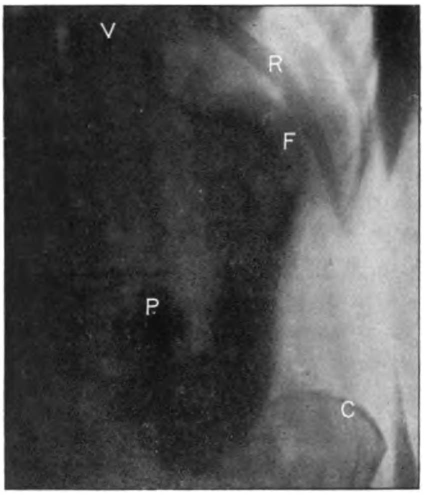


Fig. 144.—Skiagram showing the bismuth shadow of a dropped and dilated stomach, the patient being in the vertical position. C, crest of left ilium ; F, fundus of stomach ; P, pylorus ; R, twelfth left rib ; V, vertebral column. *(By Dr. A. C. Jordan.)*

(i). If the stomach be washed out with a known quantity of fluid, e.g. 30 oz., it will be found that some has been lost, e.g. 6 oz., when the return fluid is measured. Some of the fluid seems to disappear, in fact, as if it had flowed through a hole.

(ii). If the stomach is washed clean, and the tube passed a few minutes later, several ounces of fermenting liquid may be obtained, which have escaped from the pyloric pouch.

(iii). If the stomach is drained apparently dry, a splash can still be obtained over the pyloric end (' paradoxical dilatation ').

(iv). If the stomach resonance is percussed out carefully, and the viscus is then inflated with tartaric acid and soda, as described above, and then again percussed, it will be found that a great increase in resonance has occurred at the cardiac end only. If the abdomen is watched for a little, the pyloric pouch may sometimes be seen to fill gradually and become prominent. A loud gushing sound can also be distinguished on listening with the stethoscope over the site of the opening between the two pouches.

(v). x-ray examination after a bismuth meal will show the division of the stomach into two pouches (*Fig.* 128, p. 268).

IV. DIFFERENTIAL DIAGNOSIS OF FUNCTIONAL DYSPEPSIA.

Assuming that all the above forms of organic disease can be excluded, one may conclude that the case is one of functional dyspepsia.

The next task is to determine what particular variety of functional disorder one has to deal with. In attempting to do this, one is met at the outset by the difficulty of classifying functional disorders of the stomach. Three forms of classification may be adopted : (1) *Physiological,* (2) *Clinical,* (3) *Etiological.*

Physiological Classification.—In this classification, cases of functional dyspepsia are arranged according to the particular function affected, thus : —

(*a*). *Affections of secretion* :—

(i) Excess = Hypersecretion and hyperchlorhydria.
(ii) Defect = Achylia and hypochlorhydria.

(*b*). *Affections of motility* :—

(i) Excess = Pyloric spasm.
(ii) Defect = Atony, or impaired motility.

(*c*). *Affections of sensation* :—

Excess = Hyperæsthesia or gastralgia.

Any of these may be present alone, or two or more may exist in conjunction.

The diagnosis of affections of secretion and motility can be made by the aid of the stomach tube and by bismuth and the *x*-rays.

For the diagnosis of hyperæsthesia (gastralgia), see PAIN IN THE EPIGASTRIUM (p. 436).

The above is undoubtedly the most scientific method of making a differential diagnosis in cases of functional dyspepsia, but it has the inconvenience of necessitating the use of test meals.

Clinical Classification.—Clinically, cases of functional dyspepsia may be classified into certain rough groups according to their symptoms. Thus :—

(*a*). *Hypersthenic Dyspepsia.*—This is probably due to a combination of hypersecretion and hyperæsthesia. The patient is usually a young man, otherwise healthy ; and the chief symptom is pain during the late period of digestion.

(*b*). *Asthenic Dyspepsia.*—This is due to impaired motility (atony), with or without some disorder of secretion. The patient may be of either sex, and of any age, and the chief symptoms are flatulence and fullness. It is often present along with gastroptosis, especially in women ; and there may be atonic dilatation of the stomach.

(*c*). *Acid Dyspepsia.*—This is an ill-defined group in which the chief symptom is a sensation of acidity, or the presence of acid eructations. Some cases are really examples of hyperchlorhydria, with or without the presence of gastritis. In others, the cause is the production of organic acids by fermentation. Diagnosis can only be made by aid of the stomach tube.

Other clinical forms of dyspepsia are also described, e.g., 'senile' dyspepsia (essentially a hypochylia), 'gouty' dyspepsia (the same as the 'acid' form), 'flatulent' dyspepsia (usually due to defective motility), and others ; but the use of such terms is inaccurate, and should be avoided as far as possible.

Etiological Classification.—Instead of attempting to distinguish different forms of functional dyspepsia, one can regard the latter as an aggregation of symptoms of gastric disorder excited by different causes, and classify cases according to the particular exciting cause at work. This method is simple and convenient, and is also useful for purposes of treatment. Adopting it, one may say that functional dyspepsia may be induced by :—

(*a*). *Dietetic causes*, e.g., unsuitable food, hasty meals, the abuse of alcohol, tobacco, tea, etc.

(*b*). *Physical causes*, e.g., imperfect chewing, defective teeth, oral sepsis, over-fatigue, deficient exercise, etc.

(*c*). *Mental causes*, e.g., over-work, a studious life, etc.

(*d*). *Emotional causes*, e.g., shock, worry, etc.

Any of the above methods is useful, the essential point being that a classification of some sort should be adopted. Probably a combination of the first and third methods, which take into account both the particular disorder which is present and the cause which has brought it about, will lead to the best treatment.

METHODS OF EXAMINING THE STOMACH.

The following is a brief account of some special methods employed in examining the stomach, which are capable of being carried out in ordinary practice :—

1. **Determination of Size.**—This is done by light percussion, or by percussion-auscultation, with or without previous inflation. The position of the lesser and greater curvatures and of the fundus must be determined. Inflation is performed either : (*a*) Through a stomach tube connected with a Higginson's syringe ; or (*b*) By making the patient swallow 90 gr. of tartaric acid dissolved in three ounces of water, followed by 120 gr. of bicarbonate of soda.

2. **Investigation of the Contents.**—A test meal, consisting of two slices of dry toast and two cups of tea with a little milk, is given in the morning, and the tube passed an hour later. The tube should have a solid end and one bevelled lateral eye close to it. The sample drawn off should be investigated as regards :—

(*a*). *Quantity.*—A very small result containing little fluid indicates diminished secretion (achylia) ; an abundant and very liquid yield indicates diminished motility.

(*b*). *Physical Characters.*—The presence of large pieces of but slightly altered food indicates defective secretion and digestion; a large amount of liquid with a granular deposit shows hypersecretion. A very sour odour reveals the presence of organic acids. Viscidity of the contents, so that they filter slowly, is characteristic of the presence of mucus in excess.

(*c*). *Acidity.*

(i). *Test for Free HCl.*—Congo-red paper is turned blue, methyl-orange paper red, if free HCl be present. The depth of colour indicates approximately the amount of free acid.

(ii). *Total Acidity.*—Ten c.c. of the filtered contents are titrated with $\frac{N}{10}$ caustic soda solution, two or three drops of phenolphthalein solution being used as an indicator. A pink tinge appears as soon as the acidity has been neutralized. The result is expressed in terms of the amount of caustic soda solution required to neutralize 100 c.c. of the gastric contents : e.g., if 6 c.c. neutralize 10 of the contents, then the acidity is 60. The normal acidity is between 40 and 70.

(iii). *Organic Acids* need only be tested for if free HCl is absent. A sour odour of the contents indicates their presence ; acetic acid and butyric acid can be recognized by the odour of vinegar or rancidity respectively ; lactic acid by adding a few drops of the contents to some Uffelmann's reagent (equal parts of 1–20 carbolic and weak liq. ferri perchlor.) in a test tube : a bright yellow colour is produced if the acid be present.

(*d*). *Ferments.*—Rennin can be tested for by neutralizing some of the contents, and trying whether the addition of a few drops to a little milk results in coagulation when kept warm for twenty minutes.

There is no convenient test for pepsin, but its absence may usually be inferred if there is no rennin present.

(*e*). *Microscopical Characters.*—Films are made from some of the deposit, and stained with dilute gentian violet. Oppler-Boas bacilli, yeasts, and sarcinæ (*Fig.* 121, p. 241) should be looked for. The first occur specially in cases of carcinoma ; the two last in benign stenosis of the pylorus.

3. **Determination of Motility.**—Impaired motility is shown by the presence of food residue in any quantity (say about 4 ounces) six and a half hours after an ordinary dinner. In order to prove the presence of stagnation, a light meal, preferably containing some easily recognized food (e.g. currants), should be given in the evening, and the stomach washed out next morning. If food be found in the washings, stagnation exists. If there be no food, but if several ounces of greenish acid fluid are obtained, hypersecretion is present ; flakes of mucus may be found in the washings in gastritis. *Robert Hutchison.*

INEQUALITY OF THE PULSES.—(See Pulses, Unequal, p. 550.)

INEQUALITY OF THE PUPILS.—(See Pupil, Abnormalities of the, p. 551.)

INSOMNIA means inability to obtain the normal amount of sleep. It includes sleeplessness and broken or restless sleep, and admits of no closer definition because the normal amount of sleep varies widely with age, habit, and idiosyncrasy. Thus, out of the twenty-four hours, an infant at one month will sleep for twenty-one, at six months for eighteen, at twelve months for fifteen hours. A child four years old needs twelve hours' sleep, the schoolboy of twelve needs ten, the public-school man should have nine. The average hours of sleep in adult life are said to be eight for women, seven for men, but idiosyncrasy may cut down the hours necessary in certain people to no more than three or four, for long periods and without any impairment of health or the power to work. Habit may train neglected children or overworked labourers and servants to get on with short hours of rest and interrupted sleep that would speedily make an ordinary person ill. Insomnia is a symptom indicating that something is amiss, not a disease *per se*. It occurs in a great many acute and chronic disorders, but in most cases it depends on functional disturbances, faulty habits or hygiene, an ill-arranged regimen, and not upon organic disease. It is to be diagnosed whenever lack of sleep causes, or is associated with, loss of health. Most patients habitually underestimate the amount of sleep they get, without any intention to deceive ; and are apt to complain that they have been awake all night, when in point of fact they have had many hours of sleep.

The chief causes of insomnia are tabulated below in three main groups, etiologically :—

1. **Insomnia due to Faulty Habits or Hygiene,** such as—

Some sudden change in the routine of the day or evening
Exposure to undue excitement or bad atmosphere before retiring
The use of a noisy, airless, or overheated bedroom
The use of too many bedclothes, or too few
Going to bed on too full or too empty a stomach
Drinking strong tea or coffee too late in the day
The over-use of tobacco.

2. **Insomnia due to Acute Disorders,** such as—

Pain due to any cause, inflammation, injury, neuritis, etc.
The early stages of fevers
Acute insanity, meningitis, delirium tremens, acute mania, etc.
Acute nervous exhaustion.

3. **Insomnia due to Chronic Disorders,** such as—

Gastro-intestinal disorders, dyspepsia, constipation, etc.
Chronic insanity of all sorts, neurasthenia
Cerebral syphilis, intracranial tumour
Disease of the heart, valvular or myocardial
Disease of the lungs, emphysema, bronchitis, asthma, etc.
Diseases of the liver or kidneys
Arteriosclerosis and high blood-pressure, hyperpiesis
Anæmia, primary or secondary
Hysteria and malingering.

The closer investigation of the causes of insomnia may best be done by taking the age of the patient into consideration.

Sleeplessness in an Infant is most often due to indigestion, hunger, or bodily discomfort; in rare cases it is evidence of nervous instability or ear or brain disease. Enquiry into the methods and hours of feeding the infant will often show where the fault lies : the food may be improper, the hours of feeding too frequent, the practice of giving the bottle or breast whenever the infant cries may have been followed, or the habit of allowing it a dummy teat to suck at all hours. The artificially fed infant is likely to suffer from indigestion and colic, with screaming, drawing up of the legs, and rigid abdomen ; the breast-fed infant will more often fail to sleep because it is hungry. In many cases it fails to sleep because it is in discomfort from a wet napkin or bed, from having too many bed-clothes and being over-heated, or from being cold ; the bedroom may be too light or too noisy. In not a few instances it fails to sleep well for want of proper training ; especially if it finds that it will be fed or rocked in the arms or cradle as often and as long as it sees fit to cry. When the infant is six months old or more, rickets and the local irritation of teething are common additional causes. In a minority of cases the sleeplessness is due to the onset or presence of acute or chronic disease, or to the indeterminate condition described as nervousness or nervous instability, or to definite mental deficiency ; careful examination of the infant and its previous history should suffice to clear up the diagnosis in these cases. As the treatment of sleeplessness in an infant hardly ever demands the use of sedative drugs, but consists mainly in rectifying errors of diet, hygiene, or up-bringing, it is obvious that the medical man must be prepared to go deeply into these domestic—rather than medical—matters.

Sleeplessness in Children is due largely to causes similar to those described above. In a great many cases it is due to indigestion, with which may be associated flatulence, teething, and the presence of worms in the intestine ; tea-drinking is a common cause of chronic dyspepsia, nervous irritability, and disturbed sleep, in children as well as in adults. Many children sleep ill because they are put to bed within an hour or so of a late tea or early supper of too solid a character ; others, for want of fresh air in the bedroom, waking late on the following morning in a headachy and irritable condition and with little appetite for breakfast. Not a few ill-fed or anæmic school-children sleep badly during term-time because they are over-worked, or worried about their lessons or their place in the class, without being actually over-worked ; in such cases the distraction afforded by games is likely to be more successful in effecting a cure than treatment by rest. It is only in the minority of instances that the insomnia is due to disease, whether acute or chronic, such as adenoids, enlarged tonsils, or organic disease of the various viscera. A few special forms of insomnia seen in childhood call for brief mention. In *early hip disease* sleep may be disturbed by sudden starting pains ; the child goes off to sleep, only to be awakened almost at once by sudden shooting pains in the affected leg or hip. Sleep is broken by fright in *night-terrors* (see NIGHTMARES, p. 402), in which the child wakes up screaming and frightened, but conscious and able to explain, so far as excitement permits, the nature of the fright ; indiscretions in diet, or the presence of adenoids or worms, often explain the occurrence of such night-terrors. In the rarer and more serious form of night-terror, known as *pavor nocturnus*, the child awakes, screaming and frightened, but not fully conscious, and unable to recognize those around him. There is no recollection of the fright next day, and in all probability the pavor is akin to epilepsy, occurring mainly in children with a bad family history of nervous disease. It is plain from what has been said above that the diagnosis of the cause of sleeplessness in a child demands scrutiny of the daily routine, diet, and sleeping-arrangements, as well as examination of the child itself. Sedative drugs are practically never required for its treatment, except in the case of severe acute or chronic disease, and even then should be given but rarely.

Insomnia in Adults, in the majority of cases, is due to faults of *habit* or *hygiene* similar to those already mentioned in the case of children ; but it is due to organic disease of one sort or another in not a few instances, discussed later. The sleepless adult should devote thought to the economy and arrangement of his bed and bedroom, and the hours he keeps. The bed should be comfortable—whether the mattress be hard or soft is a matter of taste ; many people sleep better with a high pillow than with a low, and if a high pillow is not agreeable, the same effect can often be produced by putting blocks two or three inches high beneath the posts at the head of the bed. The bedclothes should be light rather than heavy ; they should be warm enough to prevent the occurrence of cold feet, a very common cause of sleeplessness. The bed should not be placed so that the sleeper faces the light. A supply of fresh air throughout the night is essential, and is assured if the room is heated by an open fire : stuffiness and overheating of the atmosphere seem almost inseparable from heating by stoves, hot air, hot water, or steam, and are common causes of sleeplessness. The hygiene of the bedroom having been attended to, the *habits* of the sleepless patient should be passed in review. Many well-to-do people sleep ill because they go to bed too soon after a heavy dinner ; a few because they go to bed hungry. Not a few find that they sleep badly if they take a cup of coffee after dinner, or even drink tea in the afternoon ; others sleep ill if they indulge in brain-work after dinner, or attend exciting public meetings, theatres, concerts, and so forth. It is known that bodily and mental fatigue promote sleep, and some patients with insomnia solicit sleep and aggravate their condition by pushing fatigue to the point of exhaustion, forgetting that over-fatigue often produces sleeplessness. The observance of fairly regular hours for work, food, and sleep is often neglected by busy men, and the neglect often results in disturbance of their sleep. Sudden changes in the mode or routine of daily life, or alterations in the altitude or locality inhabited, may result in acute and persistent insomnia. It is to the investigation of these and similar irregularities, trifling as many of them may appear, that one must look in diagnosing the cause of insomnia in healthy or fairly healthy patients ; its treatment will naturally turn mainly on their correction. Healthier habits of life must be advised, and the use of sedatives prohibited entirely.

In the case of adults suffering from the most various *acute disorders*, slight or severe, the occurrence of insomnia is a commonplace. It passes off with the amelioration of the disorder, and if the patient is able to give an account of himself and his symptoms, the diagnosis should not be difficult.

It is often otherwise with adults suffering from insomnia due to *chronic disease* ; the sleeplessness may be one of the earliest symptoms of illness, or the other symptoms that are present may have escaped the patient's notice. For example, persistent inability to sleep is often a prominent and early feature of *nervous* or *mental disease*—melancholia, mania, general paralysis, hypochondriasis, neurasthenia, acute nervous exhaustion, paralysis agitans, and chronic alcoholism may here be mentioned ; in old age, senile nocturnal mania may occur as a very troublesome form of insomnia. Inability to sleep may be marked in cases of cerebral tumour or cerebral syphilis. Want of sleep throws a great strain on the nervous system generally, and so is a prominent factor in the production of insanity ; the one aggravates the other, and a vicious circle is established. In *heart disease*, insomnia is frequently a distressing feature ; the patient often has to sleep propped up in bed because of breathlessness whenever the recumbent position is adopted, and when he does get off to sleep he is often awakened by cardiac palpitation or dyspnœa, within a few minutes. Restless nights are passed even while cardiac compensation is maintained ; when compensation fails the condition is much aggravated. Patients with aortic incompetence may be kept awake by the pulsating shock and noise of their own hearts. Dyspnœa is a common cause of sleeplessness in many *diseases of the lungs*. Patients with bronchitis, emphysema, spasmodic asthma, extensive pulmonary adhesions or pulmonary tuberculosis, and other kindred diseases, often pass restless nights because they are awakened by pulmonary dyspnœa soon after getting off to sleep. With these patients, as with those suffering from heart disease, the sitting or semi-recumbent position at night is often imperative, the reasons being that diaphragmatic breathing is easiest, and the amplitude of the diaphragmatic movements greatest when the patient sits, less when he lies, and least when he is in the erect position. Sleeplessness is frequent in *cirrhosis of the liver*, being accompanied by nocturnal delirium in the severer cases; it may also occur in *chronic renal disease*. It is often a persistent and distressing feature of *arteriosclerosis* and *high blood-pressure*, with hypertrophy of the heart. The mechanism whereby this sleeplessness is produced is obscure; but from the fact that any treatment that lowers the blood-pressure—massage, hot baths, high-frequency currents of electricity—cures the insomnia, it may be assumed that the high arterial pressure acts directly, preventing the establishment of the degree of cerebral anæmia that is requisite for sleep. But it must be noted that if insomnia results from the supply of too much blood to the brain, it also results from the supply of too little; hence sleeplessness occurs in *grave anæmia*, whether primary or secondary.

In conclusion, it may be noted that in *hysteria*, professions of obstinate insomnia go far beyond the observed facts; and that the *malingerer*, claiming not to have slept at all for days or weeks, may urge the sound slumber he enjoys in hospital as an argument for the prolongation of his stay. *A. J. Jex-Blake.*

INTERSCAPULAR PAIN.—(See PAIN, INTERSCAPULAR, p. 461.)

IRIDOPLEGIA.—(See PUPIL, ABNORMALITIES OF THE, p. 551.)

IRRITABILITY.—It is not very often that irritability can be regarded as a symptom of diagnostic importance. It is a relative condition, varying in its significance with the individual, and more especially with his age. Children, for instance, display irritability much more readily than adults under similar influences, owing to incomplete education of their powers of control, and a like distinction may be drawn between different persons of adult age. Varying degrees of irritability may be recognized under any condition of ill-health, and as a solitary symptom it can hardly be regarded as one of much import. An exception may perhaps be made in favour of the steadily-increasing irritability which is sometimes observed as a prodrome of *meningitis*, and which may be sufficiently remarkable to instigate a careful look-out for other early signs of that disease, such as vomiting, headache, strabismus, and head-retraction.

In adults, the personal disposition is longer established and better recognized, so that definite alterations in temperament, independent of obvious cause, and clearly not of fleeting character, must always receive attention from the medical man to whose notice they are brought. Many chronic ailments, especially those which entail mental or physical suffering, may be associated with increased irritability without exciting special remark. On the other hand there are some constitutional or metabolic disturbances which are noted for the irritability to which they may give rise. *Diabetes mellitus* and *chronic nephritis* are common examples of this kind, and the examination of the urine of patients in whom friends have observed, or who may even themselves complain of, irritability, should never be neglected. In such conditions as *jaundice*, *Graves's disease*, and *acromegaly*, other symptoms and signs are more obvious and more conclusive.

Irritability often forms part of a *neurasthenic syndrome*, but it is well to remember that the same symptom may be present in the early stages of *general paralysis of the insane*. A careful investigation of other mental changes, of the condition of the reflexes and pupils, and, if suspicion is aroused, a Wassermann test, should be carried out before coming to a definite diagnosis. It is hardly necessary to add that irritability may be associated with other depressed mental states, such as *melancholia* and *epileptic dementia*. Finally, chronic intoxications, and especially *chronic alcoholism* and *plumbism*, may be responsible for great irritability, especially in the earlier hours of the day. *E. Farquhar Buzzard.*

IRRITABILITY OF THE BLADDER.—(See MICTURITION, ABNORMALITIES OF, p. 393.)

ITCHING.—(See PRURITUS, p. 540.)

JAUNDICE is the term used to indicate the yellow or greenish coloration of the skin, conjunctiva, mucous membranes, and other tissues and fluids of the body, by bile pigment. The following are its chief signs :—

The Skin.—The colour varies from a light sulphur yellow to a deep orange, greenish, and, in some cases, dark olive tint. The greenish or dark olive shade is only found in severe cases of long standing. Intense itching is often produced, especially if the jaundice is the result of obstruction of the bile-ducts ; and this sometimes leads to vigorous scratching and the production of scratch-marks, blood-crusts, and sore places.

In certain cases, after some time, little yellowish-white or light yellowish-salmon-coloured patches of soft smooth tissue slightly raised above the surface of the surrounding skin may appear on the upper eyelids near the inner canthi. These patches may spread until the eye is surrounded entirely by this altered skin, *xanthelasma palpebrarum*. A similar condition may also occur on the palmar surface of the hands and fingers, or firm rounded nodules varying in size from ¼ in. to ½ in. in diameter, more or less raised above the level of the surrounding skin, may develop over the elbows, knees, or in other places—*xanthelasma planum* and *xanthclasma tuberosum*.

The Eyes.—The conjunctivæ are yellow. Care must be taken to distinguish deposits of subconjunctival fat from actual coloration. Occasionally, it is said, patients suffer from yellow vision (xanthopsia).

The Urine may present almost any shade, from a light saffron-yellow to yellowish-brown, medium brown, dark mahogany brown, greenish-brown, or even almost black. On looking across the upper portion of the urine in a specimen glass a distinct greenish tinge may be detected, and the froth which forms at the top on shaking possesses a distinct yellowish or greenish shade. It stains white blotting-paper and linen a bright yellow.

As a general rule, when jaundice is developing, bile pigment can be detected in the urine before the conjunctivæ become yellow, and the conjunctivæ become jaundiced before the skin. On the other hand, when jaundice is leaving a patient, the bile pigment first disappears from the urine, whilst the skin remains coloured for some time afterwards. There are certain special cases, indeed, in which the skin and conjunctivæ exhibit obvious jaundice, yet there is no bile pigment in the urine. This condition is termed acholuric jaundice (p. 332).

Other Secretions.—The sweat and milk of women who are nursing may be tinged yellow. Pleuritic, pericardial or peritoneal effusions may be similarly coloured. The tears, saliva, and gastro-intestinal secretions are not affected in this manner, nor are the meninges, brain, spinal cord, or cerebrospinal fluid.

The Fæces.—In cases of jaundice due to obstruction of the larger ducts, the fæces become greyish-white or clay-coloured from lack of stercobilin, and they may contain an excess of fat which by decomposition is likely to give the stools a very offensive smell. The bowels are usually constipated.

The Pulse.—It is frequently stated that the pulse is apt to become much slower than normal, especially in cases of catarrhal jaundice without pyrexia. It is, however, very rare to find these slow-pulse cases clinically ; more often, although physiological experiments show that the bile salts tend to slow the heart remarkably, the pulse-rate is accelerated, especially in pyrexial cases.

Bruising.—There is a marked tendency to capillary oozing and hæmorrhage in certain cases ; this is important, not only from the point of view of operations, but also because of the ready bruising of the skin which might be mistaken for evidence of violence.

Cholæmia.—In cases of severe or long-continued jaundice, cholæmic symptoms may supervene, namely, stupor, delirium, convulsions, coma, and death.

Jaundice must not be mistaken for other conditions which cause yellowness of the skin. There should be but little possibility of this, provided a careful examination is made. Slight jaundice and pernicious anæmia are perhaps the two conditions that may most readily be mistaken for each other ; in the latter, however, the conjunctivæ are generally of a pearly whiteness, however yellow the skin may be ; and if the urine should be suspiciously dark, its colour will be found to be due to urobilin, detected by its spectroscopic band between the E and F lines (*Fig.* 36, p. 80), whilst tests for bile pigments would be negative. If there is doubt as to the presence of urobilin on direct spectroscopic examination alone, further urobilin tests will be applied. One of the best is carried out as follows : A quantity of the urine, say an ounce, is poured into a suitable glass cylinder acidulated with a few drops of acetic acid ; about half an ounce of amyl alcohol is then added, and the mixture is slowly shaken to and fro in the cylinder several times, after which it is allowed to stand for some minutes whilst the amyl alcohol separates to the top ; the urobilin is now in solution in the amyl alcohol, which may be poured off into a test-tube ; on adding a few drops of a saturated solution of zinc acetate in ethyl alcohol to it, a beautiful red-green-yellow fluorescence results if urobilin is present, the appearance being similar to that of a weak solution of eosin (*Plate XXXIV*, *Fig.* 12, p. 748) ; and the absorption band of urobilin can be seen readily through it with the spectroscope. In very rare cases of pernicious anæmia there may be jaundice also. Acholuric jaundice cases are probably the most difficult to be sure of, and in some of these the diagnosis becomes a matter of opinion.

Having concluded that a patient has jaundice, the next step is to decide between the causes of the symptom. The following are the chief of them :—

CAUSES OF JAUNDICE.

I. Jaundice due to Obstruction of the larger Bile-ducts, especially of the common Bile-duct :—

A. Causes within the Duct :—

- Gall stones
- Inspissated bile
- Parasites
  - Hydatid cysts
  - Distomata
  - Ascarides

B. Causes affecting the Wall of the Duct :—

- Catarrh of the mucous membrane of the duct
- Catarrh of the mucous membrane of the duodenum involving and obstructing the ampulla of Vater
- Catarrh of the pancreas spreading to and involving the ampulla of Vater (Chronic pancreatitis)
- Carcinoma of the duct
- Cicatrization following ulceration of the duct
- Congenital obliteration of the duct

C. Causes compressing the Duct from outside or invading it from outside :—

- Peritoneal adhesions
- Enlarged portal lymphatic glands :
  - (*a*) Secondary malignant
  - (*b*) Lymphadenomatous
  - (*c*) Tuberculous
  - (*d*) Leukæmic
- Tumours of the liver
- ,, ,, pancreas
- ,, ,, duodenum
- Tumours of the stomach
- ,, ,, colon
- ,, ,, right kidney
- ,, ,, suprarenal capsule
- ,, ,, ovaries
- ,, ,, uterus
- ,, ,, omentum
- Aneurysm of the hepatic artery

II. Jaundice without Obstruction of the larger Bile-ducts :—

A. Causes associated with Disease of the Liver :—

- Carcinoma
- Cirrhosis
- Abscess
  - Single
  - Multiple or pyæmic
- Acute yellow atrophy
- Passive congestion from chronic heart failure
- Syphilis
- Active congestion

B. Jaundice in Acute Fevers and Infections, such as :—

- Typhus
- Typhoid fever
- Pyæmia
- Pneumonia
- Rheumatic fever
- Syphilis
- Weil's disease
- Yellow fever
- Relapsing fever
- Malaria
- And some other tropical fevers

C. Jaundice due to Poisons :—

- Phosphorus
- Arseniuretted hydrogen
- Antimoniuretted hydrogen
- Male fern
- Toluylenediamine
- Trinitrotoluol
- Snake poison
- Tetrachlorethane
- Dinitrobenzene
- Chloride of sulphur

D. Jaundice due to Nervous Causes :—

- Mental emotion
- Concussion

E. Jaundice due to Unclassified Causes :—

- Familial jaundice
- Epidemic infective jaundice
- Pernicious anæmia
- Splenic anæmia
- Leukæmia
- Icterus neonatorum
- Icterus gravis of children

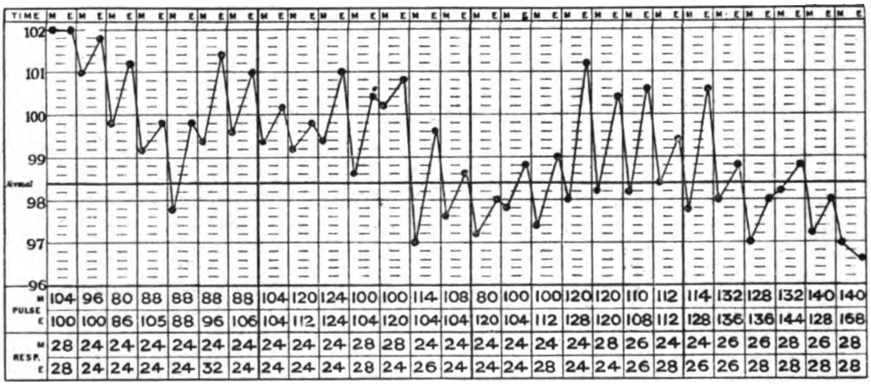
THE DIAGNOSIS.

When diagnosing the cause of jaundice in any given case, it is important to consider, not only the degree of jaundice, but also the age of the patient, the history, and the significance of any other symptoms which may be present.

Very intense jaundice and clay-coloured motions indicate some obstruction to the common bile-duct, of which the commonest causes are catarrh, gall-stones, chronic pancrea titis, or carcinoma.

Jaundice with rigors suggests : (1) Infective or suppurative cholangitis, with or without suppurating gall-bladder from gall-stones or from carcinoma ; (2) Infective or suppurative pylephlebitis, especially after appendicitis ; (3) Hepatic abscess (single or pyæmic). Almost all the different causes of jaundice may also cause pyrexia, so that without rigors the existence of pyrexia does not assist greatly in the differential diagnosis. That cirrhosis of the liver and carcinoma of the liver are both very apt to cause evening rises of temperature to as much as 100° F., 101° F., or more, is a fact that is sometimes overlooked (*Fig.* 167, p. 371, and *Fig.* 145).

The absence of pyrexia in a jaundice case will serve to exclude such conditions as abscess, pylephlebitis, cholangitis, acute specific fevers such as typhoid or Weil's disease, and epidemic infective jaundice.



PULSE	M	104	96	80	88	88	88	88	104	120	124	100	100	114	108	80	100	100	120	120	110	112	114	132	128	132	140	140
	E	100	100	86	105	88	96	106	104	112	124	104	120	104	104	120	104	112	128	120	108	112	128	136	136	144	128	168
RESP.	M	28	24	24	24	24	24	24	24	24	24	28	28	24	24	24	24	24	24	28	26	24	24	26	26	28	26	28
	E	28	24	24	24	24	32	24	24	24	24	28	24	26	24	24	24	28	24	24	26	28	26	26	28	28	28	28

Fig. 145.—Temperature chart from a fatal case of secondary carcinoma of the liver.

Jaundice with enlargement of the liver may occur in any condition of obstruction to the common bile duct, and in congestion of the liver, cirrhosis, carcinoma, syphilis, abscess, phosphorus poisoning.

Jaundice with a very greatly enlarged gall-bladder, especially persistent jaundice in a middle-aged person, suggests carcinoma of the head of the pancreas. Gall-stones seldom cause both jaundice and a large gall-bladder at the same time, perhaps because the infective process that produces the gall-stones also causes peritoneal adhesions about the gall-bladder which tie it down and prevent it from expanding.

The diagnosis is very often almost obvious. For instance, jaundice appearing in an infant two or three days after birth, and rapidly disappearing again, is almost physiological (icterus neonatorum). Transient jaundice in an otherwise healthy boy or girl will almost certainly be catarrhal. Jaundice following an acute attack of colic at once suggests a gall-stone. Recurrent attacks, extending over years, are not likely to be due to malignant disease, whereas persistent and deepening jaundice without intense pain in a person over 40 years of age, who has been wasting and has only been ill a month or two, suggests malignant disease. It often happens that the primary growth, in cases of secondary malignant jaundice, is not at once obvious, and it is important not to omit a rectal examination, lest there be a rectal carcinoma that is itself causing no symptoms.

A few words may now be said about each of the main causes of jaundice.

I. OBSTRUCTION OF THE COMMON BILE-DUCT.

A. Within the Duct.

Gall-stones may give rise to no symptoms so long as they remain in the gall-bladder. They vary in size from a grain of sand to a hen's egg, and in a few cases they may be detected by the *x*-rays (*Fig*. 146). If impacted in the cystic duct, distention of the gall-bladder may follow, but there is no jaundice. When impacted in the common duct, intense jaundice is produced, and some enlargement of the liver, but in the majority of cases no distention of the gall-bladder. Before impaction of the calculus takes place as it moves from the gall-bladder along the ducts, intense, agonizing, colicky pain is produced, which is first felt in the epigastrium and right hypochondrium. extending thence to the back of the lower part of the right chest, to the back of the right shoulder, and it may be so severe that the patient becomes collapsed. Vomiting, pyrexia, and rigors are other symptoms which are frequently associated with these attacks of pain. The latter lasts a varying time according to what happens to the calculus. If expelled into the duodenum the severe pain ceases, and the gall-stone may be found in the fæces. If found, it should be examined carefully, for if its surface is faceted it indicates that other gall-stones are present, and points to the likelihood of further attacks of colic and jaundice occurring.

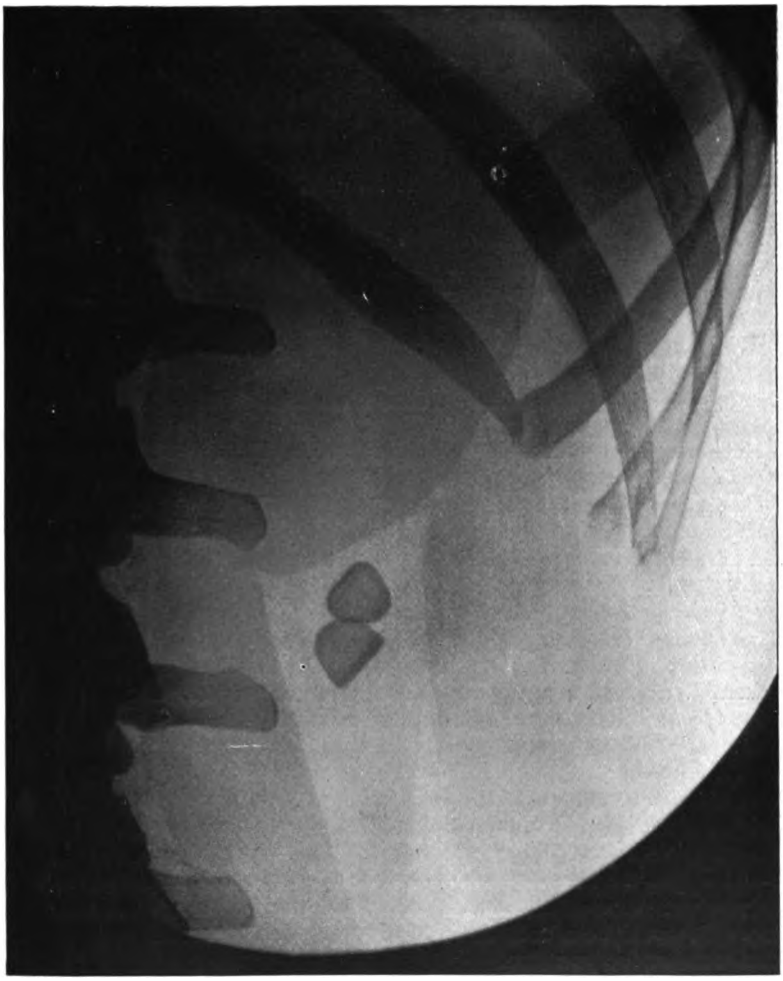


Fig. 146.—Skiagram of gall-stones: the two stones show below the outline of the kidney, and each has a distinct edge. (*Skiagram by Dr. C. Thurstan Holland, from The Medical Annual*, 1914.)

Jaundice dependent on the passage of gall-stones usually comes on about twelve hours after the commencement of the attack of colic, and persists for a varying period according to the length of time the calculus remains in the duct. Occasionally jaundice occurs without any previous colic. Recurring attacks of jaundice in a middle-aged woman, with or without attacks of colic, are almost pathognomonic of gall-stones; only one other disease produces precisely similar symptoms, namely, chronic pancreatitis. In the latter, however, the jaundice is apt to persist longer, and it may never go completely away, lessening between the attacks, to deepen again with each recurrence of the acute pancreatic pain. It is often very difficult without laparotomy to distinguish gall-stones from chronic pancreatitis. The absence of gall-stones in the fæces, and the presence of a positive pancreatic reaction in the urine (see CAMMIDGE'S PANCREATIC REACTION, p. 100), would point to chronic pancreatitis rather than to gall-stones; the former is distinguished from neoplasm by the greater amount of pain it causes and the longer the case lasts.

There are several methods of detecting pancreatic disease, most of which depend upon the non-entry of the pancreatic juice into the intestine. The percentage of fat in the stools when ordinary quantities are given by the mouth is very much greater when the pancreatic juice fails than when the bile alone fails, so that extremely fatty iridescent stools favour a diagnosis of pancreatitis or pancreatic neoplasm. The same indication is afforded when the fæces contain a large number of undigested muscle fibres; also when keratin-coated capsules are passed undigested, or when such capsules containing methylene blue are given without the urine subsequently turning blue. Other tests concern the tryptic activity of the motions, and so forth, and their value is still sub judice.

If a stone remains impacted in the common duct, the jaundice is intense; but if it soon passes into the duodenum, the jaundice is slighter and transient.

Inspissated Bile is always mentioned as a cause of jaundice, but there are no distinguishing signs of this condition, and it would require considerable boldness on the part of the physician to make this the sole diagnosis. Thickening of the bile may occur in acute fevers, poisonings, and so forth, and this is possibly the cause of the jaundice in many of the cases where there is no obstruction to the large bile-ducts; but a diagnosis of 'inspissated bile' by itself would clearly be incomplete and inadequate.

Parasites.—A *hydatid cyst* of the liver may happen to be in such a position as to stenose the common bile-duct, or it might open into the gall-bladder, cystic, hepatic, or common bile-ducts. It is, however, an exceptionally rare cause of jaundice, and it could seldom be diagnosed unless by laparotomy.

Distoma hepaticum.—The normal habitation of this parasite is the bile-ducts of the sheep; it is sometimes found occupying a similar position in man, though in England this is of extreme rarity. The chief symptoms are jaundice, ascites, enlargement of the liver, vomiting, pyrexia, diarrhœa, and pain in the right hypochondrium. If circumstances should suggest this infection, the vomit and the stools should be examined for flukes, and the stools for ova, which are large, brown, and operculated, measuring 0·13 by ·08 mm.

Ascaris lumbricoides (round worm).—This parasite inhabits the upper part of the small intestine and measures from 15 to 45 cm. in length. It seems to have a special tendency to force itself into small orifices, and it has been recorded as becoming impacted in the common bile-duct, with jaundice as the result. The worms themselves seldom produce symptoms, and unless they are actually found in the duct they could never be diagnosed with certainty as the cause of jaundice. Even if the worms or their ova were found in the patient's fæces, it would be a bold thing to diagnose that an ascaris impacted in the bile-duct was the cause of the jaundice.

B. Causes affecting the Wall of the Duct.

Catarrh of the Mucous Membrane of the Bile-duct (catarrhal jaundice).—This is a common cause of jaundice—in young people the most common of all. It is due to the obstruction caused by the swelling of the mucous membrane, and it is almost impossible to distinguish clinically between cases in which the catarrh is confined to the bile-ducts, and those in which it began in the duodenum and thence extended to the biliary papilla. It is usually preceded by gastro-intestinal disturbances, especially epigastric discomfort and dyspepsia. The jaundice develops almost suddenly in many cases, and it may become intense, the stools being clay-coloured and the urine dark with bile pigment. There may be a slight rise of temperature at first, the pulse may be less accelerated than would be proportionate to the temperature, and in quite rare cases it is absolutely slowed down to 40, or even 30; the liver and spleen may be enlarged slightly, the tongue furred and the breath foul; loss of appetite, nausea, constipation, a feeling of weight and discomfort in the right hypochondriac region may also be prominent symptoms. In mild cases the jaundice is slight and disappears at the end of one, two, or three weeks; sometimes it lasts as long as eight weeks, or even more. In considering the diagnosis, it should be remembered that jaundice in a child or a young adult is most likely to be due to catarrh. The slightness of the pain helps to exclude gall-stones and chronic pancreatitis, and malignant disease is rendered improbable if the jaundice presently clears up, and if the patient does not emaciate. There is a growing belief that so-called catarrhal jaundice is really a specific infectious fever allied to the exanthemata, and many instances of its affecting several members of a family or school or village within a short period of one another are on record.

Catarrh of the Pancreatic Ducts, extending to the ampulla of Vater and so to the bile-ducts, has already been discussed under *chronic pancreatitis* above. It differs from catarrh starting in the duodenum or in the bile-duct by being associated with periodic attacks of colicky epigastric pain resembling gall-stone colic.

Cicatrization following Ulceration of the Duct.—Simple fibrous stricture of the bile-ducts is a possible but rare result of ulceration due to gall-stones. If the cystic duct is thus stenosed, distention of the gall-bladder without jaundice follows; if the hepatic duct, jaundice and enlargement of the liver without distention of the gall-bladder; and if the common duct, intense jaundice, enlargement of the liver, and possibly, but not necessarily, distention of the gall-bladder. It is practically impossible to diagnose between this condition and impacted gall-stones during life, except by laparotomy.

Congenital Obliteration of the Bile-ducts.—Jaundice in infants is almost always transient, icterus neonatorum developing about the third day and passing off in a week or less. If an infant should remain persistently jaundiced, a grave condition is almost certainly present, though only a post-mortem examination, as a rule, can decide whether it is due to congenital syphilis with or without cirrhosis and pervious ducts, to congenital obliteration of the bile-ducts, or to 'icterus gravis,' the last term being used when the child dies and no obvious cause for the jaundice can be found post mortem. As regards congenital obliteration of the bile-ducts, boys are affected more frequently than girls. Jaundice may be present at birth or appear on the second day, or even as late as the fourteenth day. At first it is slight, but soon becomes intense. Constipation, pale motions, bile in the urine, and spontaneous hæmorrhages—especially from the umbilicus—are the most prominent symptoms. Death may take place in two or three weeks when hæmorrhage occurs, but if there is no bleeding life may be prolonged for six or seven months. Increasing jaundice, colourless motions, bile-stained urine, and spontaneous hæmorrhages would point to some condition more serious than icterus neonatorum.

C. Causes Compressing the Duct from Outside or Invading it from Outside.

When *compression* of the common bile-duct is spoken of, the term *invasion* of it would often be more correct, especially when the so-called compression is due to secondary deposits of malignant disease in the lymphatic glands in the portal fissure. In almost all cases of the kind jaundice is persistent, and it is often progressive, although there may be slight variations in its depth.

Enlarged Glands in the Portal Fissure. a. SECONDARY MALIGNANT GLANDS.—The lymphatic glands in the portal fissure are very liable to become enlarged from deposits of secondary growth in cases of abdominal malignant disease. Jaundice with or without ascites is a prominent indication of such a condition, and when both jaundice and ascites are present in a case of malignant disease of the stomach or intestine, whether the liver is enlarged or not, it is probable that there are enlarged malignant glands in the portal fissure. The difficulty of diagnosis arises in cases in which no primary growth can be found. In a fair number of these it is either in the rectum, colon, or pancreas.

b. LYMPHADENOMATOUS GLANDS.—The portal glands occasionally become enlarged in cases of lymphadenoma (Hodgkin's disease), or lymphosarcoma, with a similar result. The presence of enlarged superficial lymph glands and enlargement of the spleen and liver, together with a simple anæmia without leucocytosis, would suggest this diagnosis. In most cases of lymphadenoma in which jaundice occurs it is a late symptom, arising long after the correct diagnosis has already been made.

c. TUBERCULOUS GLANDS.—Although the glands in the portal fissure frequently become caseous in cases of tuberculous peritonitis, they rarely compress the bile-duct and cause jaundice.

d. LYMPHATIC LEUKÆMIC GLANDS.—The visceral glands may become enormously enlarged in some cases of lymphatic leukæmia, and in rare instances those in the portal fissure have led to jaundice. The diagnosis is easy, even if the spleen and superficial lymphatic glands are not enlarged, for a blood-count would show that the total number of leucocytes per c.mm. of blood was raised to anything between 50,000 and 2,000,000, whilst the differential leucocyte count would show a great preponderance of lymphocytes.

Tumours of the Liver.—Any disease which causes a local enlargement of the liver, e.g., carcinoma, sarcoma, abscess, gumma, or hydatid, in the immediate neighbourhood of the portal fissure, may compress the common bile-duct and lead to jaundice. On account of the close relationship between the bile-duct and the portal vein, ascites is equally liable to be produced. The association, therefore, of jaundice and ascites with a local enlargement of the liver would point to this last-mentioned condition being the cause of the two former. In many such cases, however, the jaundice is really due to deposits in the portal lymphatic glands ; for if the latter escape there may be numbers of malignant deposits in the liver without any jaundice at all.

Tumours of the Pancreas—A tumour of the head of the pancreas generally causes jaundice by invading the orifice of the common bile-duct. In some cases, situated far back in the abdomen, a mass can be felt which, on account of its close proximity to the aorta, may present distinct transmitted pulsation. It may prove difficult, without artificially inflating the stomach, to distinguish it from a tumour of the latter or of the liver. A pancreatic tumour is situated behind the stomach, and does not, as a rule, move on respiration, though if attached to the portal fissure it moves with the liver. Glycosuria and fatty stools would be strong evidence in favour of a pancreatic tumour, even if no tumour were palpable. The tests mentioned on page 101 could be employed here too. The gall-bladder is apt to become greatly distended ; indeed, persistent and increasing jaundice and decided enlargement of the gall-bladder in a person of the cancer age are probably the most characteristic symptoms of carcinoma of the head of the pancreas.

Tumours of the Duodenum.—Primary carcinoma of the duodenum is very rare, but when it does occur it usually arises in the immediate neighbourhood of the biliary papilla, and by obstructing the common bile-duct causes persistent jaundice, with progressive emaciation.

Tumours of the Stomach.—A carcinomatous tumour of the pyloric end of the stomach may become adherent to the portal fissure and cause jaundice by compressing the common bile-duct. If, however, the existence of a gastric carcinoma were known in a patient who developed jaundice, the chances would be strongly in favour of the latter being due to obstruction, not by the primary growth, but by secondary deposits in the portal lymph-glands. It should also be borne in mind, however, that even when carcinoma exists, a microbial catarrh of the duodenum may cause transient non-malignant jaundice.

Tumours of the Colon.—Carcinoma of the hepatic flexure or transverse colon may become adherent to the liver and cause jaundice by compressing the common bile-duct. It may be difficult to distinguish such a tumour from a local enlargement of the liver ; but constipation, vomiting, tympanitic distention of the intestine, and the passage of blood per rectum would point to a growth in the colon. In most of such cases, however, the obstruction to the bile-ducts is not by the primary growth, but by secondary deposits in the portal glands. The importance of rectal examination has already been insisted on, whilst much help in diagnosis may also be afforded by serial *x*-ray examination of the alimentary canal after a bismuth or barium meal.

Tumours of the Right Kidney.—Large tumours of the right kidney, especially malignant growths, may compress the bile-duct and cause jaundice. If the tumour becomes adherent to the liver it is difficult to distinguish it from an enlargement of that organ, as the liver and the enlarged kidney would move together during respiration. If the abdomen is palpated bimanually, however, the loin may be felt to be filled out behind ; and, in front, the edge of the liver may be distinguished lying over the front of the tumour, and it may be possible to detect a vertical band of colonic resonance over the otherwise dull mass. Hæmaturia, albuminuria, or pyuria would be additional evidence of renal disease.

Transitory attacks of slight jaundice are not uncommon in association with movable kidney. This is possibly due to compression of the common bile-duct by the kidney, but it may also result from the associated enteroptosis causing a drag on the duodenum, and a kinking of the common bile-duct. The diagnosis of movable kidney is not difficult, the position and the mobility of the tumour, and the curious sickening sensation experienced by the patient when it is compressed, being sufficiently characteristic.

Tumours of the Right Suprarenal Capsule.—Malignant growth of the right suprarenal capsule is very rare, but it may give rise to an enormous tumour which is difficult to distinguish from a renal, or even in some cases a hepatic, enlargement. Malignant disease of one capsule causes no symptoms of Addison's disease if the other remains healthy. In children, hypernephroma may be suggested by the premature development of pubic hair (see p. 408).

Ovarian Tumours.—A large ovarian cyst may extend upwards to the portal fissure, compress the common bile-duct, and cause jaundice, but such a complication is rare ; indeed, when jaundice is associated with ovarian tumour the suspicion will naturally be that the latter is malignant and that there are secondary deposits in the glands in the portal fissure obstructing the large bile-ducts. Ascites is very apt to be present at the same time, so that unless the existence of an ovarian tumour is already known, or unless its existence can be determined by abdominal, vaginal, or rectal examination, there may be much difficulty in determining the precise cause of the jaundice, though if cirrhosis of the liver can be excluded, some form of malignant disease will probably be suspected.

Tumours of the Uterus.—A large tumour of the uterus may cause jaundice in a similar manner to an ovarian tumour, but even more rarely.

Tumours of the Omentum.—A large omental tumour may compress the bile-duct and thus cause jaundice, but it is an exceedingly rare result of such a condition. Whether malignant or tuberculous, it usually lies across the upper part of the abdomen, is superficial, and moves slightly with respiration. If it has become adherent to the liver it may be difficult to distinguish it from a local enlargement of the latter. In any case, the jaundice will probably be diagnosed as due to deposits—tuberculous or malignant—in the portal glands, rather than to the omental mass itself.

Aneurysm of the Hepatic Artery, Cœliac Axis, or Abdominal Aorta.—An aneurysm of the hepatic artery is decidedly rare, but it is by no means unheard of in cases of fungating endocarditis with embolism. Jaundice is intense, on account of the close proximity of the hepatic artery to the common bile-duct. A correct diagnosis would be almost impossible during life, especially in view of the fact that jaundice may occur in fungating endocarditis cases simply from the inspissation of the bile that results from the toxæmia and fever.

Aneurysm of the cœliac axis or upper part of the abdominal aorta is also a very rare cause of jaundice. An abdominal tumour with marked expansile pulsation, a systolic bruit, and abdominal pain, are the most important diagnostic signs, especially if they occur in a person who is known to have had syphilis.

II. JAUNDICE WITHOUT OBSTRUCTION OF THE LARGER BILE-DUCTS.

A. Causes associated with Disease of the Liver.

Carcinoma of the Liver.—Jaundice occurs in more than 50 per cent of the cases of malignant disease of the liver, whether secondary or primary ; it is seldom, however, that the masses in the liver itself cause the jaundice, but rather the associated deposits in the portal glands. A liver may contain hundreds of nodules of new growth without there being either jaundice or ascites if the portal glands escape. Jaundice brought about in this manner is permanent, and when the common duct is involved is intense. The skin, which at first is a deep orange, becomes greenish, and finally the dark olive-green tint which is almost pathognomonic of jaundice due to malignant disease. Increasing jaundice in a patient over 40 years of age, who has been ill less than six months, who has progressively wasted and become weaker, and whose liver is enormously enlarged, hard, and nodular, points without much doubt to malignant disease, though careful search may be required before the primary source is found. The nodules may even be felt to be umbilicated. Primary carcinoma of the liver should not be diagnosed until a very careful physical examination has failed to furnish evidence of the primary growth in some other organ.

Cirrhosis.—In many cases of cirrhosis of the liver the late or multilobular stage of the disease may be reached without there having been any jaundice at all. If it occurs late in the disease, when ascites is already present, the jaundice is usually slight. Ascites is the most constant and characteristic feature at this late stage of cirrhosis, but when slight jaundice and ascites are associated in a patient who gives a definite history of alcoholism, and also has symptoms and shows signs of this condition (p. 726), and has a hard liver with a well-defined and beaded edge, the diagnosis of cirrhosis of the liver is not difficult. Sometimes, however, jaundice is a marked feature of the case at an early stage, when the organ is still large and the fibrosis unilobular, and at this time ascites is conspicuous by its absence. In most of these cases there is an evening rise of temperature to about 100° F. (*Fig.* 167, p. 371). The liver is considerably enlarged, its surface is smooth, firm perhaps, and tender, and its edge is even and well defined, reaching to the level of the umbilicus or even below it. The jaundice may pass off, and the patient survive many years before the multilobular ascitic stage of his malady is reached; on the other hand, if the jaundice persists and deepens, the prognosis is grave; cholæmia sets in; drowsiness and muttering delirium passing on to coma and death.

There is a peculiar form of cirrhosis of the liver (Hanot's) which affects several members of the same family, and whose first symptom in each patient is jaundice. The disease appears not to be caused by alcohol, syphilis, or malaria. It is possible for the patient to live many years with more or less jaundice all the time. The icteric tinge of the skin is often unaccompanied by bile pigment in the urine in these chronic cases—a variety of acholuric jaundice. The liver is enlarged and hard, and the spleen is also moderately increased in size. The diagnosis is arrived at by enquiring into the family history.

There is another malady, known as *familial acholuric jaundice* (*Plate XVIII*), which simulates Hanot's cirrhosis very closely during life, but is found at operation or post mortem to present no hepatic cirrhosis. Several members of the same family are affected, the spleen is enlarged considerably, the symptoms develop either soon after birth or during the first ten years of life, progress slowly, with periods of remission, and during exacerbations there is considerable chlorotic anæmia, associated with fragility of the red corpuscles as tested with varying strengths of salt solution, and a consequent tendency to hæmorrhages of all kinds—hæmatemesis, hæmoptysis, epistaxis and purpura in particular. The pathology of this condition is still obscure; some of the cases give a positive Wassermann test and appear to be due to congenital syphilis; these are differentiated by some observers from what they term true familial acholuric jaundice, in which the Wassermann test is negative. Excision of the spleen has cured not a few of these cases.

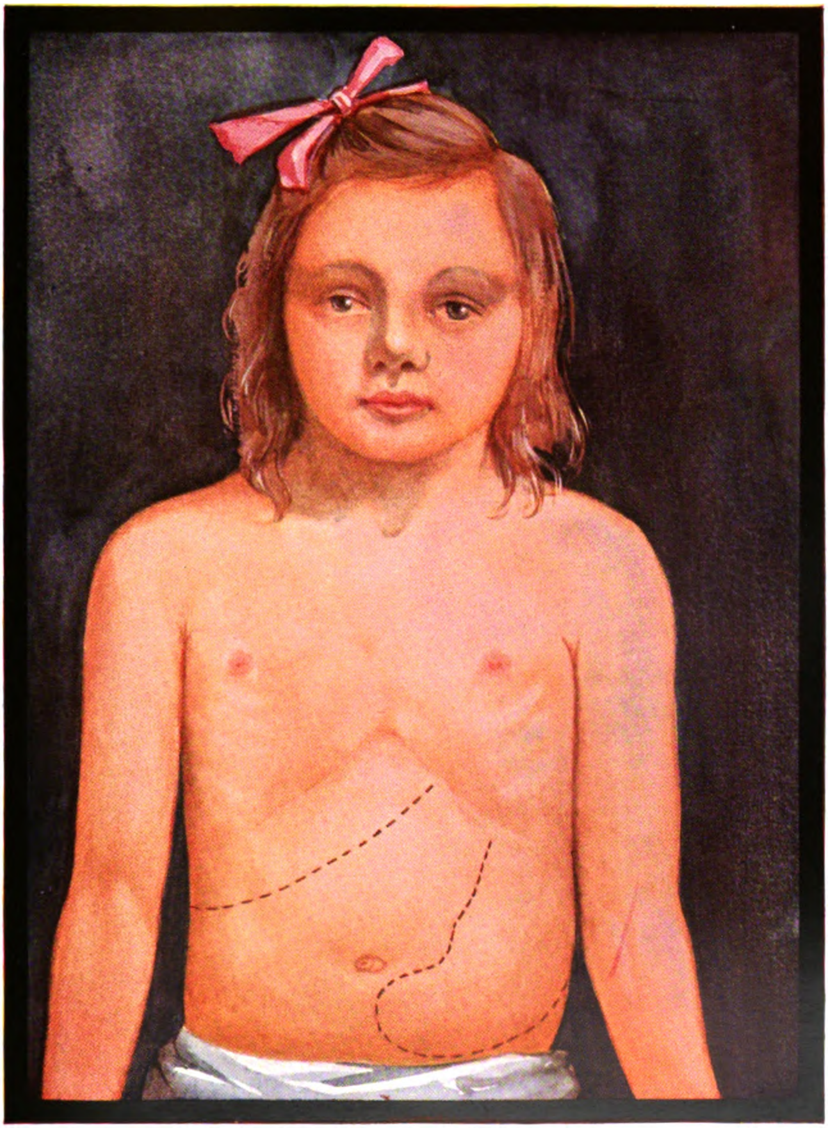
There is yet another particular variety of cirrhosis of the liver which occurs in children and young people, and is characterized by enormous enlargement of the spleen, slight enlargement of the liver, anæmia without leucocytosis, hæmatemesis, clubbing of the fingers, jaundice, and stunted growth. It differs from Hanot's cirrhosis in that the liver is smaller and the spleen larger, and from the latter feature of the case it is termed *splenomegalic cirrhosis*.

Single or *Tropical Abscess.*—In cases of single or tropical abscess of the liver intense jaundice is rare, and it is only likely to occur when the abscess bulges in the region of the portal fissure. The general appearance of a patient who is suffering from hepatic abscess may, however, be mistaken for jaundice, because the complexion is sallow, and the conjunctivæ may even have a slightly icteroid tinge. The urine, however, seldom contains bile pigment. The disease mostly affects people who have resided in the tropics, particularly those who have had dysentery. The diagnosis is discussed on p. 369). Should the abscess open into the lung, the dull reddish pus expectorated would point to its origin in the liver even though no *Amœbæ dysenteriæ* be found in the pus.

Multiple Abscesses in the Liver might theoretically arise by infection through any one of four different channels, namely, the portal vein, the bile-ducts, the hepatic artery, and the lymphatics. In practice only the first two are important, giving rise to *suppurative pylephlebitis* on the one hand, and *suppurative cholangitis* on the other. There are really no sharp lines of demarcation between non-suppurative and suppurative inflammations of these channels; there are all intermediate stages between simple catarrh of the ducts and acute suppurative cholangitis; and there are similar degrees of inflammation in the case of the portal venules. Jaundice is almost constantly a symptom of cholangitis, and the

PLATE XVIII

FAMILIAL ACHOLURIC JAUNDICE



A case of familial acholuric jaundice with enlarged liver and spleen. The jaundice fluctuated in intensity, but had been persistent since infancy. Note that the general nutrition of the patient is good. There was no obvious bile in the urine

diagnosis is arrived at when a cause for cholangitis exists, such as gall-stones, carcinoma of the gall-bladder, empyema of the gall-bladder after typhoid fever, and when the patient's liver enlarges and becomes tender, especially if rigors also occur from time to time. Suppurative pylephlebitis is diagnosed less easily, and indeed it is often overlooked as a cause for an obscure febrile illness accompanied by rigors. About half the patients who have it develop jaundice, and one very important point is that, in over half the cases, the cause of the infection of the portal vein is a recent mild attack of appendicitis. If, therefore, a patient who has recently had pains or discomfort in the right iliac fossa presently begins to do badly, developing pyrexia and rigors without apparent cause, and if that patient in the course of a week or so develops a tinge of jaundice and a slightly enlarged liver, the grave diagnosis of infective pylephlebitis should suggest itself.

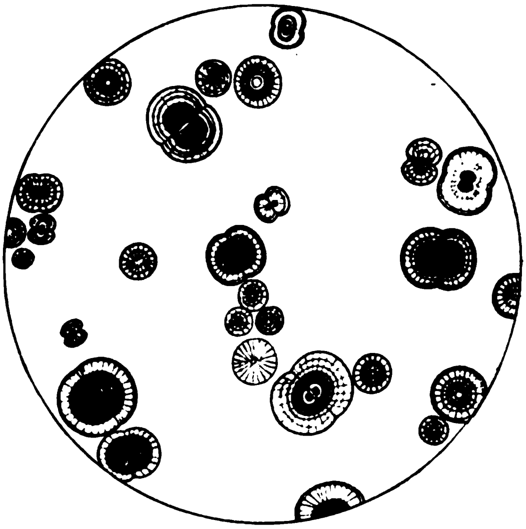


Fig. 147.—Leucin crystals, as seen under the ⅙-in. objective.

In *Acute Yellow Atrophy of the Liver* jaundice is one of the earliest symptoms. In the early stages bile pigment may be found in the urine, but towards the end, when the skin becomes green, Gmelin's reaction cannot be obtained, or only a trace of pigment can be detected (acholuric jaundice).

The disease is rare. It affects females under 30 years of age more frequently than males, and in a good many cases has been preceded by fright, or severe mental emotion, or childbirth. It usually commences in the same manner as an attack of catarrhal jaundice, with nausea, vomiting, loss of appetite, constipation, and pain in the right hypochondrium. At the end of two or three weeks a sudden change occurs, which commences with severe vomiting, headache, restlessness, followed by delirium, convulsions, and coma. The temperature rises to 101° F. or 102° F., and the pulse becomes rapid. The tongue is dry and brown. There is a tendency to hæmorrhage from various parts, e.g., epistaxis, hæmatemesis, melæna, and metrorrhagia. The most important diagnostic signs are the remarkable diminution of urea and uric acid, and the presence of leucin (*Fig.* 147) and tyrosin (*Fig.* 148) in the urine; also the rapid diminution in the extent of the hepatic dullness which takes place after the development of the above-mentioned nervous symptoms. The duration of the malady, in the majority of cases, is under fourteen days from the time of the sudden change in the type of the jaundice.

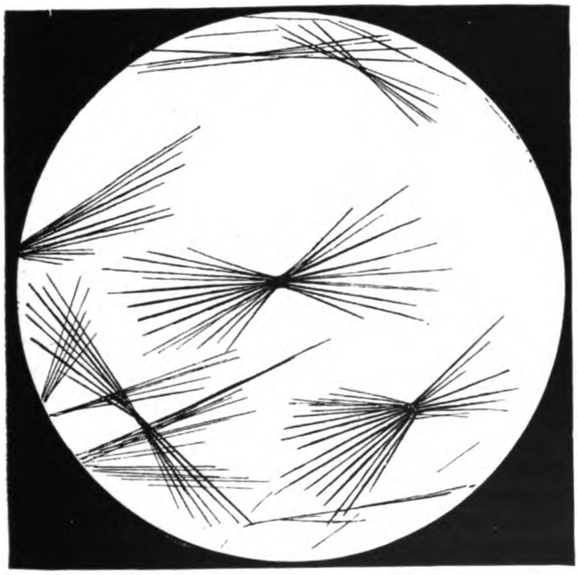


Fig. 148.—Tyrosin crystals, as seen under the high power of the microscope; sheaves of colourless needles, coarser and shorter than those of phenyl-glucosazone (*Fig.* 127, p. 262), which are yellow.

The striking resemblance between this disease and that producible by poisons suggests that in it there is toxæmic catarrh of the bile-ducts—that is, a catarrh produced by the excretion through the bile of injurious products which cause extensive degenerative changes in the liver cells. A condition which resembles acute yellow atrophy very closely in its clinical features has affected not a few persons engaged in the varnishing of the wings of aëroplanes, or occupied upon the premises in which this work is being carried on. The varnish used is a very special one, composed of acetate of cellulose dissolved in a mixture of spirit, acetone, benzol, and tetrachlorethane. It is the vapour of the latter which is the cause of the toxic symptoms ; in milder cases recovery occurs when the patient is removed from the works on account of continued ill-health with more or less severe gastro-intestinal symptoms—especially flatulent dyspepsia, vomiting, epigastric pains and loss of appetite ; those who remain exposed to the vapour for weeks or months develop jaundice in addition—at first exactly like a simple catarrhal jaundice, but soon passing on to a serious and generally fatal stage precisely similar to that of acute yellow atrophy of the liver, though generally without leucin and tyrosin in the urine. A somewhat similar condition has resulted from the effects of *dinitrobenzene*, used in the manufacture of high explosives such as roburite and bellite ; from *trinitrotoluol*, and from the use of *chloride of sulphur* by rubber workers. The diagnosis is suggested by the circumstances of the occupation. Post mortem the liver in these cases is shrunken and discoloured, just as it is in acute yellow atrophy.

Passive Congestion (nutmeg liver).—Jaundice occurs in severe cases of passive congestion, especially as the result of long-standing mitral stenosis, or of fibrosis of the lung with ultimate failure of the right side of the heart. It is usually but an icteric tinge, but when severe its association with cyanosis gives a curious dusky-green tint to the skin, especially that of the face. Œdema of the legs and ascites are also present as a rule. The liver is considerably enlarged, its edge is sharp and well defined, its surface smooth, firm, tender, and possibly pulsating. Jaundice from this cause should not be difficult to diagnose. If in a chronic heart case there are both pyrexia and jaundice, fungating endocarditis is probable.

Syphilis.—Congenital syphilis may cause jaundice in infants or young children as the result of intralobular fibrosis, but it is possible for the latter to be extensive without there being any jaundice, and even where the latter is present it is usually slight. If associated with uniform enlargement of the liver, wasting, and other signs of congenital syphilis, the diagnosis is not difficult.

In an adult it is possible for gummata to cause jaundice by compressing the ducts, but this is distinctly rare. The local enlargement of the liver and pyrexia may lead to a diagnosis of abscess or of secondary carcinoma. A careful examination must be made for signs of syphilis ; in some cases it is not until antisyphilitic remedies have been administered and the effect watched that a correct diagnosis can be made. If there are any active lesions of the skin or mucous membranes, it may be possible to detect the *Spirochæta pallida* microscopically (see *Plate XXVIII*, *Fig. J*, p. 614) ; or the patient's serum may be examined in the laboratory for Wassermann's reaction.

Probably the commonest period at which syphilis may be directly responsible for jaundice is the secondary stage, when it is apt to cause catarrh of many different glandular ducts, including those of the liver. The symptoms will be very like those of simple catarrhal jaundice, together with the roseola, the sore throat, the pyrexia, the albuminuria, and other signs of secondary syphilis.

Active Congestion.—Active congestion of the liver is a diagnosis that some observers would not hold with, whilst others are convinced that it is not an uncommon result of many of the acute fevers, such as malaria, and that it may arise from insufficient exercise associated with alcoholism and over-eating, particularly in Europeans who live in the tropics. The liver is slightly enlarged and tender. The chief symptoms are slight jaundice, pain, and a feeling of fullness, weight, and oppression in the right hypochondrium, which sensations are much increased by pressure ; also pain in the right shoulder, a bitter taste in the mouth, nausea, sickness, a furred tongue with indented edges, constipation, and scanty high-coloured urine. There may be a temperature of 102° F., and then care must be taken to distinguish it from hepatic abscess. An absence of leucocytosis would be in favour of congestion and against suppuration. If due to malaria, an examination of stained blood-films should demonstrate the presence of malaria parasites. It is clearly impossible to distinguish clinically between active congestion of the liver and catarrh of the bile-ducts.

B. Jaundice in Acute Fevers.

Malaria.—Slight jaundice may occur in long-continued tertian and æstivo-autumnal infections, and on account of the associated irregular pyrexia it may lead to a mistaken diagnosis of hepatic abscess. Microscopic examination of stained blood-films, and the discovery of the characteristic parasites in the red blood-corpuscles (*Plate XXVIII.* p. 614), are the most conclusive evidence of malaria. Jaundice may also occur as a result of malarial cirrhosis. It should be borne in mind that the parasites disappear rapidly from the blood in cases in which quinine has been administered recently. It is important, therefore, to examine blood-films before quinine is given, and if possible at the very start of an ague fit, at which time they are at their most typical stage of development. If quinine has been given already, however, there will still be presumptive evidence of malaria if there is no leucocytosis, and if the differential leucocyte count shows a decided increase in the proportion of large hyaline lymphocytes—up to 15 per cent or more.

Typhus Fever.—Jaundice may occur occasionally in this disease, which, fortunately, is now extremely rare in Great Britain, but it may reappear in any country during times of famine or distress ; or during war, as in Servia and other regions during the European War. It is a disease of poverty, famine, dirt, and squalor, and is spread by lice. The onset is more sudden, and the prostration occurs earlier and is more marked, than in typhoid fever, There is often a slight leucocytosis. The rash appears from the third to the fifth day, and consists of a dusky red mottling—the mulberry rash—rose-coloured papules which appear on the abdomen and chest, together with a certain number of petechiæ—the latter not being found in typhoid cases. The fever tends to terminate by crisis rather than by lysis. Widal's reaction is negative.

Typhoid Fever.—Jaundice is rare in this disease ; it occurred in only three out of Osler's series of 829 cases. It is due to an inflammation of the bile passages by typhoid bacilli. The gall-bladder may become enlarged and tender, and give rise to a typical palpable tumour, though this may also occur in typhoid fever without any jaundice at all. It arises occasionally as a complication in the course of the disease, or it may be a sequela, or again it may be an early and prominent symptom for which the patient seeks advice. Cases have been recorded of primary typhoid infection of the gall-bladder and bile-ducts without any accompanying ulceration of the intestine. The low pulse ratio when compared with the temperature, e.g., a pulse of 90 with a temperature of 104° F., the presence of typical rose-red spots on the abdomen, enlargement of the spleen, leucopenia, and a positive Widal's reaction, are the most important signs which would point to a diagnosis of typhoid fever.

Pyæmia and *Septicæmia.*—Jaundice is frequently a late symptom of pyæmia, and may or may not be associated with the presence of multiple abscesses in the liver. It is more likely to occur in cases of portal than arterial pyæmia. Rigors, high irregular temperature, rapid pulse, profuse sweating, rapid emaciation, and progressive loss of strength, are symptoms which, if developing after parturition, wounds, or operations, would point without much doubt to a diagnosis of pyæmia. In some instances of acute septicæmia due to streptococci, staphylococci, and perhaps other micro-organisms, there has been intense jaundice of the skin and conjunctivæ of a peculiar mustard-yellow tint, without the urine giving a positive Gmelin's test. The urine may or may not be discoloured—in some instances it looks merely concentrated, in others it looks almost like porter, and yet it gives no play of colour to the nitric acid test ; the cause of this would seem to be the oxidation of the pigment before it reaches the urine. This occurrence of acholuric jaundice in septic cases is very remarkable, and it may be a confusing factor in the case.

Pneumonia.—Jaundice occurs occasionally as a complication of pneumonia. It varies very much in its frequency in different epidemics of the disease. It is noticed soon after the initial rigor, but is rarely intense. It is probably due to engorgement of the liver and catarrh of the bile-ducts. Its more frequent association with right basal pneumonia is suggestive. The sudden onset with a rigor, the high temperature, the rapid respiration-rate, which is above the ordinary temperature and respiration ratio, and the comparatively slow pulse (e.g., T. 104° F., R. 40, P. 100), the characteristic tenacious, russet-brown sputum, the short catchy cough, the pain in the side, the pleuritic rub, and the signs of consolidation of the lung, the hot dry skin, the deficiency of chlorides in the urine, and the occurrence of herpes facialis, are the accompanying indications which in the majority of cases would point to a diagnosis of pneumonia.

Infectious or *Epidemic Jaundice* (*Weil's Disease*) is characterized by a sudden onset with pyrexia, severe pain in the back and limbs, headache, and giddiness, followed in a day or two by jaundice, enlargement of the liver and spleen, and nephritis. The jaundice becomes intense within twenty-four hours, the temperature rises to 103° F. to 104° F., and the pulse becomes rapid. Nephritis, next to jaundice, is one of the most constant features of this disease. Males between 15 and 30 are most affected, and it is connected with insanitary surroundings. Butchers appear to be particularly susceptible. It is practically unknown in England.

Yellow Fever in some respects resembles acute yellow atrophy of the liver, but the liver does not atrophy, neither does the spleen enlarge, and crystals of leucin and tyrosin are not found in the urine. It is essentially a tropical or sub-tropical disease, prevalent in the West Indies and Central and South America. The incubation period is from three to four days, and the onset sudden, with rigors, headache, pain in the back and limbs, and constipation. Jaundice is an early symptom, and one of the most characteristic, but it varies in intensity, being much more severe in fatal than in mild cases. The temperature rises to 102° F. or 103° F.; the pulse is rapid at first, but may fall as the temperature rises, and this is regarded as a very typical sign of the disease. Albuminuria, black vomit, hæmorrhage from the gums and beneath the skin, are other important symptoms. A sporadic case occurring in this country would probably be looked upon as acute yellow atrophy of the liver unless a definite history of exposure to infection was obtainable. It may be difficult to distinguish it from dengue (p. 466) and pernicious malaria. From the latter it can be diagnosed if crescents are discovered in the blood (*Plate XXVIII, Fig. E.* p. 614).

Relapsing Fever.—Jaundice is a common symptom of this contagious fever, which is prevalent in India, and is liable to arise in other countries in times of famine. It is spread by bed-bugs. Considerable enlargement of the liver and spleen, and a good deal of abdominal pain and tenderness, are present in most cases; also epistaxis and hæmatemesis. The most characteristic feature of the disease is the temperature, which rises abruptly to 104° or 105°, and even to 108° F., remains high for five or six days, and then suddenly falls to normal when, after an interval of about a week, it again rises and remains high for three or four days (*Fig.* 5, p. 27). During the periods of pyrexia the *Spirochæta obermeieri* (*Plate XXVIII, Fig. I,* p. 614) may be found on examining blood-films prepared and stained in the same manner as for the detection of malaria parasites. The blood examination serves to distinguish it from malaria.

C. Jaundice due to Poisons.

Phosphorus.—Jaundice, though by no means constant, is one of the most characteristic symptoms of phosphorus poisoning. It is slight at first, appearing on the second or third day in severe cases, but in mild ones not until the end of the first week, or even later. This form of poisoning is rare in this country since the stringent law regulating the manufacture of matches from the non-poisonous form of the drug has been in force. In the cases which do occur, the phosphorus has been taken in the form of match-heads or rat paste, with suicidal intent. At first the signs are those of acute irritant poisoning, coming on soon after the phosphorus has been swallowed, viz.: nausea, vomiting, severe burning pains in the epigastrium, collapse, extreme thirst, rapid feeble pulse, rapid respiration, and tenderness in the epigastrium and right hypochondriac regions. In many cases that receive treatment early these acute irritant symptoms subside in a day or two, and recovery results. If they do not thus subside, however, after from two to five days the symptoms change, the vomit becomes black or brownish from the presence of blood, jaundice appears and rapidly deepens, the liver enlarges, and headache, drowsiness, delirium, convulsions, and coma supervene, followed shortly by death. If hepatic enlargement cannot be ascertained it may be difficult to distinguish phosphorus poisoning from acute yellow atrophy of the liver. Hæmorrhages, although common, are not as frequent as in acute yellow atrophy. The urine is concentrated and strongly acid; the total nitrogen is first reduced, as in cases of starvation, to about one-fourth the usual, and then, in spite of the fact that the patient can retain no food, it rises to the usual amount. Urea forms the greater part of the total nitrogen, but towards the end the total amount of ammonia is increased. Leucin and tyrosin are not usually found, and the chlorides are diminished. The condition of the urine, therefore, forms a contrast to the changes which are found in cases of acute yellow atrophy. The chief indications of the disease that are found post mortem are jaundice, multiple punctiform hæmorrhages, fatty degeneration of the liver, kidneys and heart, and enlargement of the spleen.

Arseniuretted Hydrogen causes jaundice in a similar manner to toluylenediamine, through blood destruction and extreme concentration of the bile; the increase of bile pigment may be to three and a half times more than its normal amount. The bile acids are diminished.

Toluylenediamine has been used for experimental purposes, and its action has helped to prove that so-called hæmatogenous jaundice is really due to obstruction of the smaller ducts through increased viscidity of the bile. When injected into dogs it soon produces intense jaundice; it causes destruction of blood, and the hæmoglobin thus liberated increases the viscidity of the bile, so that temporary obstruction of the smaller ducts, followed by jaundice, results.

Tetrachlorethane, *Dinitrobenzene*, and *Chloride of Sulphur* are discussed above.

Snake Poison.—Jaundice is a common result of snake-bite, and is produced in a similar manner to the last three forms described, viz.: as a result of concentration and increased viscidity of the bile leading to obstruction of the smaller bile-ducts. The diagnosis depends upon the history. The symptoms vary with the kind of snake that has bitten the patient. In England the only poisonous variety is the adder or viper, whose bite is followed almost at once by a burning local pain, quickly succeeded by acute ascending œdema and darkening discoloration of the limb. The patient is nauseated and presently vomits, he turns giddy and faint, has to lie down, and in from one to three hours is completely prostrated, often comatose, and covered with clammy perspiration. The temperature falls below normal, the pulse may be almost imperceptible, and death may result at this stage. More often the severe constitutional symptoms pass off gradually, improvement beginning within twenty-four hours; but the swelling and discoloration of the bitten limb remain extreme, and there may be generalized œdema all over the body. Suppuration or even gangrene is common, and recovery is apt to be very slow, even when appropriate surgical measures are employed at once. It is during the period between the passing off of the initial coma and the beginning of convalescence that jaundice is prone to develop.

D. Jaundice due to Nervous Causes.

Icterus Nervosa—Mental Emotion.—Cases are on record of jaundice following almost immediately after some violent mental emotion, but they are of extreme rarity. The jaundice has been explained as being the result of a sudden spasm of the bile-ducts. In another class of these cases icterus does not appear until twelve or fourteen hours after, and it is then probably due to catarrh of the bile-ducts, associated with gastric and duodenal catarrh, for it is well enough known that severe mental emotion, grief, or anxiety may give rise to acute dyspepsia. Jaundice may occur similarly after concussion of the brain. *Herbert French.*

JAW, SWELLING OF THE.—(See SWELLING OF THE JAW, p. 683.)

JERK, ACHILLES.—(See ANKLE-CLONUS, p. 39.)

JERK, KNEE.—(See KNEE-JERK, ABNORMALITIES OF THE, p. 357.)

JOINTS, AFFECTIONS OF THE.—It will be well to place these in two groups (1) *Acute*; (2) *Chronic.*

1. Acute Joint Affections.—*Arthritis due to Rheumatic Fever* is the most frequent of these. The patient has often had the disease before, or other members of the family may have had it; there may be manifestations of past rheumatic affection of other parts of the body; thus the presence of organic mitral disease is of great help in the diagnosis of a doubtful case. A history of past chorea, several attacks of tonsillitis, pericarditis, or rheumatic erythema or nodules will help. The distinguishing features of the arthritis are that it is acute, and affects first and chiefly the larger joints, although in a very severe case even the joints of the hand and fingers may be implicated; it does not occur in all the affected joints simultaneously, but appears in one, a few hours after in another, and so on. As the arthritis often lasts a few days in any one joint, in some it may have passed away while others are being affected. The pain is very severe and is greatly increased by any jar of the bed; it is more fleeting than the arthritis, but like it, flits from joint to joint, hardly ever returning to the same joint in the same attack. The swelling of the joint is usually only slight or moderate; it is due to synovial effusion, never suppurates, generally subsides in a few days, and usually in at most a fortnight the joint returns completely to its normal condition. Permanent distortion or stiffness of the joints after rheumatic fever occurs, but it is highly exceptional. Often there is a faint red blush over the joint when first affected.

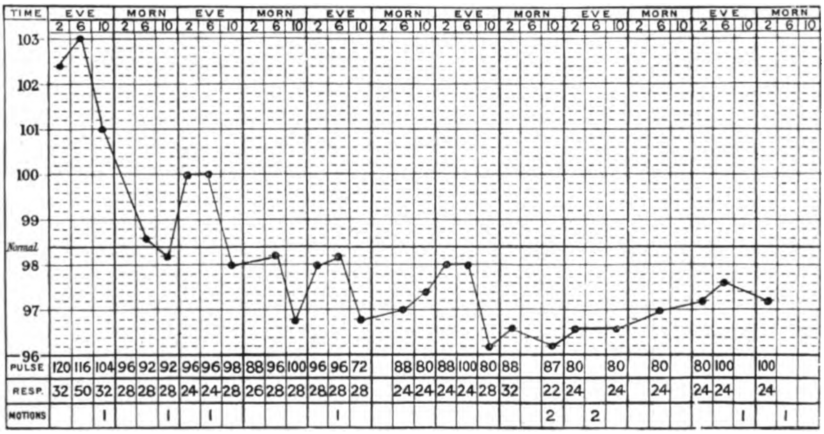


Fig. 149.—Temperature chart in a case of acute rheumatism, showing rapid fall of temperature by use of salicylates.

The most important diagnostic characteristics of this arthritis are: (1) The fact that it flits from joint to joint. Never diagnose rheumatic fever so long as only one joint is affected. I have known failure to remember this lead to a diagnosis of rheumatic fever in traumatic arthritis, tuberculous arthritis, arthritis due to acute necrosis of a bone near a joint, and acute suppurative arthritis; in each of the last two, the mistake has cost the patient his life. (2) The arthritis in turn quickly leaves joints. Failure to remember this has often caused septic, gonococcal and various forms of infective arthritis to be called rheumatic arthritis, a mistake which has also cost lives. The drenching, sour-smelling sweats, and the relief of the pain by salicylates, are very characteristic of rheumatic fever, but septicæmia also causes sweating. The sweating of rheumatic fever is particularly liable to be accompanied by minute glassy vesicles—sudamina. Finally, the arthritis of rheumatic fever being transient, is not accompanied by much arthritic muscular atrophy. Rheumatic nodules are rare, but when present are almost diagnostic; they are most often seen in young boys affected with rheumatic fever and heart disease; but very rarely they are met with in osteo-arthritis, and once I have seen them with gonorrhœal arthritis.

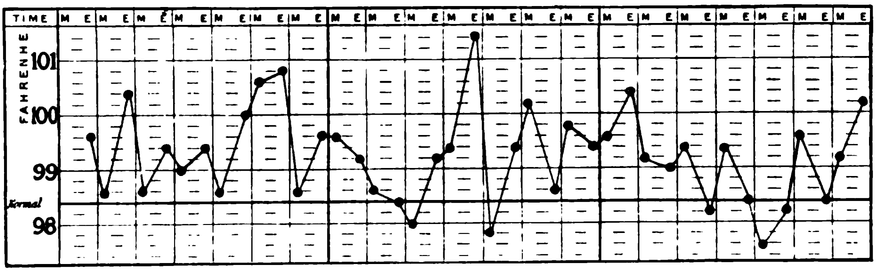


Fig. 150.—Temperature chart in a case of gonococcal arthritis of ordinary severity, to show the absence of effect of salicylates upon the pyrexia. The case was mistaken at first for acute rheumatic fever.

(3) The joint pains and the pyrexia are generally (*Fig.* 149), though not absolutely invariably, relieved by salicylates within forty-eight hours, whereas gonococcal arthritis (*Fig.* 150), acute rheumatoid arthritis (*Fig.* 152, p. 341), gout (*Fig.* 151), and other acute joint affections, are not quickly influenced by salicylates in the same way.

Septic Arthritis is constantly being thought to be rheumatic fever: a bad mistake, especially for the patient. In septic arthritis, it is true, several joints may be affected; but it may be one only, which it never is in rheumatic fever; further, in septic arthritis the trouble does not clear up in one joint and then pass to another; a joint once affected remains affected till the source of infection is removed; the soft tissues around are thickened and brawny, quite unlike rheumatic fever, and if the colour is altered—which is not often the case—it is dusky, and not the bright red of rheumatic fever. Suppuration often occurs: in rheumatic fever, never. Whether or not suppuration takes place, the joint often becomes fixed, which is excessively rare in rheumatic fever. Then, if proper search is made, the source of infection can usually be found; common places that are overlooked are the sockets of the teeth, and the vagina and uterus, but the source may be anywhere, e.g., septic arthritis may follow dilated bronchial tubes, cystitis, prostatic abscess, a boil on the skin, otorrhœa, inflammation of the nasal cavities, and perhaps ulceration of the intestine. Sometimes the most careful seeking fails to find the source, but the search must not be given up readily. The irregular temperature, usually hectic, the leucocytosis, sweats, and other signs of septicæmia are often a help.

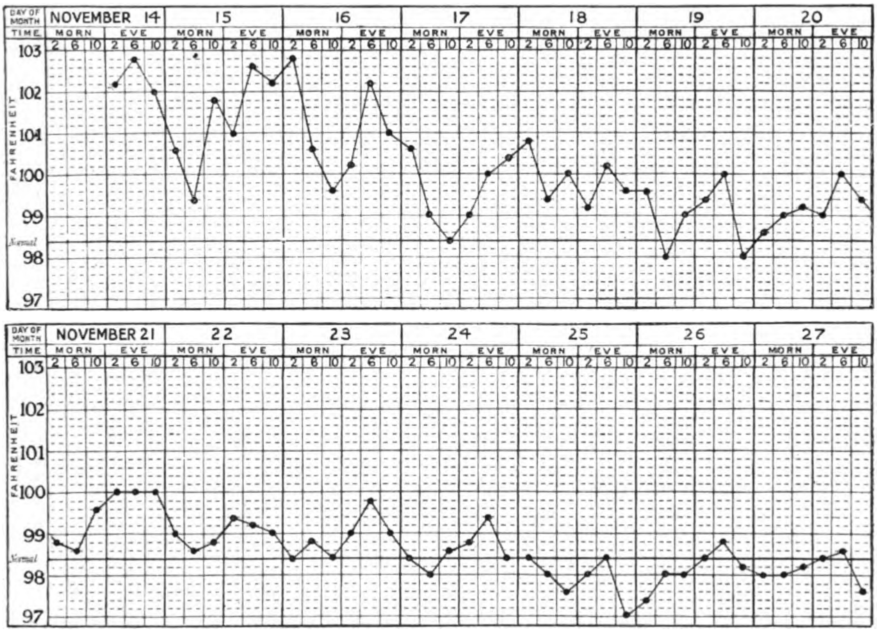


Fig. 151.—Four-hourly temperature chart in a case of acute gout. Woman, age 46, had had several previous attacks of typical gout; recovered completely for the time being. Salicylates had no effect upon the pyrexia.

Pneumococcal arthritis is rare in adults, and nearly always exists as a complication of acute pneumonia. It may, however, be found without evidence of pneumococcal disease in any other part of the body. Pneumococcal otitis media should not be forgotten as its possible source. Generally only one joint is affected, usually the knee, less often some other large joint, such as the shoulder or elbow. Often there is a history of recent injury to the part. The patient suddenly feels a pain in the joint; within a few hours of this the temperature is raised; the joint swells rapidly, is very painful and exquisitely tender; yellowish-green pus quickly forms. The diagnosis is obviously easy if the patient has pneumonia, but may be difficult if he has not; it is important to come to a diagnosis early, for it is a serious disease, and if allowed to go far without incision and drainage the patient may succumb to a general septicæmia. Pneumococcal arthritis is the commonest form of infective arthritis in children under five years old. As in adults, it is confined generally to one large joint. The swelling may be very great, and extend to the soft tissues beyond the joint. The pain is less than in adults, and redness is not common. Its possible presence must be remembered, for as in adults, so in children, it is necessary to drain the joint early. The child has a raised temperature, and looks ill.

Typhoid Arthritis.—There are two varieties, both very rare: (1) That which precedes the typhoid fever; this is a multiple arthritis, not of severe degree, which subsides just before definite symptoms of typhoid show themselves. It is impossible to diagnose it until the appearance of the typhoid fever. (2) This occurs during the typhoid fever; one or many joints may be affected; the arthritis is of varying severity; it may subside completely, or require incision and drainage. In a few cases even when there has been no arthritis during or before the attack of typhoid fever some chronic arthritis may appear later; most often the joints and ligaments of the spine are affected, and during the convalescence from his fever the patient complains of much pain and stiffness of his back; he is then said to have a *typhoid spine*. In a similar way the hip may become stiff, and very rarely there is chronic osteitis of the head and neck of the femur.

Scarlatinal arthritis affects many joints, is not severe, soon subsides, and is easily diagnosed when there has been recent scarlet fever. It is commonly known as scarlatinal rheumatism, a bad name which quite gratuitously assumes a connection between this arthritis and rheumatic fever, for the existence of which there is no evidence.

Arthritis occurs commonly in association with *meningococcic meningitis* and *Malta fever*, less commonly with *dysentery*, rarely in association with *influenza*, *glanders*, *small-pox*, *measles*, and *diphtheria*. In all these cases the presence of the principal disease determines the diagnosis.

Gonorrhœal Arthritis is often called gonorrhœal rheumatism, but this phrase should be discarded, for there is no association between gonorrhœa and rheumatic fever. Gonorrhœal arthritis is frequently overlooked. I have repeatedly demonstrated its presence when the family physician has believed its existence impossible. It is particularly likely to be missed in women. I have met with it in married women of fifty; it is probable in these cases that they are infected by their husbands. It may follow gonorrhœal ophthalmia and even ophthalmia neonatorum. The diagnosis may be very easy, as when a patient is seized with an acute arthritis, either of a single joint or of several joints, while he or she is suffering from gonorrhœa. If it is possible to withdraw a little fluid from the cavity of the swollen joint, the discovery of the gonococcus makes the diagnosis certain, but this is usually quite unnecessary, and unless done very carefully may, by introducing micro-organisms from without, greatly increase the damage to the joint. Often a urethral discharge may be found, though sometimes in long-standing cases of gleet it is very slight; if the gonococcus cannot be found in the discharge, it may be detected in a swab taken from the posterior urethra or vagina. If in women it is thought undesirable to excite suspicion by taking a vaginal swab, the nature of a doubtful arthritis may be determined by the wide variations of the opsonic index to the gonococcus.

It is difficult from the clinical character of gonorrhœal arthritis to tell it certainly from other forms of arthritis. Mistakes happen least often to those who constantly think of the possibility. It is of varying degrees of acuteness; in the chronic cases of gleet the corresponding arthritis is chronic, but in the acute cases of gonorrhœa it may be so acute that I have more than once known the disease called rheumatic fever. Gonorrhœal arthritis may be limited to one joint, and then most often to a large one, especially the knee; but it may be multiple, and very many joints, even those of the wrists, hands, and fingers, may be implicated; there is often much swelling of the soft tissues around, and this is more responsible for the swelling than is the effusion in the joint. Gonorrhœal arthritis is usually very painful. The sheaths of tendons are often inflamed and tender, and so are some fasciæ, especially the plantar fascia. The patient often complains of pain at the back of the sole of the foot, and in a chronic case he has flat-foot. There is no variety of arthritis in which muscular atrophy is more striking. I have known a severe case of gonorrhœal arthritis of the hand called progressive muscular atrophy. When gonorrhœal arthritis is chronic throughout the whole of its course, and is limited to one joint, the cause of the trouble is often erroneously set down to tubercle. Suppuration is very rare. Some cases are extremely chronic, and may lead to fibrous ankylosis with deformities, but with our modern means of diagnosis and treatment this has become exceptional. Salicylates have no decided effect either upon the joint pains or upon the co-existent pyrexia (*Fig.* 150).

All the acute affections hitherto mentioned, except rheumatic fever, are often included under the phrase '*infective arthritis*,' because they are known to be due to infection by a micro-organism ; but this is a loose term that ought only to be used in a general sense, for a diagnosis of the precise cause of the infection is nearly always possible if care be taken. Sometimes in an infective arthritis there is more than one micro-organism at work to cause it ; thus, in the late stages of gleet various micro-organisms flourish in the diseased urethra, and the arthritis may be due to a mixed infection in which the gonococcus is not the preponderating micro-organism ; under such conditions pus may form in the joints. Indeed, I have known a mixed infection of gonococci and streptococci from the genitals of a woman cause in her a very severe acute arthritis with a temperature of 105° F. In exhausting diseases, e.g., typhoid fever, the patient may suffer from a secondary streptococcal infection which may cause arthritis.

Acute Secondary Arthritis.—By this is meant arthritis due to spread of disease from the bone in the neighbourhood of the joint. It is limited to one joint ; the most acute and dangerous form is that which follows acute osteomyelitis. More than once I have known this called rheumatic fever, because the onset has been sudden and the temperature raised. It is a most unfortunate mistake, for patients suffering from this form of arthritis are liable to die quickly from general septicæmia, to prevent which the joint ought to be opened and drained at once. The mistake may usually be avoided by remembering that rheumatic fever does not affect one joint only. The more difficult cases are those in which more than one joint is acutely diseased, as a result of disease of the ends of the bones. To avoid this mistake, disease of the bones themselves must be carefully sought. Fortunately for diagnosis, this disease of several joints is most frequent in infants, and in them severe arthritis due to rheumatic fever is unknown. It is called *acute arthritis of infants.* Disease of joints may be secondary to an abscess in the bone or to tuberculous disease of the bone. These varieties are diagnosed by discovering the underlying disease of the bones ; *x*-rays are often of much use.

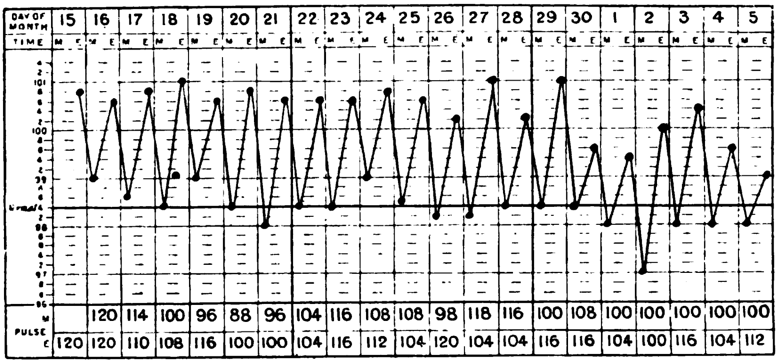


Fig. 152.—Chart showing the pyrexia and rapid pulse of rheumatoid arthritis ; the temperature was uninfluenced by salicylates.

Rheumatoid Arthritis.—The first attack is often acute : therefore the disease is best considered here. The name is bad and has led to confusion ; it owes its origin to the fact that the disease in some respects resembles rheumatic fever ; hence the name implies an arthritis resembling that of rheumatic fever. It would probably lead to worse confusion to invent a new name, so the present had better stand until the micro-organism causing the disease has been discovered. The first attack is ushered in with fever ; the temperature is rarely higher than 100° or 101° F. in the evening and 99° or 100° F. in the morning. This fever lasts from three to six weeks, slowly subsiding towards the end (*Fig.* 152). The

pulse is generally rapid, out of proportion to the fever, the hands and feet sweat profusely, patches of freckle-like pigment are prone to appear on the body. Most of the patients are young women. The pyrexia is less severe and longer lasting than that of rheumatic fever, the pulse is, considering the temperature, faster, the sweating is almost confined to

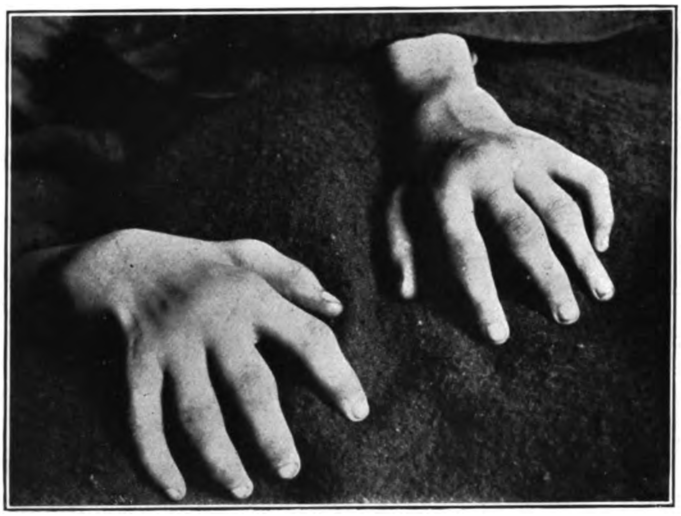


Fig. 153.—Acute rheumatoid arthritis: showing the spindle-shaped swelling of the joints between the first and second phalanges, and the swelling in connection with the wrist and metacarpo-phalangeal joints.

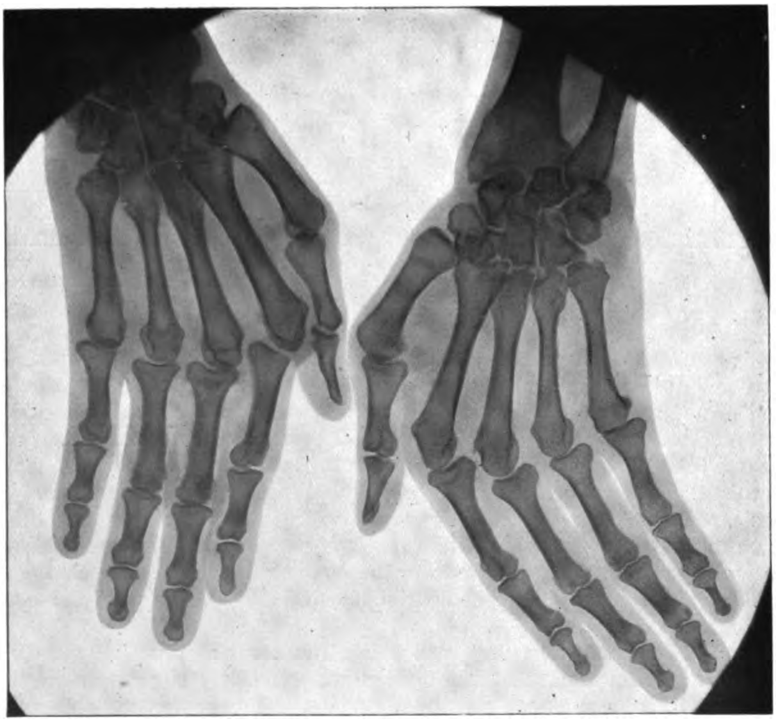


Fig. 151.—Severe rheumatoid arthritis; skiagram of the hands, showing ulnar deflection but no bony intra-articular changes.

hands and feet, pigment is frequent. Nor are the differences with regard to the arthritis less striking, for in rheumatoid arthritis the characteristic joints to be affected are those between the first and second phalanges, and as it is an affection of the synovial membranes, and also considerably of the soft tissues around the joints, quite early in the disease we get a spindle-shaped swelling of these joints (*Fig.* 153); but soon many other joints are affected, and before long almost every joint in the body is implicated, so that we have the simultaneous affection of a great many joints. The temporo-maxillary joint is often involved; so is the spine. It will be noticed that in every respect the arthritis is clinically different from that of rheumatic fever. The joints never suppurate, but the epitrochlear gland may be found enlarged. Slowly the attack subsides; as it does so, passive movements and massage should be undertaken, for if not, the thickening of the tissues around the joints leads to their fixation. That this may be prevented is shown by the fact that the jaw rarely becomes fixed, presumably because of its frequent movement. There is never any endocarditis. The arthritic muscular atrophy is often as extreme as in any variety of arthritis. In a few months a second attack comes on, but both the general symptoms and the arthritis are less severe than in the first; then a few months later another, less severe than the second, and so on, until after four, five, or six attacks the disease wears itself out. In the later stages, if the joints have been allowed to become stiff, the disease is often confused with osteo-arthritis; but rheumatoid arthritis occurs in younger subjects, and there are no bony outgrowths (*Fig.* 154), except in a few cases in which chronically thickened fringes of synovial membrane have, by friction during movements of the joints, worn away a little patch of cartilage, exposed and irritated the bone, and led to a slight outgrowth. In such a case the erosion of the cartilage may lead to bony grating, but in even a very chronic and extreme case of rheumatoid arthritis, in which the joints have not been treated, grating and bony outgrowths are quite inconsiderable, and are not a leading feature as in osteo-arthritis. Heberden's nodes (*Fig.* 155) are not seen in rheumatoid arthritis. The spindle-shaped swelling of the phalangeal joints of this disease is not seen in osteo-arthritis. The joints principally affected are different in the two diseases, as will be seen on reference to osteo-arthritis, and the history is entirely different. Formerly some importance was attached to the transparency of the bones to the *x*-rays in the neighbourhood of the affected joints which may be seen in rheumatoid arthritis (*Fig.* 156), but this is now known to be visible in other forms of arthritis. The *x*-rays are, however, of use as showing the bony outgrowths of osteo-arthritis. In chronic cases of both rheumatoid arthritis and osteo-arthritis ulnar deflections may be seen (*Fig.* 154).

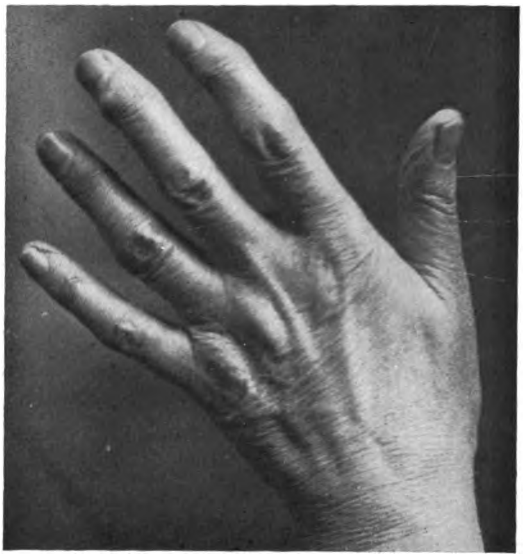


Fig. 155.—Heberden's nodosities situated on the terminal phalanges of the index and middle fingers. (From *Arthritis Deformans*, by Llewellyn Jones Llewellyn.)

Henoch's Purpura.—This disease is confined to children between infancy and fifteen years old, and early in its course pain and slight swelling of some of the large joints, with a little elevation of temperature, are often present. As in children the pyrexia and arthritis of rheumatic fever are inconspicuous, mistakes have occurred between it and Henoch's purpura, but the pain in the latter is trivial. The attacks of abdominal pain, with perhaps vomiting and diarrhœa, are characteristic, and so is the purpura (*Fig.* 157), together, in many cases, with bleeding from some internal organ—hæmaturia, hæmatemesis, or melæna. The purpura should not give rise to any difficulty; rheumatic purpura is unknown under the age of fifteen.

Gout.—This is often said to be present when it is not. The most certain points in the diagnosis of gout are, first, the detection of urate of sodium, usually as white hard masses in connection with a joint (*Fig.* 158), in a bursa, or as a deposit in the cartilage of the edge of the ear ; here it is frequently not easy to be sure if a white nodule is urate of soda or a projection of cartilage with the skin stretched tightly over it. If it is possible to remove a minute fragment with a needle, crystals of urate of sodium may be seen under the microscope. Secondly, there may be a history of repeated characteristic attacks. The gouty arthritis that we see now-a-days is generally strongly inherited, but not often by women, and therefore the family history is of importance ; it rarely shows itself before the age of twenty, though I have seen it in a boy fifteen years old. Most of the sufferers from gout now alive get their attacks quite independently of any errors in diet ; many of them are most abstemious. The diagnosis is not difficult when the patient has one or more attacks of arthritis in the characteristic joint—that of the ball of the great toe, more often the right than the left ; the attack usually begins at night with excruciating pain, which subsides towards the early morning ; the patient, exhausted with pain, drops asleep, to wake later and find his joint swollen and tense. There is some fever (*Fig.* 151, p. 339). Probably during the day his toe does not cause pain unless he walks on it ; but he has another attack the next night, not so severe as that on the first, and on each successive night the attacks are less. He may have another bout at any period of his life, and he may have many bouts, and other joints may become affected subsequently. The real difficulty

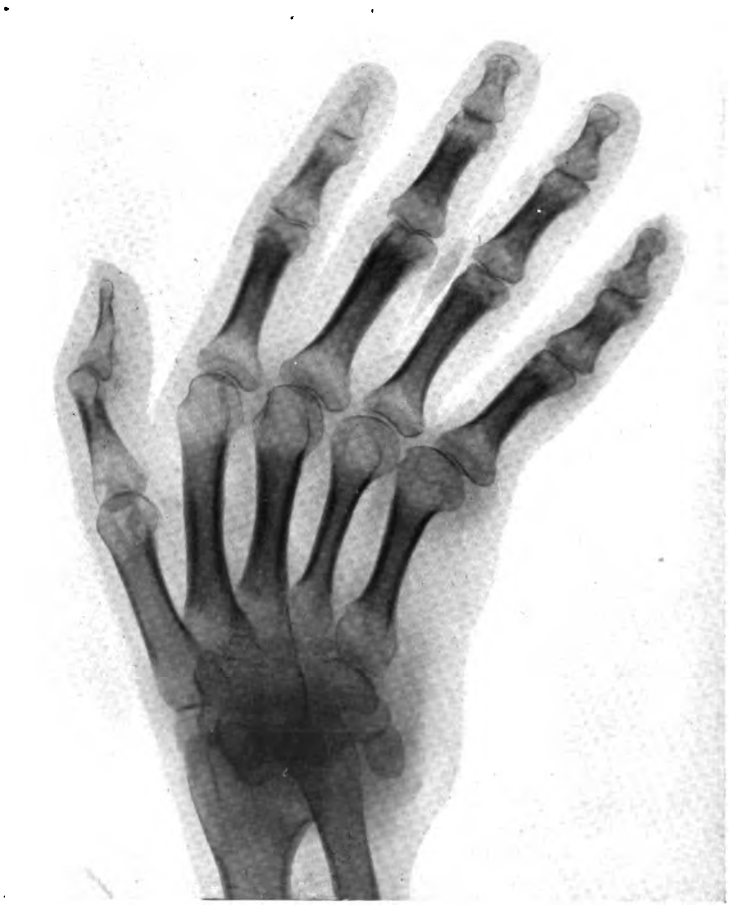


Fig. 156.—Rheumatoid arthritis ; skiagram showing the transparency of the ends of bones.

in the acute cases comes when it is suggested that an acute arthritis with pyrexia, and swelling and redness of a joint other than that of the great toe, is caused by gout. I have recently seen the difficulty in one patient in the wrist, in another in the knee. Such cases, if they are not gout, are some bacterial arthritis. If more than one joint is affected with acute arthritis at the same time, the probability is against gout, and the greater the number of joints affected the less likely is the case to be one of gout. The history and presence of urate of sodium are often conclusive in favour of gout. If pus forms, the case is almost certainly not gout, for gouty joints very rarely suppurate except late in the chronic disease. On the other hand, the implication of tendon sheaths and pain in the back of the soles of the feet are in favour of gout, though it must be remembered that both these occur in gonorrhœal cases. The presence of a source of infection is of course against gout. The difficulty is especially great in cases in which the general symptoms and arthritis, although gouty, are continuous rather than paroxysmal; but on the whole, continuously increasing severity of general symptoms is against gout. The goutily-inflamed joint looks especially shiny, is exquisitely tender on the surface, and is more painful at night than during the day. Cases of extreme difficulty have been recorded in which the first joint affected by pyæmia chanced to be that of the great toe.

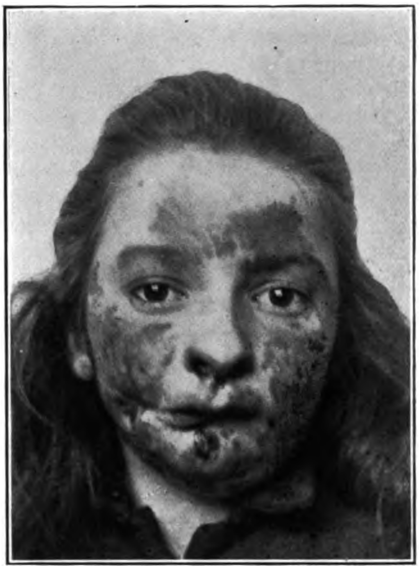


Fig. 157.—Henoch's purpura.

Probably most examples of acute arthritis said to be gouty are so, but mistakes are common about chronic gout. Many patients with chronic arthritis are quite wrongly said to have gout; usually they have osteo-arthritis. The presence of visible urate of sodium in places already mentioned (*Fig.* 158), the history of previous acute attacks, the history of gout in ancestors, the age and sex, will all help. The presence of bony outgrowths is strongly against gout, though it is not conclusive, for such may occur in true gout, either more or less all round the joint, or in the form of little nodules; but they never attain the considerable size common in osteo-arthritis. If no urate of soda is visible anywhere, the diagnosis may be very difficult; the reader should consult the principal points mentioned under the heading of osteo-arthritis. Any joints in the body may be affected by gout, but it is very rare in the joints of the trunk, the shoulder, or the hip. The spine, shoulder, and hip are commonly affected in osteo-arthritis. Urate of sodium may be seen in and near the joints as light spots in x-ray prints (*Fig.* 159).

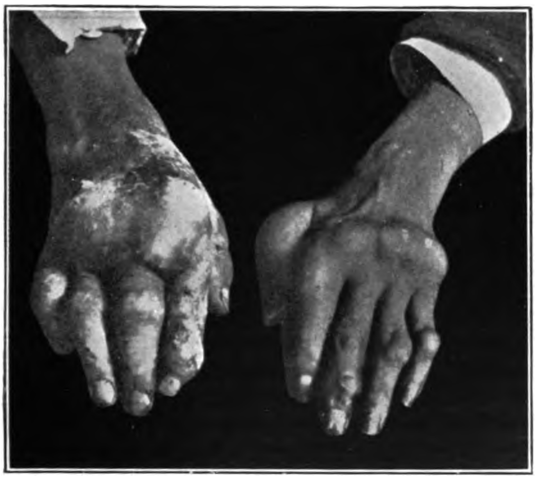


Fig. 158.—Chronic gout: deposition of urate of sodium in and near the joints.

2. **Chronic Joint Affections.**—We will now pass on to consider the diagnosis of varieties of arthritis which are for the most part chronic, but it must be remembered that many of those mentioned as acute become chronic, and their diagnosis has been described.

Osteo-arthritis is a chronic disease frequently confused with rheumatoid arthritis, from which it is completely distinct, both clinically, and from the point of view of morbid anatomy. Rheumatoid arthritis (p. 341) is primarily a disease of the synovial membrane and soft tissues of the joints. Osteo-arthritis is primarily a disease of the cartilage and bones, leading to the destruction of the cartilage, eburnation of bony surfaces, and the production of much new bone at the edges of the joint; hence bony outgrowths (osteophytes), grating of the joint, and locking of it so that movement is difficult, are common. Thickening of the synovial membrane occurs, but is less important; the ligaments become implicated and may soften; if so, the joint becomes flail-like; there may be some thickening of the tissues around the joint and some increase of synovial fluid, and then the joint becomes enlarged. It is easy to distinguish in most cases between osteo-arthritis and rheumatoid arthritis by the appearance of the affected joint. In the former we have an irregularly enlarged joint, with palpable bone excrescences and much grating; often the joint is fixed by these bony excrescences, rarely it is flail-like from destruction of ligaments; often all the causes just mentioned combine to make it very large. This is altogether different from the spindle-shaped swelling of rheumatoid arthritis (p. 341). Then osteo-arthritis is often confined to one joint, and that a large one, e.g., the knee; rheumatoid arthritis affects many joints, and is most characteristically seen in small joints, e.g., those between the first and second phalanges; but when osteo-arthritis does show itself in small joints, those most often affected are the terminal joints of the phalanges, where the bony excrescences form Heberden's nodes. Rheumatoid arthritis is far more commonly seen in young women; osteo-arthritis in women at the menopause. Rheumatoid arthritis nearly always begins with fever, although often slight; osteo-arthritis is almost always afebrile. The pulse is often rapid in those who have active rheumatoid arthritis; it is not particularly affected in those who have osteo-arthritis. The spine is more often affected by osteo-arthritis than rheumatoid arthritis, and it is quite common in the dissecting-room to find that elderly subjects have osteo-arthritis of the spine. Muscular atrophy is far greater with rheumatoid arthritis than with osteo-arthritis. Osteo-arthritis is especially liable to attack the hip—usually only one—and this form is commonest in elderly men. It was formerly called *morbus coxæ senilis*. Great care must be taken to distinguish the pain due to this from that of sciatica. The chief point of distinction is that in the latter the nerve itself is tender to pressure; but it must not be forgotten that in very rare cases osteo-arthritic outgrowths from the hip may implicate the sciatic nerve and so cause

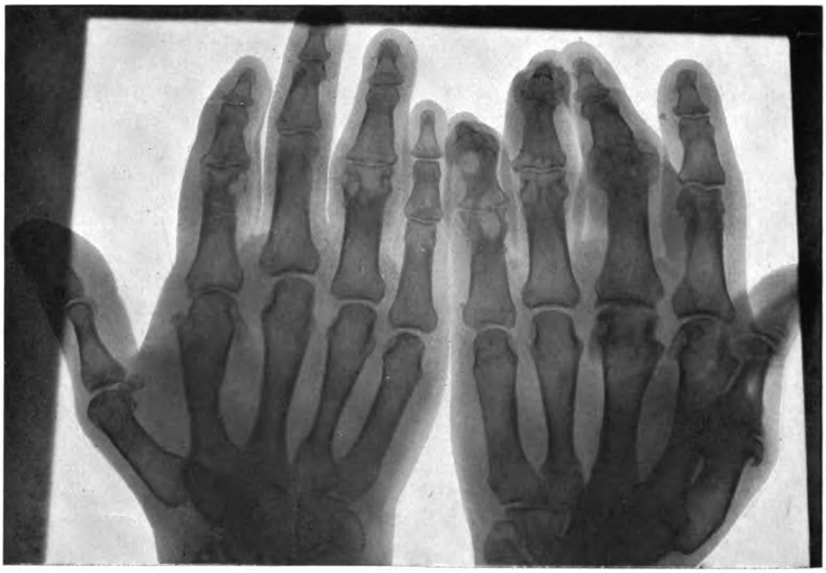


Fig. 159.—Chronic gout: skiagram of the hands, showing sodium urate deposits about the ends of many of the phalanges.

genuine sciatica. Although osteo-arthritis of the hip usually causes lameness, so many other conditions do this, e.g., sacro-iliac disease, that the symptom is of little value. The knee is the joint most often affected by osteo-arthritis—usually both, but sometimes only one is implicated. This disease of the knee is very common in women of ages between forty-five and fifty-five. They complain of pain and stiffness. Often the pain and tenderness, if present, are confined to one spot. There is usually considerable enlargement of the joint, bony irregularities may be felt, and grating and crackling on movement are very common ; these are due to bony outgrowths, erosion of cartilage, and thickening of synovial membrane, which also gives a feeling to the observer's hand placed over the joint when it is moved as though he were feeling the movement of wet sand in a bag. The grating may be heard very loudly through the stethoscope. Other joints often implicated in osteo-arthritis are the shoulder, elbow, ankle, wrist, and temporo-maxillary joint ; but what has been said about the disease in general, and that of the knee in particular, applies to them. The disease may be considerably advanced and yet confined to one joint, or any number may be affected. The points which have been especially mentioned as helping to distinguish osteo-arthritis from rheumatoid arthritis will aid in the distinction of it from other forms of arthritis. Pads (*Figs.* 160, 161) on the dorsal aspect of the joints between the first and second phalanges are not rare. They vary in size from a split pea to a hazel-nut. The joints are not diseased, but these pads, which are due to a great growth of fibrous tissue underneath the corium, have been confused with osteo-arthritis. They are often associated with Dupuytren's contracture (*Fig.* 59, p. 142). Ulnar deflection (*Fig.* 154, p. 342) is seen in osteo-arthritis, but is also present in so many other affections of the fingers and wrist that by itself it is of no value in diagnosis.

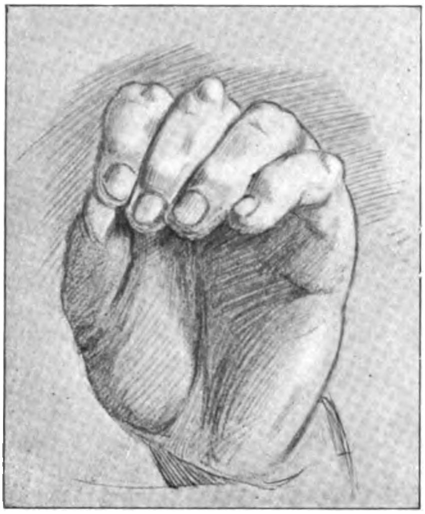


Fig. 160.—Pads on the dorsal aspect of joints ; not to be confused with osteo-arthritic changes. (*By* permission from *The Quarterly Journal of Medicine*, vol. i.)

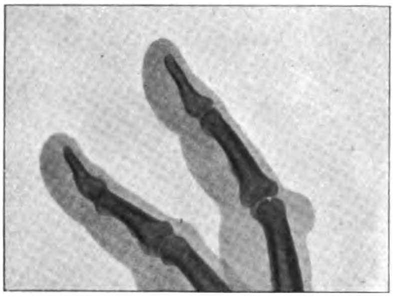


Fig. 161.—Skiagram showing that the pads depicted in *Fig.* 160 affect the soft parts and not the underlying joints. (*By* permission from *The Quarterly Journal of Medicine*, vol. i.)

Tuberculous Disease of Joints.—This is most common in children of between three and five years, and becomes rarer as age advances. About 40 per cent of the cases are in the spine, 40 per cent in the hip, 10 per cent in the knee, and the other joints which are affected not uncommonly are the ankle, shoulder, elbow, and wrist. The disease is essentially slow, so that the early stages are often overlooked. It is stated that tuberculous arthritis is so insidious in its onset that for one case in which the affection is detected and adequately treated in the first month of its existence, there are twenty in which it is allowed to drift on for three or four months, or even longer, before it is recognized. For some time there may be only slight transient impairment of movement, or an occasional twinge of pain ; gradually impaired movement, showing itself as slight lameness in the lower extremity, becomes evident, but it must not be concluded that there is no tuberculous disease of a joint because there is no impairment of movement. In the same way, although pain, often worse at night, and causing screaming, is an important sign, yet pain may be absent for a long while, or altogether. In all the joints except the shoulder and the hip—which are so deeply covered by soft parts that unless it is considerable it cannot be detected—swelling is a very important symptom, for it is almost invariably present, even in the earliest stage. It may be very slight. Although there may be no defect of movement in the early stages, sooner or later, and often quite early, this symptom develops and is of great value. Tuberculous arthritis is usually accompanied by wasting of muscles moving the joint. It must never be forgotten that a tuberculous arthritis is so slow in its development that often it is not ascribed to its correct cause ; also that for a time the symptoms are so slight that no attention may be paid to them. Mistakes are very serious, and tuberculous arthritis ought always to be present in our minds when examining a diseased joint. It is very rare for more than one, or perhaps two, joints to be affected in the same person ; tuberculous disease elsewhere, e.g., phthisis, is not common ; lardaceous disease, formerly so frequent a complication, is now seldom seen ; and general symptoms, e.g., pyrexia, are often absent and rarely extreme ; on the other hand, those affected are often pale. Bony outgrowths are not to be detected ; the joint is swollen and feels thick : hence the phrase 'pulpy knee.' There is often a history of injury to a joint which later becomes tuberculous, and then the transition from a traumatic to a tuberculous arthritis is often overlooked. Tuberculous disease of the sacro-iliac joint is particularly difficult to diagnose. Tuberculosis of the hip is often overlooked because the pain is referred to the knee, and the slight wasting of the hip muscles is not detected.

Acquired Syphilitic Arthritis.—This is most easily recognized by those who constantly bear in mind the possibility of its existence. If the characteristic pains of syphilis, which are usually worse at night, happen to occur near a joint, they may be ascribed carelessly to gout or osteo-arthritis. In the secondary stage of syphilis, and more particularly early in it, a syphilitic synovitis of any joint may occur. It is subacute, slow, is attended with stiffness, swelling, and occasionally tenderness, and usually is confined to one joint. Pain, too, may be present, but commonly neither pain nor tenderness is a prominent symptom. There is some, but not much, enlargement of the joint from distention with synovial fluid ; in a few cases the size of the joint varies considerably in a short time. These cases are often mistaken for tuberculous arthritis, but the error can usually be avoided if the patient is examined carefully and questioned for other evidence of syphilis ; and in this, and all other forms of arthritis in which there is any possibility of syphilis, a Wassermann test should be done. Syphilitic arthritis in the tertiary stage is rare : there are two varieties of it, both of which produce considerable swelling and disorganization of the joint ; in one there is a deposit of gummatous material in the subsynovial tissue, in the other in the ends of the bone. Both varieties are usually confined to a single joint, neither is painful, and both are liable to recur. Great effusion of synovial fluid is not common, but when the disease is in the subsynovial tissues the joint is enlarged and the thickening of the synovial membrane can be felt.

Congenital Syphilitic Arthritis.—In children and young adults congenital syphilis may cause an arthritis which is very like that caused by tubercle. The knees are affected most often, and the disease is often symmetrical. If there is much synovial exudation, fluctuation is detected easily ; if there is much gummatous deposit in the subsynovial tissue, the synovial membrane feels thickened and irregular. There is no pain, and very little impairment of movement. The existence of this disease must always be remembered ; the history and examination for other signs of syphilis, especially nerve deafness or interstitial keratitis, must be thorough, and the Wassermann reaction must be tested.

In *infants* congenital syphilis may cause osteochondritis in the sub-epiphyseal plate of cartilage and adjacent bone ; the epiphysis becomes separated from the shaft, so that there is motility and dull grating, as if a fracture had occurred. At the same time there is considerable swelling of the soft parts around, from the inflammation having spread to them, so that there is much swelling about the joint, although the joint itself is usually not implicated. Separation of the epiphysis from the shaft makes the limb paralyzed : hence the phrase *syphilitic pseudo-paralysis* applies to this condition. Suppuration is very rare, and the improvement with mercury is rapid. This condition may be noticed at any period from one month after birth till the age of two or three years, but it is most often seen when the child is two or three months old. It is usually multiple, and there is some tenderness and slight pain. Other signs of congenital syphilis are generally present, but if not, the condition is so characteristic that the child must at once be given mercury.

Intermittent Hydrarthrosis.—This rare disease should be diagnosed easily. It is commonest in women. Cases have been recorded between the ages of eight and fifty, but the patients are most often between twenty and thirty years old. Fluid is poured out rapidly in the joint, so that it is distinctly swollen in a few hours ; the distention attains its maximum in one or two days ; it then recedes, and has disappeared by the fourth or fifth day. The effusion leads to stiffness of the joint, and generally there is some pain, but usually very little tenderness, and the joint is neither red nor hot. The knee is affected most often ; it may be one or both knees ; if not the knee it is almost always a large joint that is the seat of the effusion. It is rare for more than two joints to be affected at once. The remarkable part of the affection is that the effusion is periodic, and often the interval of time between the attacks in the same patient is on each occasion exactly the same : thus in one patient the effusion always began on the ninth day from the beginning of the previous effusion ; the interval has been known to be less than this, and it is often more. It may be that for a period the intervals are of a certain length, and then for a period they are of a different but uniform length. In other cases there is no periodicity. In each attack the same joint or joints are affected in the same patient. After three or four years the attacks cease in most cases, but occasionally there are recurrences.

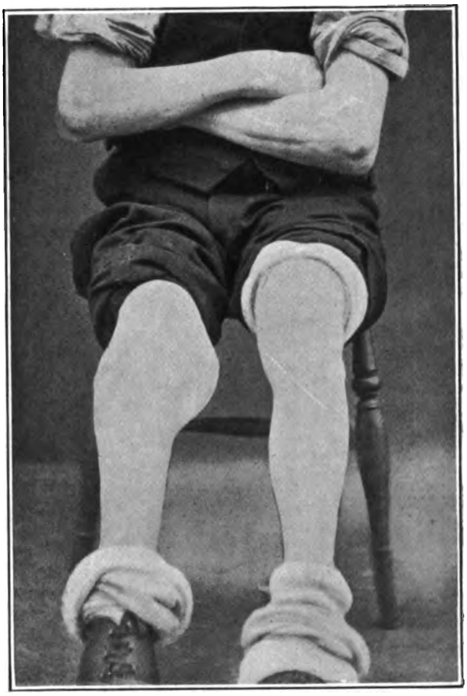


Fig. 162.—Charcot's disease of the right knee-joint in association with tabes dorsalis : showing distention, and also displacement of tibia to the right.

Charcot's Disease (*Fig.* 162).—This is the arthritis met with in tabes dorsalis, and if any patient, of such an age that he could be suffering from tabes, has chronic arthritis of a single joint, we ought always to examine him for signs of tabes. Because this is not done many cases are overlooked, for the arthritis may exist even when the patient is unaware that he has any signs of tabes. There is nothing characteristic of tabetic arthritis, and many joints affected with it might, for all the clinical symptoms of the arthritis, or from the appearances after death, be equally well affected by osteo-arthritis ; but the following points will often make one suspicious. The effusion is frequently very great—some of the biggest joints seen are those affected by tabetic arthritis ; the ligaments may be much softened, so that the joint becomes flail-like, but the growth of new bone at the edges of the joint is often quite slight, and there is considerable atrophy of bone ; thus I have seen the floor of the acetabulum as thin as paper, and because it was so thin, the pressure from the neck of the femur had expanded the floor of the acetabulum so far into the pelvis that it formed a large projection into the pelvic cavity. Tabetic arthritis is usually chronic and never acute, but it may be rapid ; thus there may be advanced destruction of the joints in a few weeks (*Fig.* 163) ; it is almost always painless ; generally large joints—e.g., knee, hip—are affected ; usually only one joint, but I have seen two. The rarefaction of the bones makes them liable to fracture. When tabetic arthritis occurs in the bones of the hand or foot the considerable swelling may cause it to be mistaken for tuberculous disease. In 75 per cent of cases of tabetic arthritis the joints affected are those of the lower extremity.

Arthritis in Syringomyelia.—In 75 per cent of the patients affected with this form of arthritis the joints affected are those of the upper extremity. There is nothing absolutely distinctive of this variety ; it resembles closely that due to tabes ; perhaps, on the whole, some of the cases more nearly resemble osteo-arthritis. Owing to the loss of pain-sensation in syringomyelia, wounds are common ; hence the joints may become septic. Mistakes in diagnosis can only be avoided by always having in mind the possibility of the occurrence of syringomyelia, and examining the patient for it. Happily it is rare, and often the symptoms of syringomyelia (p. 608) are evident before the arthritis shows itself. In about half the cases of syringomyelia there is scoliosis of the spine.

Arthritis in Hæmophilia.—In this disease blood may be poured out into either the synovial membrane or the cavity of the joint. This is probably always the result of a blow, often so slight as to pass unnoticed. It is most common in the knee and ankle. If the bleeding is at all considerable the joint swells, the rate of swelling depending upon the rate of effusion of blood. The joint is puffy; there may be fluctuation, pain on movement, and tenderness. The whole trouble often subsides, but sometimes more or less swelling persists for a time, and even if a joint gets well, relapse is likely. In other cases, either the impaired blood-supply resulting from damaged vessels or the friction of the clots leads to erosion of the cartilage, and permanent disease of the joint results. Forgetfulness of the fact that disease of the joints occurs in hæmophilia has led to the serious mistake of incising one into which bleeding has occurred. The condition is to be diagnosed by observing the other signs of hæmophilia.

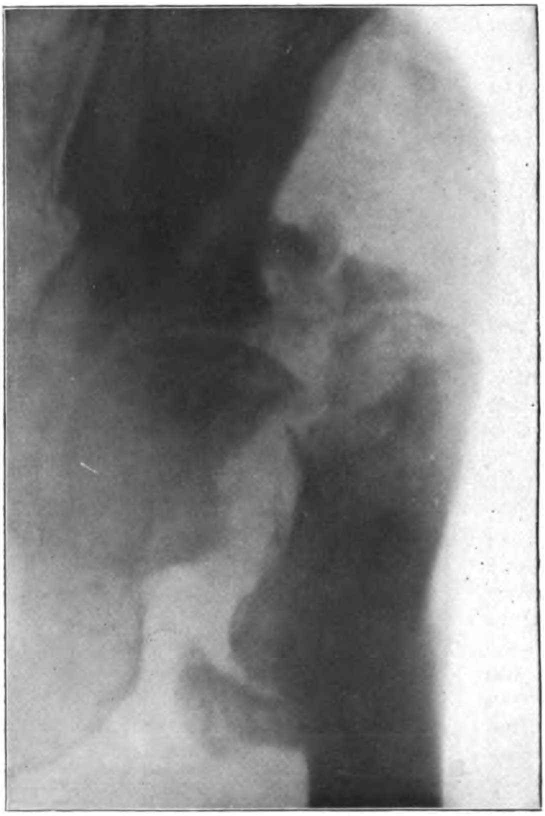


Fig. 163.—Skiagram of Charcot's disease of the hip-joint. The appearances are typical and diagnostic; they show extensive destruction of normal bone and large masses of abnormal new bone thrown out around it.

(*Skiagram by Dr. C. Thurstan Holland.*)

Malignant and Hydatid Disease of Joints.—Both these are very rare, and in each case the disease almost always begins in the adjacent bone, and therefore properly belongs to diseases of bones. Both are very serious; hydatid disease of a joint is very liable to lead to suppuration in it.

Displacement of a Semilunar Cartilage may cause much synovitis, and the cause of the latter is very apt to be overlooked. There is often a history of a wrench, or the patient complains that he feels something in the joint slip or catch; this may cause considerable pain, and there is often tenderness over the internal semilunar cartilage. Sometimes similar symptoms are produced by a thickened fringe of synovial membrane becoming nipped. This may occur in osteoarthritis. The thickened fringe may become detached, and then it forms a loose body inside the joint.

Nervous Mimicry, Neuromimesis, or Hysterical Joints.—In these cases some of the symptoms of arthritis are imitated without there being any actual disease of the joint. It is important to remember that hysteria is a disease and is not mere malingering. The malingerer can voluntarily get rid of his supposed disease if he wishes; the hysterical girl cannot, as she has not sufficient power of will. There are three main varieties: (1) The joint is kept constantly in an abnormal position, e.g., the knee may be considerably flexed; (2) The joint cannot be moved, e.g., the hand may hang down from the wrist, as in extensor paralysis, and it cannot be raised; (3) There may be acute pain in the joint. In all these cases careful examination will usually show that there are no real symptoms of arthritis: there is no swelling, no heat, no grating, no bony outgrowth, the immovably fixed joint can be moved freely under an anæsthetic; on the other hand the pain, if present, is far in excess of any pain due to arthritis, and the tenderness may be so great that the patient will not let the joint be touched. Both pain and tenderness disappear if the patient's attention is diverted, and neither keep the patient awake nor affect the general health. The pain may radiate far beyond the joint; very rarely in hysteria there is trivial swelling, but it is not such as would be produced by the distention of the synovial cavity; it is often more in the neighbourhood of the joint than over it; but nearly always there is no swelling about a hysterical joint. Usually the joint supposed to be diseased is cold; very rarely it is hot and perhaps a little red; but this phenomenon, when present, is only a local blush due to the fact that the patient's attention is directed to the joint, and it passes away quickly. The stiffness of a hysterical joint can be made out to be due to contraction of muscles and not to alteration of the joint itself; occasionally it is variable, and it is often extreme, out of all proportion to any possible joint disease; and often, too, the attitude of the joint is not that usually seen in arthritis. It has been pointed out repeatedly that muscles which move a joint usually atrophy, often rapidly, when that joint is diseased, quite apart from disuse. In hysterical affections of joints the muscles waste only slowly in proportion to the disuse of the joint.

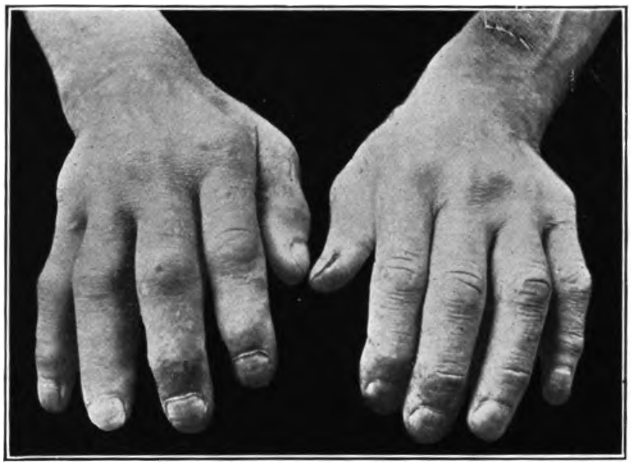


Fig. 164.—Pulmonary osteo-arthropathy: the patient had chronic fibroid phthisis. The bony parts of the fingers were becoming progressively thicker, especially in the right hand.

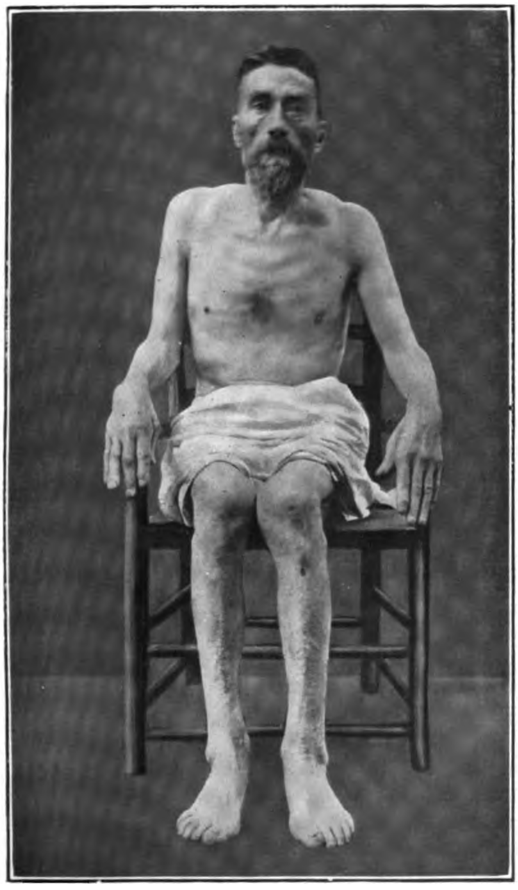


Fig. 165.—Pulmonary osteo-arthropathy: showing affection of hands and feet, and the wasting due to chronic phthisis.

Muscular Paralysis.—Often, e.g., in peripheral neuritis, the muscles which undergo rapid wasting as a result of disease of the lower motor neuron soon begin to contract, and this leads to considerable alteration in the usual position of joints. Thus, the knee and the elbow become strongly flexed, and at first it may be thought that these unusual positions are the result of disease of the joint, for long-continued chronic disease of a joint will lead to unusual permanent positions from contracture of ligaments, from the pull of muscles on a weakened joint, and from contracture of muscles wasted from arthritic atrophy; but a little estimation of the history, the condition of the joints, the symptoms of nerve disease, and the electrical reactions of the muscles will soon lead to a correct diagnosis. There is no reaction of degeneration in muscles that have atrophied secondarily to arthritis.

Hypertrophic Pulmonary Osteo-arthropathy is rare and not really a disease of joints at all, for the change consists in an enlargement of the ends of the bones, and hence the joints appear large and the patient cannot bend them properly. Often this is all that is the matter with them, but in advanced cases there is some thickening of the synovial membrane and some erosion of cartilage. The upper extremity is affected more often than the lower, and the joints usually deformed are the wrist, and the carpal and interphalangeal joints (*Figs.* 164 and 165); when the condition exists in the lower extremity the corresponding joints are implicated. In extreme cases the enlargement extends up the shafts of the affected bones. The condition is distinguished easily, for it is almost always accompanied by clubbing of the fingers, and in 80 per cent of the cases it is associated with chronic pulmonary disease, especially fibrosis, bronchiectasis, or chronic empyema, of which there are generally ample physical signs. The remaining 20 per cent of the cases are associated with such diverse conditions that text-books must be consulted; the most interesting is aneurysm of the subclavian artery. Hypertrophic osteo-arthropathy used to be confused with acromegaly; not only ought the clubbing of the fingers and the associated conditions to prevent such a mistake, but also in acromegaly there is considerable enlargement of the head and characteristic changes in the face (see *Fig.* 116, p. 237).

W. Hale White.

KIDNEY, ENLARGEMENT OF.—A renal swelling may be so slight that it is only found upon clinical examination, or it may be large enough to attract the patient's attention to it. A number of pathological changes in the kidney may give rise to a tumour of that organ, such as hydronephrosis, pyonephrosis, renal tuberculosis or abscess, new growths, and various forms of cysts in the kidney; it is necessary to be able to diagnose any one, not only from any other, but also from other tumours simulating a renal swelling.

The chief characteristic points of a renal tumour are:—

1. *The large intestine is in front of the tumour.* When either kidney is merely slightly enlarged, both large and small intestine will be in front of it; but when the organ is so enlarged as to reach the anterior abdominal wall the coils of small intestine are pushed aside. The anatomical relation of the large intestine to the kidney, and the absence of a mesentery, do not allow of the same mobility of the colon, which retains its position in front of the kidney. Hence an area of resonance can usually be obtained in front of a renal swelling; if the colon be empty, it can sometimes be felt in a thin subject and rolled by the fingers on the surface of the tumour. Bowel is never placed in front of a splenic tumour, and only rarely in front of a hepatic tumour.

2. The *area of dullness to percussion* is continuous from the lateral aspect of the swelling to the mid-line posteriorly—that is, there is no area of resonance between the mass and the vertebral spines, as in a splenic or ovarian tumour.

3. A renal tumour usually *retains the shape of the kidney*; it is rounded at its borders and poles, and does not possess any edge or sharp margin, as in splenic or hepatic swellings.

4. A *renal tumour* in the process of enlargement *projects forwards and downwards*. It may fill up the natural hollow of the loin, but very seldom causes any prominence posteriorly. A perinephric abscess, which often simulates a renal swelling, may cause a distinct prominence in the loin.

5. A renal tumour *does not descend so freely upon deep inspiration* as a splenic or hepatic tumour. A renal tumour may be movable downwards or inwards, or may be fixed in the loin by preceding inflammation. An enlarged kidney can be felt with ease bimanually, and if grasped between the two hands *can be pushed into the loin.*

6. When a renal tumour is large enough to reach the anterior abdominal wall, it commonly comes in contact with the latter at the level of the umbilicus, at the same time bulging out the ilio-costal space. There is usually a line of resonance between the upper margin of the tumour and the hepatic dullness.

7. A *varicocele* may be developed on the same side as the renal tumour.

8. With a renal tumour there may be *changes in the urine* pointing to renal disease; but on the other hand, the urine at any one time may be normal, free from blood or pus,

from the fact that the ureter of the diseased side is blocked, or that the disease does not involve the renal pelvis.

9. In exceptional cases, a tumour of the right kidney may extend upwards into the dome of the diaphragm, rotating the liver so that the anterior margin descends below the costal margin, and prevents satisfactory palpation in the renal area.

Although, from the above physical characters, it would seem that a renal tumour should present little difficulty in diagnosis, yet it is by no means infrequent to find that a tumour possessing several of these characters may give rise to considerable doubt in the determination of the organ from which it arises. The following points will assist in the diagnosis of renal swellings from other tumours with which they are likely to be confused :—

1. **Tumours of the gall-bladder** (p. 252) are placed immediately below the costal margin, so that no interval exists between the tumour and the lower margin of the liver. They are usually oval in outline, with the long axis in the line between the ninth costal cartilage of the right side and the umbilicus ; are freely movable with the respiratory movements, and movable from side to side about an axis at the costal margin. There is dullness on percussion over them, and they cannot be felt in the loin or be grasped bimanually. With a tumour of the gall-bladder there may be attacks of colic, with or without jaundice.

2. **Enlargements of the liver** (p. 366) pass downwards from beneath the costal margin so that there is no line of resonance, or area in which the hand can be depressed, between the tumour and the costal margin. Hepatic tumours do not impair the normal resonance in the loin in the same manner as a renal tumour. A tongue-shaped lobe of the liver (Riedel's lobe) may cause difficulty in diagnosis ; but here the lower margin is seldom so rounded as in a renal tumour, nor will the mass be felt in the loin on bimanual examination. A tumour or cyst in the concave aspect, or of the left lobe, of the liver is especially liable to cause error in diagnosis, whereas, on the other hand, a tumour of the right kidney which projects upwards behind the liver may so rotate the latter that its anterior margin descends below the costal margin and completely obscures the kidney. In a case of a large carcinoma of the right kidney, the liver was in this way so depressed as to render palpation of the kidney impossible.

3. **Enlargements of the spleen** (p. 628) descend from beneath the left costal margin, and have no bowel in front of them. The edge of a splenic tumour is usually well-defined and often notched, and there is resonance between the posterior aspect of the tumour and the spinal column. A splenic tumour is more movable than a renal tumour. A blood-count may help in splenic enlargements.

4. **Perinephric effusions,** whether of blood, pus, or urine, may form a tumour in the loin which upon physical examination may be mistaken for a renal swelling. A perinephritic effusion may arise from some suppurative condition of the kidney, so that the previous history and examination of the urine will not assist in differentiation ; or it may be due to conditions entirely distinct from renal disease. An effusion of blood around the kidney is, in nearly all cases, caused by an injury to the loin, and will be accompanied by other signs of injury. A perinephric abscess forms a much more ill-defined tumour than that caused by a renal swelling, is more acute in its general symptoms, such as pain and temperature, and fills up the ilio-costal space. The skin over it may be thickened or œdematous, and fluctuation may be felt to be more superficial than in a renal swelling. A perinephric abscess is most likely to result from suppuration about a carcinoma of the large bowel, from appendical inflammation, or from suppuration in a perinephric hæmatoma due to injury.

5. **Tumours arising from the pelvic organs,** from the ovary or uterus, may in some cases simulate renal tumours. An ovarian cyst with a long pedicle occupying the loin has frequently been mistaken for an enlarged or movable kidney, and any sudden attacks of pain occurring from torsion of the pedicle may be looked upon as due to renal colic. The usual ovarian cyst or uterine fibroid will seldom be confused with a renal swelling, for it is placed in the middle line of the body, can be felt to come up from the pelvis, and can be felt readily upon bimanual vaginal examination to be attached to the uterus or its appendages. These tumours also give rise to dullness anteriorly, and do not alter the normal resonance in the loin. In cases of malignant ovarian tumours associated with ascites, the lumbar resonance may be lost, but on turning the patient over upon one side, the previously dull note becomes replaced by resonance in the uppermost

loin. In the case of an ovarian cyst with a long pedicle, or of a uterine fibroid of pedunculated, subserous form, the position in the loin may sometimes suggest a renal tumour; it will be found, however, to occupy a more anterior position in the abdomen than a renal tumour, and to possess a much greater range of movement, and it does not slip back into the loin under the costal margin in the same manner as an enlarged kidney does; there is resonance posteriorly, the kidney may be actually palpated as well as the abdominal tumour, whilst a distinct connection with the pelvic organs can sometimes be traced from the tumour when the latter is drawn up.

In contradistinction to the above, a very large cystic renal swelling may be mistaken for an ovarian cyst. In may occupy the greater part of the abdomen, and even be felt per vaginam to be encroaching upon the pelvis; but on careful examination in a renal tumour of this form there will be no line of resonance between the mass and the vertebral column posteriorly, the natural hollow of the loin will be filled up, and there is frequently a distinct bulging in the lower thoracic wall, together with an increased length of the ilio-costal space on the affected side. Some assistance may be obtained from the history, when a hydronephrosis may have been first noticed as a tumour commencing under the costal margin, and gradually increasing downwards towards the iliac fossa and inwards across the median line, whereas an ovarian tumour may have been noticed to increase upwards from the pelvis.

6. **Suprarenal tumours** may occasionally be of sufficient size to form an abdominal tumour, presenting a rounded, movable swelling in the hypochondrium. It is practically impossible to distinguish them from renal tumours without laparotomy.

7. **Fæcal accumulations in the colon, cæcum,** or **sigmoid flexure** may give rise to a tumour and pain of a colicky nature in the loin. They will be distinguished from renal swellings by the general intestinal symptoms, flatulence, and the changes in form consequent on the administration of large enemata. It must be remembered that a patient with a collection of fæces in the colon may not complain of constipation, but may in fact have a small daily evacuation from the overloaded bowel.

8. **Inflammatory thickenings about the appendix** will be diagnosed from renal tumours by the situation of the pain and by the swelling being in the iliac fossa rather than in the loin. In some cases, however, the pain may be referred to the lumbar region, or an appendical inflammatory thickening may spread upwards. The onset of the trouble, the acute symptoms, and the febrile disturbance will usually distinguish these cases from renal lesions.

9. **Malignant growths of the large intestine,** especially of the ascending or descending colon, may form a tumour in the loin which closely resembles a renal swelling. The mass formed by the growth may be grasped bimanually, is movable in the same directions as a renal tumour, and comes forward under the costal margin. The percussion note over the front of the lump is resonant, and there is usually an aching pain in the loin. If the growth has infiltrated through the wall of the bowel uncovered by peritoneum, the perirenal tissues may be thickened, or albuminuria may be produced by direct invasion of the kidney, when the case will even more resemble a renal lesion. Cancer of the large intestine should be suspected if there is any irregularity in the action of the bowels, mucus or blood in the motions, or any symptom of commencing obstruction in the intestine. The tumour may be irregular and nodular, whereas a renal tumour presents rounded margins. The occurrence of a tumour in either side, associated with discomfort or palpable distention of the cæcum from the accumulation of fæces, would render a growth in the colon the more suspicious. The appearances seen with the *x*-rays at a suitable interval after a barium or bismuth meal may assist the diagnosis by showing organic intestinal stenosis.

10. **Tumours of the omentum, mesentery,** or **pancreas,** either cystic or malignant, are more median in position, do not project into the loin, and seldom resemble a renal tumour.

In many cases in which difficulty arises in the diagnosis of a swelling in the loin, great help may be obtained by pyelography—that is, the injection into the renal pelvis by means of a ureteric catheter of some substance such as collargol in a 7 per cent solution, followed immediately by radiography. By this means the renal pelvis may be definitely outlined in its normal position, and any change in position or shape may indicate that the swelling is of renal origin.

A kidney may be enlarged but yet not palpable from the fact that it is either wholly above the costal margin or obscured by the liver or the thick abdominal walls of the patient. On the other hand, a kidney may be so diseased as to be functionless and shrunken, when it cannot be felt; but the remaining organ may be enlarged in a compensatory degree and may be distinctly palpable. One must remember the danger of regarding an enlarged kidney as the diseased organ when it is in reality the only functionating one. Aching pain may be present on the functional side, as a reno-reflex pain from the disease on the other side. The kidney of normal size and position is not palpable from the abdomen, or on bimanual examination with one hand on the loin; but, in a thin subject, the lower pole may be felt to descend between the hands on the patient taking a full inspiration; if, therefore, a kidney can be felt easily on bimanual examination, it is either unduly mobile or enlarged. It is often difficult to say if a kidney that is movable is also enlarged to a slight decree; and a kidney which was thought clinically to be enlarged has often been found to be of normal size when exposed; this is in part due to the thick coverings of the abdominal wall, or to the amount of fatty tissue surrounding the organ.

If the kidney is definitely enlarged, it remains to determine the nature of the enlargement; in this one is guided, not only by the physical characters of the tumour present, but also by other symptoms that are associated with it, more especially, perhaps, by the altered characters of the urine. The kidney may be enlarged only slightly, as in tuberculosis, pyelonephritis, commencing hydronephrosis, or carcinoma; or may be enlarged to a considerable degree in polycystic disease, hydro- or pyonephrosis, and in some forms of malignant growth. From the physical examination of the enlarged organ it is often possible to say that the swelling is fluid or solid in nature, but it is seldom that a true diagnosis of the lesion can be made from palpation of the kidney alone. In the following diseases in which renal enlargement is usually present, the diagnosis must be arrived at by the consideration of associated symptoms.

In *renal tuberculosis* the disease occurs in a miliary or in a caseous form. Miliary tuberculosis occurs as a part of a general tuberculosis, usually in children, is bilateral, and causes no tumour. The caseous variety occurs as a primary disease in one kidney, in which one or several foci may be present. These enlarge and soften to form a tuberculous abscess, which invades the medullary tissues, to open eventually and discharge its contents into the renal pelvis. The kidney is enlarged and tender, and there are persistent pyuria and hæmaturia in small amount. The lining membrane of the ureter is quickly invaded by the tuberculous process, becoming thickened and infiltrated, and at the same time shortened in length, so that cystoscopically it is seen to be drawn upwards (*Plate XV*, *Fig. D*, p. 282). An early symptom of renal tuberculosis is increased frequency of micturition, even before the bladder has become infected in the downward progress of the disease. The ureter may be felt to be thickened per rectum or per vaginam, or other tuberculous foci may be found in the prostate, vesiculæ seminales, or testes in the male. A thorough search should be made for tubercle bacilli in the urine.

In *pyelonephritis* the kidney may be slightly enlarged, together with renal pain, pyuria, and general malaise. Pyelonephritis is usually bilateral, and due to some infective or obstructive lesion in the lower urinary tract, symptoms of which are usually obvious (see PYURIA, p. 574).

Malignant tumours of the kidney give rise either to an irregular nodular enlargement of the kidney, or to a general, uniform, solid tumour. There is usually aching pain in the loin, with intermittent attacks of profuse hæmaturia, the latter occurring as soon as the growth has infiltrated the renal pelvis. The bleeding may be so profuse that clots are formed in the renal calices, pyramidal in shape, which in their passage down the ureter give rise to typical renal colic. The malignant tumours found in the kidney are of several varieties, and their origin and exact pathological nature have given rise to much discussion in recent years. The true carcinoma and sarcoma exist, but are very rare, forming but a small percentage of the malignant renal tumours. They give rise to renal enlargement and intermittent hæmaturia, are usually extremely malignant, and are accompanied by early metastases. The more common type of renal tumour in the cortical portion of the kidney—the hypernephroma—was formerly supposed to arise in small aberrant areas of suprarenal tissue which are frequently found in this situation. Recent observers maintain that these tumours arise from the renal elements. The tumours commonly arise in

the upper pole of the kidney, are of yellow or brown colour, and are usually fairly well defined from the renal tissues. Microscopically, their structure is similar to that of the suprarenal gland, and their metastases are of the same nature. They were formerly classified as angiosarcoma, alveolar sarcoma, endothelioma, or carcinoma, but are now classified under the term hypernephroma. They form a comparatively slowly-growing tumour of the kidney, and give rise to less severe symptoms than the true sarcoma or carcinoma. There is aching in the loin, and enlargement of the kidney may be found on examination, but at first the symptoms are slight. Hæmaturia occurs without any apparent exciting cause, and there may be renal colic from the passage of clots down the ureter; the tumour may be of fair size before any hæmaturia is noticed.

Another form of malignant tumour that occurs in the kidney is that which is supposed to arise from embryonic tissues, and to which the name of embryoma has been applied. These tumours are formed of striated muscle (rhabdomyoma) or of mixed tissues, such as striated and non-striated muscle, cartilage or bone, and epithelial structures in tabular or glandular form. They grow in the renal tissues, expanding the latter to form a spurious caspule. They occur most frequently in children, and hæmaturia is infrequent.

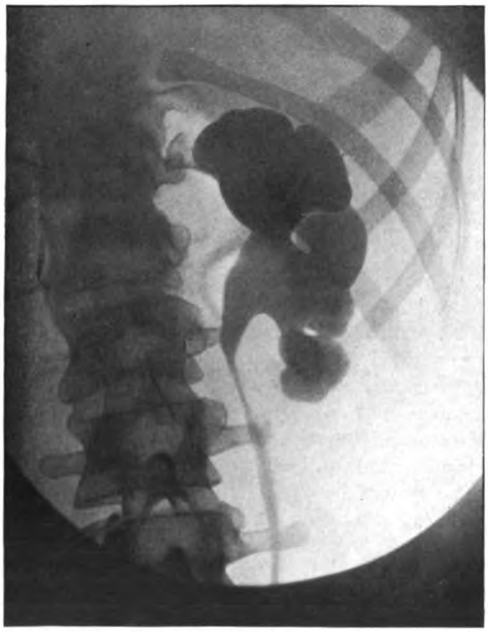


Fig. 166.—To illustrate pyelography. Collargol has been injected into the right ureter and renal pelvis after ureteral catheterization. The case was an adult female with right-sided hydronephrosis, the result of calculous disease. Two stones had been removed from this kidney a year previously, at which time the pyelographic appearances were practically the same. (*Skiagram by Dr. C. Thurstan Holland.*)

Thus, the occurrence of a renal tumour, accompanied by intermittent attacks of hæmaturia, especially if profuse, should always give suspicion of renal growth in an adult. Renal tuberculosis and calculus both may give rise to renal enlargement, but the hæmaturia is seldom profuse; with calculus, the hæmaturia is often brought on or increased by exertion, whereas with growth it may come on at any time, even during rest. At the same time, it should be remembered that both profuse hæmaturia and renal enlargement may arise from a vesical tumour which obstructs the normal flow of urine from the ureteric orifice; in all cases therefore a cystoscopic examination should be made before any operative measure is carried out. The rapid development of a *varicocele*, especially on the right side, is a point significant of renal growth.

***Hydronephrosis* and *pyonephrosis* form definite enlargements of the kidney, which may attain a large size. The tumour is oval or rounded, smooth, and gives a sense of tenseness or elasticity, whilst occasionally distinct fluctuation may be obtained. Pyelography assists the diagnosis in difficult cases (*Fig.* 166). A hydronephrosis occurs when there is a partial obstruction to the ureter, or in cases of repeated attacks of temporarily complete ureteric obstruction. Bilateral hydronephrosis may also arise from the back-pressure due to any obstruction of the normal passage of urine from the bladder. Hydronephrosis is usually unaccompanied by pain or hæmaturia; but the tumour may show marked changes in size, from the varying character of the lesion producing the obstruction; thus, if the ureter be wholly blocked, the tumour will increase in size and become more tense; whilst if the obstruction be partially relieved, the tumour will diminish, synchronously with the passage of a larger quantity of urine of low specific gravity. The presence of any obstruction to the normal flow of urine from the kidney predisposes to the onset of infection of the kidney by micro-organisms, so that a hydronephrosis may become converted into a pyonephrosis, or the latter may arise from the obstruction to the ureter of a kidney already the seat of pyelitis. The physical examination**

of a kidney distended with urine or with pus shows practically no difference between them, but with pyonephrosis other indications are usually present to assist the diagnosis. Examination of the urine will reveal the presence of pus at some time, although, if the ureter is wholly obstructed at the time of examination, pus may be absent if the other kidney and the bladder are normal. If, however, the ureter is blocked only partially, pus will be found in the urine ; in the intermittent form, pus may be present in large quantities at intervals coinciding with the decrease in the size of the renal tumour. With pyonephrosis, also, there will be the general evidence of suppuration, namely, raised temperature, sweating, pallor, and often diarrhœa. The most frequent causation of pyonephrosis is renal calculus, so that a careful inquiry into the history of the case for symptoms of calculus may give important indications, and *x*-ray examination may be of service (*Fig.* 133, p. 279) unless the stone has been passed. Very occasionally palpation of a kidney enlarged from calculous disease will give rise to distinct crepitation from the friction of one stone upon another.

A serous or *hydatid cyst* of the kidney may give rise to a tumour in the loin exactly resembling a hydronephrosis, and would usually be diagnosed as such. The discovery of hooklets (*Fig.* 18, p. 49) or hydatid elements in the urine, or in the fluid aspirated from a renal cyst, will point to the nature of the disease.

Polycystic disease of the kidney may occur in children or in adults, and forms a tumour which is commonly bilateral, though that of one side may be larger than the other. In adults the disease causes practically no trouble, except the presence of the tumour, in the early stages ; but later, symptoms of renal inefficiency develop. The tumour gives the usual physical signs of a renal enlargement, and may attain a great size on both sides. There may be aching pain in the loins and, occasionally, marked hæmaturia. The urine is of low specific gravity, is increased in amount, and in the absence of blood often contains a small amount of albumin. The disease is usually accompanied by arteriosclerosis. The character of the urine and the bilateral renal tumour are usually sufficient data upon which to form a diagnosis ; but with unilateral tumour, as occasionally occurs, the diagnosis is very difficult. A hydronephrotic or pyonephrotic kidney may give evidence of fluctuation which will not be obtained with a polycystic kidney.

R. H. Jocelyn Swan.

KNEE-JERK, ABNORMALITIES OF THE.—Before discussing the abnormalities of the knee-jerk, it is desirable to say a few words about the methods used for eliciting this valuable physical sign, and what may be considered to be its normal variations.

It is essential, if mistakes are to be avoided, to test the knee-jerk with a suitable instrument. The fingers, or the edge of a hand or of a book, are unsatisfactory. Several percussors are made for the purpose, the best being a wooden stethoscope with a moderately heavy ear-piece surrounded by a thick indiarubber ring. The patient should be either sitting or lying down. If seated in a chair, he may be directed to cross one knee over the other, or, better still, place both feet on the floor as far away from him as is possible, so long as the whole sole of each is in contact with the ground. In either position a tap on the patellar tendon will provoke a contraction of the quadriceps extensor muscle, which will extend the leg on the thigh, and may be seen or felt even if it fails to actually move the leg. If the patient is in bed, he should lie flat on his back, and be told to allow the observer to move his legs without resistance. The latter then flexes the knee by grasping the thigh above the joint and raising it until an obtuse angle is formed by the popliteal space, the foot resting on the bed. The position of the manipulator's hand will enable him to detect whether the quadriceps and hamstring muscles are sufficiently relaxed. In the case of small children or infants, it is advisable to stand at the end of the bed and to grasp the ankle with the left hand. The knee can then be flexed easily by pushing the foot towards the patient, and, at the moment when the limb feels relaxed, a tap on the patellar tendon be given with the instrument in the right hand. If difficulty is found in making the patient relax his limb in any of these positions, his attention should be directed to carrying out some other voluntary movement, such as pulling apart his grasped hands while he looks at the ceiling. This is known as 'reinforcement.'

The Normal Knee-jerk.—It is impossible to define a normal knee-jerk, because the extent of the reaction varies much in individuals and much in the same person at different times. Absence of the knee-jerk indicates an abnormality, and must be regarded as pathological. Inequality of the jerk on the two sides must also be regarded as very strong evidence of some organic morbid condition.

Abnormalities.—The knee-jerk may be exaggerated, diminished, or lost.

The knee-jerk is *exaggerated* when the reflex arc which governs the tone of the quadriceps muscle is insufficiently inhibited or controlled by the higher nervous centres. This occurs under two chief conditions, one of which constitutes a functional, the other an organic, loss of control.

Functional loss of control occurs whenever the general health or nervous tone of the patient is below par. Exaggeration of the knee-jerk may therefore be met with in *almost any constitutional ailment*, and is nearly always to be observed when a person is seriously out of health. For instance, a phthisical patient, a case of chronic renal disease, a convalescent from enteric fever, or a neurasthenic, may present very brisk jerks, and their presence may only be looked upon as an indication of a general loss of nervous tone. This fact emphasizes the necessity for never being satisfied with an examination of the knee-jerk alone in attempting to diagnose the condition of the nervous system. The examination of the knee-jerk must at least be supplemented by that of certain other reflexes, the most important of which are the abdominal and plantar. If exaggerated knee-jerks are associated with normal abdominal reflexes and with the flexor type of plantar response, and if the knee-jerks are approximately equal on the two sides, it may be assumed with some exceptions that the exaggeration is due to a functional loss of control over the reflex arc. If, on the other hand, the abdominal reflex is absent and the plantar response is of the extensor type, the exaggeration of the knee-jerk is due to some organic change in the cells of the motor area of the brain or in the pyramidal tracts which are made up of the axonal processes of those cells. Exaggeration of the knee-jerk due to organic disease is always, or nearly always, associated with other reflex changes, and particularly with the extensor type of plantar response. Frequently, but not invariably, these two signs are supplemented by the presence of ankle-clonus, by a spastic condition of the lower extremities, and by a loss of voluntary control over the vesical and rectal sphincters.

When the pyramidal tract is affected equally on both sides, the jerks will also be exaggerated equally ; but if, as in hemiplegia, one pyramidal tract is more diseased than the other, there is a corresponding difference in the knee-jerk on the two sides, that of the paralyzed leg being brisker than that of the sound limb. Inequality of the knee-jerk is also observed in certain cases of *general paralysis of the insane* for the same reason.

A very brisk knee-jerk is sometimes associated with a phenomenon which goes by the name of patellar clonus. With the limb resting relaxed and fully extended on the bed, the patella is sharply pressed towards the foot, with the result that clonic contractions of the quadriceps are provoked and continue as long as the pressure is sustained. The presence of well-sustained patellar clonus is generally indicative of organic disease.

The knee-jerk may be *diminished* as the result of some pathological processes similar to those which abolish the jerk. On the other hand, owing to the natural variations in the activity of the reflex, it is often difficult to be sure that the sluggish character of a knee-jerk is of pathological origin unless there is evidence to show that it had been obtained previously with greater facility. Most infants suffering from acute febrile or debilitating disorders present very diminished knee-jerks ; often the latter cannot be obtained at all, at the height of bronchopneumonia or epidemic diarrhœa for instance, though they return to normal as convalescence progresses.

The knee-jerk is *lost* only in organic disease, and the absence of that reflex, therefore, is evidence of some pathological process. The conditions under which the knee-jerk is lost may be classified in the following manner :—

1. Affections of the quadriceps extensor muscle, as in the myopathies.
2. Affections of the afferent path of the reflex arc, as in cases of tabes in which the lumbar region of the spinal cord is involved.
3. Affections of the anterior horn cells, such as occur when myelitis involves the third and fourth lumbar segments of the cord.
4. Affections of the efferent fibres in the anterior crural nerve innervating the quadriceps muscle, as in some forms of peripheral neuritis.
5. In complete transverse lesions of the spinal cord above the lumbar enlargement. This is usually the result of a dorsal myelitis, or of a fracture-dislocation of the vertebral column with severe injury to the cord.

6. When the intracranial pressure is greatly increased, particularly in cases of intracranial tumour, and more especially when the tumour occupies the posterior fossa of the skull.

It should be noted carefully that the absence of the knee-jerk in most cases affords evidence of some lesion of the structures which constitute the reflex arc, on the integrity of which it depends. It is a localizing sign, not necessarily a sign of some particular disease. For instance, it is quite possible for patients suffering from tabes to retain their knee-jerks so long as the morbid process has not involved the lumbar region of the spinal cord, or one knee-jerk may disappear before the other. For the same reason the jerk may be present in certain cases of acute poliomyelitis, or one may remain when the other has been lost.

It is also desirable to point out that the abolition of the knee-jerk may be the *only* indication of any affection of the nervous mechanism. For example, the knee-jerk is often lost after an attack of *diphtheria*, even when there is no evidence of paralysis of the leg muscles or of any sensory loss in the lower extremities. Another instance of the same kind is afforded by many cases of *lobar pneumonia*, especially in children, in which the pneumo-toxin is sufficiently poisonous to interfere with the sensitive patellar reflex without producing other signs of disturbance of the nervous system. In *diabetes mellitus* the knee-jerks may be absent without any further signs of peripheral neuritis developing.

Attention has been drawn to the occasional absence of knee-jerk in cases of intracranial tumour. The explanation of this is not very clear, but reference may be made to the great variability of this phenomenon from time to time. At one examination the knee-jerk is obtained ; at another, a few hours later, it is lost, perhaps to return on the following day. This ebb and flow of the knee-jerk is highly characteristic of increased intracranial pressure, and is rarely found under other conditions.

Two other forms of abnormal knee-jerk deserve brief reference. One of them is what is sometimes called the *choreic knee-jerk*. In many cases of chorea, when the leg is extended on the thigh as the result of tapping the patellar tendon, it is held in that position for an appreciable length of time before relaxation takes place and the foot falls to its former position. In *myasthenia gravis* it is sometimes, but only rarely, possible to tire out the knee-jerk. A ready response is obtained at first, but rapid repetition of the test leads to abolition of the reflex excitability, which quickly recovers itself after a short rest.

E. Farquhar Buzzard.

KRAUROSIS VULVÆ.—(See Index at end.)

KYPHOSIS.—(See CURVATURE, SPINAL, p. 153.)

LEG, ULCERATION OF THE.—(See ULCERATION OF THE LEG, p. 736.)

LEGS, PAIN IN THE.—(See under PAIN, p. 438.)

LEGS, SWOLLEN.—(See ŒDEMA, p. 411.)

LEUCOCYTOSIS is a word which has been used to denote two different conditions, namely : (1) An absolute increase above the normal of the number of leucocytes per c.mm. of blood, without distinction as to which particular variety of leucocyte is mainly increased ; and (2) An absolute increase in the total numbers of polymorphonuclear cells per c.mm. of blood. If it is used in the latter restricted sense, then there is no clinical term to express an absolute increase of all the leucocytes in the blood, whatever their kind, and it seems preferable to use the term leucocytosis in the broader sense, as being an absolute increase in the total number of white corpuscles per c.mm. of blood, indicating the kind of leucocytosis by means of a differential leucocyte count. It is used in this sense here.

The point at which an increase in the total number of leucocytes per c.mm. of blood can be called leucocytosis is arbitrary, for whereas 5000 per c.mm. is regarded as the average in health, there are considerable variations during the day, either in relation to digestion, exercise, or what not, and the same person who at one time of the day may have 5000, may at another have even as many as 14,000 per c.mm. If the differential leucocyte count remains normal, no total leucocyte count less than 15,000 per c.mm. can be regarded as abnormal, and it is not until the figure reaches 20,000 or more that much stress can be laid upon it. The numbers tend to be higher in children and in pregnant women than in other healthy individuals.

From a clinical point of view there are only two main groups of conditions in which the existence of leucocytosis is really of diagnostic importance, namely: (1) In cases of splenomedullary, lymphatic, or mixed leukæmia, the differential diagnosis of which is discussed under ANÆMIA (p. 24); and (2) In connection with infective processes, particularly those associated with suppuration.

There are many maladies in which moderate leucocytosis may occur, but in which the behaviour of the leucocytes themselves is of little diagnostic significance. Thus, whether there is or is not any leucocytosis makes little or no difference in the diagnosis of the following conditions, in all of which the number may be anything from 5000 to 20,000 per c.mm.: acute rheumatism, scarlet fever, myxœdema, intestinal obstruction, diphtheria, cholera, fœtid bronchitis, bronchiectasis, urethritis, acute follicular tonsillitis, whooping-cough, carcinoma, sarcoma, rabies. In all of these, and probably in many others, whereas many cases show no leucocytic change at all, a certain proportion exhibit leucocytosis. If there were a universal rule, either that there was leucocytosis or was not, the fact might be used in differential diagnosis; for instance, the occurrence of leucocytosis in scarlet fever might be used as a point in distinguishing it from measles, in which such leucocytosis is rare; but it is just possible that there may be a leucocytosis in a case of measles, and it is more than possible that scarlet fever may present no leucocytosis, so that whereas the general rule is to the contrary, it is not so constant as to be a safe ground upon which to make a differential diagnosis. It can only be said, broadly speaking, that whereas leucocytosis is not uncommon in the conditions already enumerated, it is upon the whole not common in measles, malaria, typhoid fever, typhus fever, influenza, smallpox, mumps, and tuberculosis other than caseous bronchopneumonia, secondary infected phthisical cavities, or tuberculous meningitis.

It is stated that certain *drugs* may produce leucocytosis, though careful experiments with some of them have by no means always confirmed this. Succinic acid, protargol, and essential oils such as turpentine, peppermint, or cinnamon, are examples of those said to produce slight leucocytosis. After severe *loss of blood*, such as may result from excessive hæmatemesis, venesection, post-partum hæmorrhage, and the like, the leucocytes may rise in a comparatively short time to over 15,000, and perhaps to over 20,000 per c.mm.

It is clear, therefore, that when so many conditions may lead to leucocytosis, its importance is much diminished as a means of differential diagnosis. One may say, however, that unless there are other clinical indications to the contrary, a definite leucocytosis of 20,000 or more, the figure sometimes reaching even 50,000 or 60,000, together with a relative increase in the polymorphonuclear cells from their normal 65 per cent to 80, 85, or even 90 per cent of all the leucocytes present in the blood, is considerable evidence of there being *suppuration* somewhere. It is worthy of notice, that in a suppuration which produces leucocytosis with a relative increase in the polymorphonuclear cells, the pus requires to be confined under pressure, for instance in an appendicular abscess, an abscess of the liver, empyema of the gall-bladder, suppurative pylephlebitis, infective cholangitis, perineal abscess, pyosalpinx, suppurating ovarian cyst, thoracic empyema, abscess of the lung, cerebral abscess, subcutaneous or pyæmic abscess, an unopened whitlow, an infected thrombosed vein, or suppurating lymphatic glands. When an abscess which has hitherto been associated with leucocytosis is opened, the number of leucocytes in the blood falls quickly to normal; there is little or no leucocytosis in conditions in which pus is not under pressure, for instance in cases of impetigo and other forms of pyodermia, superficial gangrene of the skin, suppuration connected with opened hip-joint disease or psoas abscess, and so forth. It is probably on this account that fungating endocarditis often produces a slight, but hardly ever any considerable, leucocytosis; the same applying to pyelitis, pyonephrosis, and pyelonephritis, in all of which, if there is free drainage through the ureter, leucocytosis is absent, whilst if there are abscesses in the kidney substance the leucocytosis may be considerable. Gangrene of the lung is another instance of the same kind, for there may be extensive gangrene without leucocytosis if there is free expectoration; whilst if the gangrenous tissue is prevented from escaping, leucocytosis may result. Erysipelas is an exception to the rule that superficial suppuration does not produce leucocytosis, for here considerable increase in the leucocytes is common.

Amongst diseases in which, though they are not in the ordinary sense suppurative, leucocytosis is the rule, are *acute meningitis* and *pneumonia*. Unfortunately, all forms of acute meningitis, whether tuberculous, suppurative, or meningococcal, lead to more or less leucocytosis, so that this point cannot be made much use of in the differential diagnosis between them; but upon the whole the greatest leucocytosis, up to 40,000 or more, is to be expected in the acute cerebrospinal form. The fact that pneumonia, whether of the lobar or lobular type, produces leucocytosis with a relative increase in the polymorphonuclear cells, more often than not makes it impossible to rely upon this point in determining whether or not an empyema is developing after the lung inflammation, unless it is known that up to the time of the crisis there was only a moderate leucocytosis, and that after a continuance of the fever, or a recurrence of it after the crisis, there is a greater leucocytosis, with a still further rise in the relative percentage of polymorphonuclear cells. When there has been no pneumonia, and when the physical signs are such as to suggest fluid in the chest, it is to some extent helpful to know that acute pleurisy, with effusion of the type sometimes spoken of as 'simple,' shows little leucocytosis, whereas empyema nearly always produces a considerable leucocytosis of the polymorphonuclear type.

The value of the knowledge that there is leucocytosis, when a given case has been hitherto regarded as one of some disease not associated with leucocytosis is obvious (see LEUCOPENIA below); thus, typhoid fever may have been diagnosed in a case of obscure pyrexia, in which the existence of polymorphonuclear leucocytosis indicates that the diagnosis of typhoid fever is wrong, and that there is really deep-seated suppuration, such as an appendicular abscess or a pyosalpinx. Another similar example of the possible value of this in differential diagnosis is in distinguishing malaria, in which there should be no leucocytosis, from hepatic abscess, in which leucocytosis is the rule. *Herbert French.*

LEUCOPENIA denotes the presence of a smaller number of leucocytes per c.mm. of blood than normal. When there are less than 5000 leucocytes per c.mm. one may call the condition leucopenia. There are a large number of affections in which this occurs, in most of which the fact is of little, if any, diagnostic importance. It may result from simple *starvation*, either voluntary, or due to stenosis of the œsophagus or other similar lesion. It is the rule in most *chronic intoxications*, particularly those which result from *plumbism* or poisoning by *mercury*, *arsenic*, *ether*, *alcohol*, or *morphia*. It is to be found in certain of the *severe anæmias*, more particularly *pernicious anæmia*, *aplastic anæmia*, and some cases of *lymphadenoma*, particularly in the later stages. *Acute miliary tuberculosis* is often associated with leucopenia, and so also is *tuberculous peritonitis* in more cases than not.

The chief diagnostic importance of leucopenia is in connection with two diseases in particular, namely, *typhoid fever* and *malaria*. In the former there is leucopenia almost from the beginning, and cases are not few in which, during the earlier days of the illness, before Widal's reaction could be positive, typhoid fever has seemed probable until the discovery of leucocytosis instead of leucopenia has suggested suppuration rather than typhoid, the pus being discovered subsequently perhaps in the pelvis in connection with a pyosalpinx, or in an appendicular abscess or the like. The differential leucocyte count may also assist in the same direction, for the leucopenia of typhoid fever is associated with a relative increase of the smaller lymphocytes and diminution of the polymorphonuclear cells, whilst with suppuration the reverse is the case. Leucopenia will not serve to distinguish between typhoid fever on the one hand, and either general tuberculosis, influenza, or malaria upon the other; but granted that there is a pyrexial illness suggestive of typhoid fever, the occurrence of leucopenia with a relative increase in the small lymphocytes helps considerably in confirming the diagnosis days before the Widal's reaction would be positive. The leucopenia persists unless perforation or other complication leading to pus formation supervenes.

Malaria is generally associated with a reduction of the total number of leucocytes per c.mm. down to perhaps 3000, 2000, or even less. Associated with this leucopenia there is relative increase, not in the small lymphocytes as in typhoid fever, but in the large hyaline lymphocytes; the association of these two things together, in a patient whose history points to the possibility of malaria, assists considerably in clinching the diagnosis, and it may be of particular value in cases in which quinine has been administered so that the most conclusive proof of the nature of the complaint, namely, the discovery of the malarial parasites in blood films, is not for the moment possible. One difficulty, which is not at all uncommon in the tropics, is to decide between malaria on the one hand and abscess of the liver upon the other. Leucopenia and a relative increase in the large lymphocytes strongly favours malaria, whereas an abscess would cause leucocytosis and a relative increase in the polymorphonuclear cells. *Herbert French.*

LEUCORRHŒA.—(See Discharge, Vaginal, p. 185.)

LIMPING.—(See Gait, Abnormalities of, p. 251.)

LIMPING IN CHILDREN. (See also Gait, Abnormalities of, p. 251.)—Limping in a child may be present from the time it first begins to walk, or it may develop in one who has previously walked normally. In either case it may be due to pain, to deformity, to some form of paralysis, or to any two or to all three of these. Apart from absolutely acute affections, such as suppurative osteomyelitis of the tibia, the one condition that it is most important to diagnose or exclude as the cause is tuberculous disease of the hip-joint, for limping may be the earliest and only sign of this malady, in its most curable stage if proper treatment by complete and prolonged rest is adopted forthwith. The next most important causes, generally recognized with greater ease, are tuberculous disease of the knee, the ankle, or the tarsus. Most other lesions need little discussion, for their nature is generally obvious from the history or upon careful examination of the leg and foot.

Any painful or deforming affection of the lower limb, from toes to spine, may lead to limping, and one may enumerate the following :—

1. Causes affecting the Foot or Ankle, and associated with Pain in the Foot :—

Ill-fitting boots, especially those which are too short
Chilblains
Corns
Whitlow of a toe
Blister
Abrasion of the skin on any part of the foot
Injury by a crush, blow, kick, sprain, fracture, etc.
Foreign body, such as a thorn or a needle
Rubbed heel
Inflammation of the bursa beneath the tendo-Achillis
Tuberculous dactylitis
Tuberculous disease of the tarsus
Tuberculous disease of the ankle
Rheumatic ‘ growing pains ’
Still’s disease (p. 378)
Suppurative arthritis in the ankle or foot
Peliosis rheumatica } with hæmorrhage into
Henoch’s purpura } the ankle
Hæmophilia.

2. Causes affecting the Foot or Ankle, and associated with Deformity rather than with Pain :—

Talipes varus
Talipes valgus
Talipes equinus
Talipes calcaneus
Talipes equinovarus
Talipes calcaneo-valgus
Hammer toe
Flat foot.

3. Causes affecting the Calf :—

Bruising
Infantile paralysis, with atrophy of the calf muscles
Peripheral neuritis (e.g., post-diphtheritic)
Muscular dystrophy, especially Tooth’s peroneal type (p. 60)
Chronic periostitis of the tibia or fibula : (*a*) traumatic, (*b*) tuberculous, (*c*) syphilitic
Greenstick fracture of tibia or fibula
Sarcoma of the tibia
Sarcoma of the fibula
Osteomyelitis of the tibia or fibula
Epiphysitis of the tibia or fibula
Erythema nodosum
Rickets
Phlebitis and thrombosis of veins.

4. Causes affecting the Knee-joint :—

Traumatic synovitis
Loose cartilage
Tuberculous knee-joint
Congenital syphilitic disease of the knee-joint
Rheumatic fever
Still’s disease (p. 378).
Foreign body, such as a needle, pin, or thorn
Suppurative arthritis in patellar region
Peliosis rheumatica
Henoch’s purpura } with hæmorrhage into
Hæmophilia } the joint.

5. Causes affecting the Thigh :—

Bruising
Infantile paralysis
Chronic periostitis of the femur : (*a*) traumatic, (*b*) tuberculous, (*c*) syphilitic
Greenstick fracture of the femur
Sarcoma of the femur
Osteomyelitis of the femur
Epiphysitis of the femur
Rickets
Tuberculous disease of the bursa beneath the tendon of the gluteus maximus.

6. Causes affecting the Hip-joint Region or Groin :—

Tuberculous disease of the hip-joint
Traumatic synovitis of the hip-joint
Congenital syphilitic disease of the hip-joint
Dislocation of the hip : (*a*) congenital, (*b*) from injury
Inflamed glands in the groin
Inguinal hernia
Femoral hernia
Retained testicle
Psoas abscess
Peliosis rheumatica
Henoch's purpura } with hæmorrhage into
Hæmophilia } the joint.

7. Causes affecting the Pelvis or the Lower Part of the Spine :—

Injury
Tuberculous caries
Sacro-iliac joint disease
Acute osteomyelitis of the ilium.

Many of the above conditions need no detailed discussion ; the diagnosis may be clear from the locality of the pain, the transient nature of the limp, or the existence of visible inflammation or swelling. Doubts are likely to exist in connection with the earlier stages of tuberculous disease of the digits, tarsus, ankle, or knee, and it may not be until persistence of the painful limping points to the lesion being other than simple, that the real nature of the case forces itself upon one's mind. Examination with the *x*-rays may help materially, and the same applies to other affections of the bones—periostitis, greenstick fracture, new growth. Treatment by rest will be enjoined pending diagnosis ; the latter may not become clear until the case has been watched and the course of the symptoms followed. Growing pains may be relieved by salicylates ; they are nearly always to be regarded as acute rheumatic, and a careful watch will be kept upon the heart lest the child be allowed to be up and about with acute endocarditis after the pains have been relieved by the salicylates.

It is in connection with the hip region that the diagnosis of the cause of the pain that leads to limping is so difficult, especially when the child complains that it is the knee which hurts, though the disease is really in the hip—an example of referred pain due to the obturator nerve which supplies the hip, sending a small geniculate branch to the knee. The hip-joint is embedded so deeply in muscles that it is often difficult to make out any local swelling, such as is generally distinctive of similar pulpy disease of the knee or ankle. Even *x*-ray examination may fail to give clear evidence of disease when the latter is in an early stage, though skilled radiologists may detect rarefaction of the trabeculæ in the head of the femur on comparison of the two sides, long before there is bony destruction to cause any difference in contour. When, however, the patient complains of persistent pain in the hip-joint region, less severe some days than others perhaps, yet not disappearing as the days go by ; when this pain makes him walk with a limp, or wake with a start and cry out at night ; if there are no pains elsewhere in the body, and if there is some irregular though possibly only slight pyrexia ; tuberculous disease of the hip-joint would be suspected even if the child looked well, and still more so if he looked delicate and had not a robust appetite. The suspicion would be rendered almost a certainty if, on getting the child to stand stripped, in a good light, one found definite asymmetry of the buttocks, that of the painful side being flabbier or obviously smaller than the other ; the natal fold beneath it running at the same time obliquely downwards and outwards, instead of nearly horizontally, as it should normally ; it is always the muscles immediately above an affected joint that waste first—the thigh muscles when the knee-joint is diseased, the glutei in the case of the hip-joint, and so on. It may also be noticed that the patient holds the whole limb on the painful side in an abnormal attitude—slightly abducted and outwardly rotated in the very earliest stages, so that there is apparent (but not actual) lengthening ; adducted and inwardly rotated in most cases, with apparent shortening. Mensuration is of little assistance in arriving at a diagnosis, because it is only at a much later stage, after considerable bony destruction has taken place, that there is real shortening, and the diagnosis will have been made long before this has occurred. On attempting to move the various joints of the legs when the patient is lying flat upon his back, those of the sound limb will show no limitation of movement; on the affected side the child will allow one to move the ankle and the knee freely if care is taken not to jar the hip, but when one tries to move the hip itself, muscular contractions will resist attempts at passive flexion, extension, or rotation, or the child will cry out so that one desists from trying. If one flexes both thighs slightly on the trunk, so as to allow the spine and pelvis to lie flat along a firm mattress, one finds that the sound limb can be extended until it also lies flat along the bed without altering the position of the pelvis or spine; but if one now presses the affected limb gently down to straighten it similarly on the bed, a hand held beneath the child's lumbar vertebræ detects the fact that as the thigh of the affected side gets straighter the lumbar vertebræ begin to arch; this is owing to muscular rigidity preventing free play at the acetabulum, so that instead of the head of the femur rotating as it should, without moving the pelvis, the pelvis moves with the thigh, and arching of the back results. If attention is paid to all these points, it is generally possible to detect tuberculous hip-joint disease at a comparatively early date, though in some cases one may fear it without being able to diagnose or exclude it definitely.

Sacro-iliac joint disease may simulate hip-joint disease at first sight, but on careful examination it will be found that all movements at the hip-joint can be made painlessly if the sacro-iliac joint is not jarred; whereas the least jarring to the sides of the pelvis may be acutely painful. It is generally possible to locate both pain and tenderness clearly to the sacro-iliac joint region posteriorly, and thus arrive at the diagnosis.

Psoas abscess secondary to tuberculous *caries of the spine* may also simulate hip-joint disease closely, for owing to the extension of the caseous abscess in the psoas muscle downwards over the front of the hip-joint, in the direction of the lesser trochanter of the femur, movements at the hip-joint may be very painful—especially those of extension. The patient is apt to keep the thigh flexed, and inwardly or outwardly rotated according to the direction in which the psoas abscess is burrowing. One would look for fullness of the deeper structures of the groin, or a definite swelling here; actual fluctuation obtainable from above to below Poupart's ligament is to be expected theoretically, but in practice it is very seldom obtainable. The main thing to look for in verifying the diagnosis is evidence of disease of the dorsal or lumbar vertebræ. If there is Pott's angular curvature of the spine, the diagnosis is obvious; often, however, the disease is confined to the anterior aspects of the bodies of the vertebræ, and no bony deformity is visible in the back; one would then look specially for rigidity of the back, local pain or tenderness on gentle percussion of successive spines, or deficient mobility of the dorso-lumbar region of the vertebral column when the patient attempts to bend forward, or turns from side to side. The *x*-rays are sometimes valuable in detecting the disease (*Fig.* 195, p. 460); but on the other hand, absence of *x*-ray evidence of caries does not exclude it:

Rickets sometimes gives rise to considerable difficulty on account of the ill-defined but often severe pains in the bones the patient suffers. The child is generally quite young —two or three years old—when these rickety pains are worst, and therefore cannot give a personal account of where they are. The bones may be variously deformed, the patient may be unable to walk properly, and one may have grave doubts as to whether there is not tuberculous disease of the spine, sacro-iliac joint, hip-joint, knee, or ankle, as well as rickets. Repeated examinations will be made, and yet the doubts may remain. One would, however, adopt treatment by recumbent rest and good hygiene in matters of food, air, and cleanliness in either case, and it is better to prolong the period of rest lest there is tuberculous mischief, than to curtail it on the presumption that the lesion is rickets only.

When pain causing limping in a child is confined to a single joint, such as the hip or knee, it is a safe rule to say that it is never rheumatic; neglect of this rule has led to the ruin of many a joint.

Long though the list above is, the remaining conditions given in it do not call for detailed discussion. They will be diagnosed from other symptoms presented by the case besides limping. *Herbert French.*

LINEÆ ALBICANTES, sometimes termed lineæ atrophicæ, consist of areas of skin many times longer than broad, somewhat shiny, and bluish-white, produced by atrophy of portions of the true corium.

There is no disease that really resembles them, and indeed no other condition of the skin with which they can be confounded when once they have been pointed out. Morphœa and leucodermia are the only possible exceptions, and these only under the rarest circumstances, for the patches of these affections are not linear, do not shine, and do not show those small cross wrinkles of epidermis at right angles to the axis of a linea so characteristic of lineæ albicantes ; these wrinkles can be smoothed away by stretching the skin in a direction parallel to the linea, but they return at once on relaxing the tension.

The usual meaning of these lineæ is that the skin has been unduly stretched over some fairly long period of time ; but they give not the slightest indication as to the cause of the stretching ; this caution is very necessary, because when these lineæ are found on a woman's abdomen or breasts, it is commonly assumed that they constitute evidence of a past pregnancy ; it is perfectly true that this is the commonest origin ; but any other cause of swelling, such as tumour, ascites, and even fat and œdema, etc., will produce them by stretching the skin.

They are also apt to appear over the shoulders or in front of the knees, or on the flanks, thighs, or buttocks, without it being possible to draw any conclusion as to their causation or significance.

Fred. J. Smith.

LIPS, AFFECTIONS OF THE RED PART OF THE.—The simplest affection to which the vermilion of the lips is liable is that known as 'chapping,' a condition frequently due to exposure to keen winds, and sometimes aggravated by the habit of 'picking.' In some cases the fissuring is sufficiently deep to cause appreciable pain and great disfigurement.

The vermilion of the lips may be involved also in a number of cutaneous diseases, among them *lupus vulgaris*, *lupus erythematosus*, *lichen planus*, *herpes febrilis* and *zoster*, *tinea circinata*, *urticaria*, *psoriasis*, and some forms of *syphilis*. The lesions of the epithelium of the lips, as of mucous membranes in general, are seldom characteristic enough to warrant a confident diagnosis ; in none of the above affections is the red of the lips alone affected, and guidance as to the diagnosis will be found in the more distinctive lesions of the skin.

Ordinary *eczema* is sometimes limited to the lips and immediately adjacent parts. Associated with a slightly seborrhœic condition of the scalp, there is sometimes a persistent and repeated exfoliation of the vermilion of the lips (*cheilitis exfoliativa*). In a somewhat similar yet not identical case, the lips as a whole were covered with a thick accumulation of scales, which caused them to protrude. When the scales were removed the lips were blue instead of red. The patient complained of some burning pain, but chiefly of a feeling of deadness in the lips. The condition had persisted for eleven years, and appeared to have been set up by a habit of biting the lips and tearing off the skin. The subjects of cheilitis exfoliativa are usually neurotic, and in this case there was some tendency to that condition, but there was no associated seborrhœa. In *cheilitis glandularis* there may be neither seborrhœa nor neurosis : the chronic inflammation of the lower lip, with swelling of the mucous glands, appears to originate in catarrh of the mouth and pharynx. It is chiefly the vermilion that is affected, but the inflammation spreads to the inside of the lip, and sometimes also to the neighbouring skin, which presents an erythematous aspect. The conditions here described are all rare, and are not likely to be confused with more ordinary affections of the labial epithelium.

In *syphilis* the red of the lips is sometimes the seat of the primary sore (see *Fig.* 23, p. 73), and in the secondary stage condylomata may occur in this situation. The chancre may be flattish and covered with a false membrane, or it may present itself as a crateriform infiltrated ulcer.

In *epithelioma* the lip—usually the lower one—is frequently the point of attack, the growth beginning as a slight abrasion, crack, or papule, and running the usual course. (See under TUMOURS OF THE SKIN, p. 730.)

Fordyce's disease specially attacks the red of the lips and the oral mucous membrane, the lesions consisting of small whitish or yellowish milium-like bodies, which may be discrete or coalescent, profuse or scanty. Inside the mouth the milium-like bodies are whiter than those on the lip, and are also more projecting. If subjective symptoms are present, they take the form of slight burning and itching, with a feeling of stiffness. The signs can hardly be confounded with those of any other affection. When the lesions are very abundant, they may simulate a solid patch ; but if the tissues are stretched, the milium-like bodies can be distinguished.

Perlèche is a contagious affection almost peculiar to children, and due probably to streptococci. It usually starts at both angles of the lips, as a whitening and maceration of the epithelium, which is easily detached ; it extends along the epithelium towards the middle line, involving also the surrounding skin and the mucosa of the inside of the lips. There are usually some hyperæmia and inflammation, and the feeling of heat and discomfort prompts the child constantly to lick its lips—hence *perlèche.* The affection often appears in association with impetigo contagiosa, or impetiginous stomatitis, or vesicular erythema. In some cases it can only be discriminated from the mucous patches of syphilis by the absence of other secondary signs. From herpes it can be diagnosed by its symmetry and its not beginning as a vesicular eruption. *Malcolm Morris.*

LIPURIA.—(See CHYLURIA, p. 108.)

LIVER DULLNESS, DEFICIENT.—The most common cause for diminution of the hepatic dullness is emphysema. The chest is barrel-shaped, the lower ribs are everted, and the diminution of the dullness is at its upper part. The dullness is diminished from above downwards in cases of *tight lacing,* which forces the liver down, and in cases of *hepatoptosis,* but in these two instances the hepatic dullness descends lower than is normal, so that the total liver dullness is often natural. The hepatic dullness is diminished very considerably and rapidly in *acute yellow atrophy* : the signs of this disease are so striking that the diagnosis is not as a rule difficult (p. 333). It slowly diminishes when the liver shrinks in the terminal stage of *cirrhosis.* It is often said that in *perforative peritonitis* the presence of free gas in the peritoneal cavity leads to a diminution of the hepatic dullness ; this is undoubtedly true sometimes, but the sign is so often absent that, considering there are other causes of diminution of hepatic dullness, it is unwise to lay much stress on its presence or absence in coming to a diagnosis of perforative peritonitis. Considerable *gaseous distention of the bowels* will also cause diminution of the hepatic dullness, and so will a *pneumothorax* on the right side. *W. Hale White.*

LIVER, ENLARGEMENTS OF THE.—In adults the liver is about $\frac{1}{36}$, but at birth it is $\frac{1}{24}$ to $\frac{1}{18}$ of the weight of the whole body ; therefore in infants and young children it is relatively larger than in adults. Unless this is remembered the liver may in such patients be thought enlarged when really it is of normal size. On deep inspiration, in thin people whose abdominal muscles are lax, the lower edge of the normal liver can, in the supine position, be felt to descend to touch the fingers if they are thrust up under the ribs outside the right rectus. In the upright position it may descend half an inch lower than this. In the epigastric angle a small portion of the anterior surface of the left lobe is in contact with the anterior abdominal wall, but often this cannot be felt owing to rigidity of the recti abdominales muscles.

The hepatic dullness to the left of the sternum cannot be distinguished from that due to the heart ; on the right, it begins at the middle of the ensiform process of the sternum, in the right nipple line it reaches the upper part of the fifth intercostal space, in the mid-axillary line the seventh, in the line of the angle of the scapula the ninth. In health the edge of the liver is firm and uniform, and the surface feels smooth. In excessively rare instances the whole organ is lobulated. This is probably not, as has been supposed, a developmental abnormality, but represents past disease, possibly intra-uterine. If the liver is transposed, the right lobe is small and the left large. Occasionally either lobe is dwarfed by disease, e.g., alcohol or syphilis. A tongue-like projection of the right lobe may protrude from its lower right-hand part. This projection, known as *Riedel's lobe,* is often associated with disease of the gall-bladder such as gall-stones, or with tight lacing, and is commoner in women than in men, but as it may be found in quite young children it must be regarded as sometimes an anatomical abnormality. A Riedel's lobe may give rise to great difficulties of diagnosis; if the connection between it and the liver is only peritoneum, it may be mistaken for a floating kidney, especially as in such a case there may be a band of resonance between it and the liver; or the lobe may be confused with any tumour that may be found on the right side of the abdomen. When palpating the abdomen it is often very difficult to tell the right-hand lower part of the liver from the kidney, even when there is no projection which can be called a Riedel's lobe.

Many conditions quite unconnected with the liver cause an apparent alteration in its size. Thus, a general weakness of the tissues may lead to its dropping downwards in the erect posture from laxness of its supports, which are chiefly its ligaments, and to a less extent the abdominal walls. I have known this occur in wasting diseases, the fact that the liver was not enlarged having been evident on post-mortem examination; indeed, in such a case I have known the dropped liver to be regarded as enlarged from cancer, which was believed to be the cause of the wasting, when in reality the patient was wasted because he had diabetes. Again, if the liver is somewhat enlarged from disease, its extra weight may cause it to drop, and hence it appears larger than it really is. Thus it is not uncommon for a nutmeg liver to appear during life larger than it is; but that it is not may be proved by noticing that percussion shows the upper line of hepatic dullness to have descended.

Alterations in the chest may lead to depression of the liver, which may then be thought erroneously to be enlarged. Thus, in an extreme case of fibrosis of the lungs with adherent pleura I have seen the sucking in of the ribs on inspiration lead to depression of the liver down to the umbilicus: the right lobe may be depressed into the right loin by compression of the chest due to tight lacing, this being often associated with a movable right kidney. Deformities of the chest due to rickets or curvature of the spine may lead to great depression of the liver. It may be depressed by large collections of fluid in the right side of the chest, but they must be quite large, for the fluid will more easily compress the lungs and push the heart to the left than depress the diaphragm. It may also be depressed by a right-sided pneumothorax. If in diaphragmatic pleurisy the diaphragm is not working, and is in a more or less constant position of inspiration, the liver is also constantly in this position, and hence seems to be a little depressed. Extreme pericardial effusion is said to depress the liver, but this must be very rare. It is often stated that a subdiaphragmatic abscess will depress the liver considerably; but this also is very rare, for the numerous adhesions in connection with such an abscess generally prevent depression of the liver.

Tight lacing may cause a deep furrow on the liver palpable during life. I have known so deep a furrow caused by a man's belt that the part of the liver below the furrow felt almost separated from the rest of the organ; in such a case there may be a false impression of enlargement. The effect of corsets or other artificial pressure is often such as to give an incorrect impression of enlargement, because the organ is pressed down; most commonly the liver is forced down, flattened, and elongated from above downwards. Such a pressure often leads to a transverse depression across the right-hand lower part of the right lobe, so that a more or less detached portion of it lies in the position of a Riedel's lobe.

It is quite rare for enlargement of the liver to lead to any upward extension of the hepatic dullness. This is what might be expected, for the mere weight of the enlarged liver will lead to its falling, and the resistance of the intestines and abdominal walls being much less than that of the diaphragm, it will therefore grow in the direction of least resistance, that is, downwards. Raising of the upper limit of hepatic dullness is best observed when some local disease of the liver directly implicates the diaphragm; thus, a tropical abscess of the liver growing from its upper surface will soften the diaphragm and extend upwards; a hydatid will do the same. So, when there is an extension upwards of the upper hepatic dullness, it is a local extension forming a dome-shaped addition to the hepatic dullness. Very large collections of ascitic fluid or very large abdominal tumours may push the liver up, but this is excessively rare, for such conditions will more readily compress the intestines and bulge the abdominal walls. A subdiaphragmatic abscess, by its extension of dullness up into the chest, may appear to extend the liver dullness upwards.

There are three moderately common tumours in the abdomen which may give a false impression of increase in the size of the liver. They are: *A stomach affected with malignant disease*, especially when the growth infiltrates much of the greater curvature; *malignant disease of* or *impaction of fæces in the transverse colon*; and the great omentum thickened and puckered up towards the transverse colon by some form of *chronic peritonitis*. Any of these tumours may move up and down with respiration, for they are all directly or indirectly attached to the liver; but the movement is not usually so extensive as that of the liver should be, and a band of resonance may sometimes be detected between the liver and the tumour, or the edge of the liver may be felt above it. Enlargements of the pylorus, and thickening in connection with a gastric or duodenal ulcer, may all be difficult to distinguish from an enlarged gall-bladder. The hepatic dullness may be altered by gas, and it may be almost obliterated by the descent of an emphysematous lung; slight lowering of the upper margin of the hepatic dullness from this cause is quite common. In emphysema, too, the lower ribs stand so far forward that it may be impossible to feel the lower edge of the liver. When, as in perforative peritonitis, there is free gas in the peritoneal cavity, the gas getting in front of the liver may diminish the hepatic dullness, but this sign is so often absent that its absence must not be used as an argument against the existence of perforative peritonitis. On the other hand, partial obliteration of the hepatic dullness may be due to the fact that some of the intestine is between the liver and the anterior abdominal wall, or that there is much gaseous distention of the colon behind the liver. A large collection of ascitic fluid often renders it difficult to estimate the size of the liver.

Hepatoptosis, and *wandering liver*, are terms applied to a liver which, being unduly displaceable, leaves its normal position. It is rare, but must be borne in mind, for if not a liver which is only displaced may erroneously be thought to be enlarged. Extreme degrees are met with in cases of general visceroptosis. It is commoner in women than men, and mostly after forty. The abdominal walls are usually pendulous, and as the abdominal muscles are powerful agents for keeping the abdominal viscera in place, this weakness, combined with a laxity of the hepatic ligaments, is probably the cause of the hepatoptosis. Tight lacing leads to weakness of the abdominal muscles, as well as pressing the liver down; it is flattened, often extending to the umbilicus with its greatest prominence near its lower part and on the right. It may form a protrusion of the abdominal walls; it is easily palpable, moves up and down with respiration, and can usually be pushed back into its normal position when the patient lies down; indeed, when the patient is in the supine position it sometimes goes back of its own accord, only to fall again when she stands up. It is movable laterally, and can be rotated with the hands about a horizontal axis passing through the attachment of the organ to the inferior vena cava. There is considerable diminution in, or even absence of, the hepatic dullness in the chest; in an extreme case the hand may be passed up between the liver and the ribs, and at the upper right-hand part of the abdomen there is a depression between the liver and the ribs. There may be no symptoms, but the patient usually complains of a dragging pain and a heaviness in the hepatic region. These are much worse in the erect posture. so that she may have always to lie down. Often, sudden attacks of pain occur in the right of the abdomen; these may be due to gall-stones or to a movable kidney, both often present with hepatoptosis, or to kinking of the bile-duct, which may lead to jaundice. The patients are usually neurotic, dyspeptic valetudinarians. As the abdominal muscles are weak, the blood stagnates in the abdominal vessels in the erect posture; hence faintness, palpitation, exhaustion, and dyspnœa on exertion are common, these symptoms passing away when the patient lies down.

We shall now consider each of the pathological enlargements of the liver, and indicate the chief points to be utilized in the diagnosis of each.

Venous Congestion of the Liver, or Nutmeg Liver.—There must be heart disease, usually of the mitral valve, or perhaps incompetence of it secondary to severe aortic disease, or disease of the valves on the right side, or severe disease of the myocardium, or chronic pulmonary disease, usually bronchitis, or arteriosclerosis or chronic nephritis with high blood-pressure and secondary heart failure. The enlargement of the liver is uniform, its edge is firm, its surface smooth. It may reach to the umbilicus, and as the abdominal muscles are often weak in these cases, especially in women, and the liver is very heavy from the extra amount of blood in it, the organ is often a little dropped. Pain and tenderness over it are common; due in some cases to stretching of the hepatic capsule, in others to local patches of perihepatitis. The skin over the liver may be tender. In severe cases there is often slight jaundice. Dyspeptic symptoms are frequent. Ascites may be present; if so, it is associated with the œdema due to the heart disease.

In a severe degree of nutmeg liver the organ may pulsate. If so, the tricuspid orifice must be incompetent and the right ventricle must be beating strongly; then a pulse-wave travels back in the inferior vena cava and hepatic veins to reach the liver, and makes the whole organ expand synchronously with each contraction of the right ventricle. Such incompetence of the tricuspid orifice is nearly always secondary to mitral disease. Great care must be taken not to mistake a thrust downwards of the liver by the contraction of a hypertrophied heart, or the thrust forwards by a pulsating aorta, for hepatic pulsation. The distinguishing feature of this is, that when one hand is placed on the front and the other on the back of the abdomen over the enlarged, congested liver, the two hands can be felt to be separated by the expansile pulsation. This is not the case when the pulsation is transmitted. Pulsation of the veins of the neck is generally pronounced in cases in which the liver can be felt to pulsate.

General Congestion of the Liver.—This is frequently said to be present in those who suffer from dyspepsia, but if this be so it does not give rise to a demonstrable enlargement. When, however, a European lives for many years in a tropical country he is liable to suffer from attacks of congestion of the liver, and these, when frequently repeated, lead to an enlargement called *tropical liver*. The organ is uniformly enlarged, smooth, somewhat hard, and has a uniform edge. Pain and tenderness are not such prominent features as they are in a nutmeg liver. The condition is often associated with indigestion, errors of diet—especially the taking of too much alcohol—and attacks of pyrexia. When these are present the liver becomes tender, painful, and more enlarged, and I have known such a condition mistaken for hepatic abscess. The sufferer complains of a sensation of weight in the hepatic region ; he is constipated, and the urine is full of lithates. In an extreme and chronic case the organ may extend four inches below the ribs ; the patient is depressed, irritable, and of a sallow complexion. The spleen may be enlarged.

Obstruction to the Common Bile, whatever the cause, is often associated with uniform enlargement of the liver owing to the fact that the bile is dammed back into it and so swells it up ; even simple catarrh may do this ; jaundice will always be present at the same time, and the differential diagnosis will be found discussed under that heading (see JAUNDICE, p. 327).

Suppuration within the Liver.—Multiple pyæmic abscesses within the liver, which constitute part of the condition known as portal pyæmia, generally do not cause enlargement of the liver, nor, as a rule, do multiple abscesses connected with the bile-ducts—*suppurative cholangitis*—unless there is sufficient obstruction to cause jaundice (p. 326) at the same time. Rigors (p. 594), pyrexia, and tenderness of the liver will be prominent features of most such cases. Enlargement of the liver is more often present with a *large single abscess*. There is usually a history of dysentery, for amœbic dysentery is by far the commonest cause of a large single abscess ; therefore it usually occurs in the tropics, and is then commonly called a tropical abscess. Very rarely it is secondary to other specific fevers, it may be due to suppuration round a gall-stone, or may spread from some neighbouring suppuration, e.g., a perinephritic abscess. Or again, it may be caused by suppuration of a hydatid or by injury. The presence of any of these causes may help the diagnosis ; but sometimes, even when the abscess is due to the dysenteric amœba, it may be difficult to obtain a history of dysentery ; and indeed, the dysenteric ulcers of the intestine may have healed years before the symptoms of hepatic abscess show themselves. Very rarely it appears to follow intestinal ulceration which, as far as we know, is not dysenteric ; this is so in some of the examples of single large abscesses in which the patient has never left this country. Indeed, sometimes a single large hepatic abscess is found in the tropics when the most careful search fails to find any amœbæ in the pus of the abscess, or to obtain any history of dysentery, or only a history of bacillary dysentery. Tropical abscess is most common in men between the ages of twenty-five and forty-five. It is much commoner in Europeans than natives. Eighty per cent are in the right lobe, usually in its upper part. The colour of the pus depends upon the amount of broken-down hepatic tissue present ; if there is much, it is the colour of anchovy paste ; if there is none, it is yellow, but the anchovy-paste-like pus is characteristic. Amœbæ may be found in it (see *Fig.* 25, p. 77), or more often in the granulation tissue forming the wall of the abscess. Bacteria may be present, but if the abscess has existed some time the pus is often sterile. The symptoms and physical signs to which attention must be directed are as follow :—

General.—The most important is pyrexia ; often this is the initial symptom. At first the rise of temperature is slight and irregular ; gradually it becomes hectic, with a wide daily excursion, say from 99° F. in the morning to 103° F. or 104° F. in the evening. Often the patient is thought to have malaria, but an examination of the blood will show that no malarial organisms are present, and generally there is leucocytosis, whereas in malaria the tendency is towards leucopenia. There are sometimes considerable intermissions during which the temperature is normal for weeks or months, and then there is a week or so of pyrexia. When such a case occurs in this country mistakes in diagnosis are very likely. I know of a man afflicted with tropical abscess whose attacks of pyrexia were separated by such long intervals of normal temperature that he was thought by many physicians to have recurrent influenza, and this although it was well known that he had been in the tropics. Rigors are striking and severe, and in cases of doubtful diagnosis are very suggestive of hepatic abscess, though they also make this disease resemble malaria. In mild cases the rigor is reduced to a mere feeling of chilliness. Often there are profuse sweats. The pulse is rapid in proportion to the temperature. Jaundice may be present, but generally is not. In bad cases the patient is excessively ill and weak, anæmic, and wasted to a mere skeleton. In this country we see such cases on their arrival from India, the disease having made rapid progress on board ship. On the other hand, if there are long intervals of apyrexia the patient hardly suffers in his general health ; in such cases the abscess usually has thick walls. The blood may show a great increase of polymorphonuclear cells, but this leucocytosis is often absent, especially if the pus is sterile or the abscess has thick walls. During the fever the patient has a dry tongue, anorexia, and is thirsty ; the urine is scanty and high-coloured, and may contain albumin.

Local.—The abscess is most often at the upper part of the right lobe, grows upwards between the layers of the coronary ligament, and thus forms an extraperitoneal subphrenic abscess which softens the diaphragm and pushes it up, giving a dome-shaped area of dullness varying in size from one to several inches across, added to the top of the normal line of the hepatic dullness, and best seen by mapping out the dullness with a blue pencil. It is usually posterior to the mid-axillary line. Sometimes the abscess is in such a place that a rounded swelling may be felt, or even seen, on the liver when the patient draws a deep breath. The measurement round the lower part of the chest may be longer on the affected side, the intercostal spaces may be obliterated, and if the abscess be very large, the lower ribs may bulge. Not uncommonly the abscess is of such size and position that the greatest care is necessary before it can be detected. The whole of the hepatic area should be pressed carefully by one finger, for local tenderness is often a great aid in the diagnosis. If the abscess presents in the abdomen the rectus muscle over it may be rigid. Pain is very variable ; it may be absent, it may be severe ; often coughing, drawing a deep breath, or shaking the patient, will cause pain. In about one-sixth of the cases there is pain in the right shoulder ; if the abscess is in the left lobe, there may be pain in the left shoulder. If the abscess comes close to the skin, there may be œdema and redness over it, and in excessively rare cases, fluctuation. Often the liver is enlarged generally as well as locally. If the abscess is large, it may be seen with the *x*-rays, for pus casts a very dark shadow. If it implicates the diaphragm, infection may spread through it and cause bronchitis, pleurisy, empyema, pneumonia, or gangrene of the lung, but this is not nearly so common as with other subphrenic abscesses ; hepatic pus may be coughed up from the lung when the abscess has ruptured into it, may be vomited when it has ruptured into the stomach, or may be passed by the bowel when it has ruptured into the intestine. Lastly, I would again remind the reader that in some of the chronic cases seen in this country, both the local and general signs may be so slight that great skill is necessary to detect the abscess.

Cirrhosis of the Liver.—Nearly always in this country the patient has taken more alcohol than he should, but cirrhosis of the liver, indistinguishable from alcoholic cirrhosis, occurs in children and others who have not taken alcohol, especially in Egypt. Here we have to consider only the stage in which the liver is enlarged. It has been known to weigh 200 oz., but anything over 100 oz. is exceptional. In the early stages the liver is not altered in shape, and the surface and edge are smooth ; later on, as the fibrous tissue contracts and the fat is absorbed from the cells which have undergone degeneration, the surface becomes finely uneven ; this unevenness increases, the liver becomes hard and more uneven until the irregularities on it are like hobnails, and can be felt through the abdominal wall. At this stage the edge of the liver is irregular and very firm. As the irregularity increases, the diagnosis from cancer becomes more difficult, but no irregularity from cirrhosis ever exceeds the size of a small cherry, nor is it ever umbilicated, nor does it ever enlarge suddenly; whereas a cancerous nodule may be umbilicated and may enlarge suddenly from hæmorrhage into it. Usually a cirrhotic liver is not painful; if it be, the pain is due to some local perihepatitis. Other symptoms to be looked for in cirrhosis, and to be borne in mind when making a diagnosis, are that in cirrhosis the spleen is often enlarged, and sometimes much so; the increased fibrous tissue in the liver constricting its small portal veins leads to engorgement of the veins of the stomach, and hence hæmatemesis, which may be accompanied by melæna, is common at some period in the case; and occasionally we see dilatation of the veins round the umbilicus. There are often symptoms of chronic gastritis and enteritis. Cirrhosis is commoner in men than women in the proportion of three to one; the patients are usually over thirty; there is a more frequent association of alcoholic excess in the lower classes than among those who are socially above them. Dyspepsia and morning sickness are common; there are much impairment of strength, wasting, a sallow look, dilated venules on the cheek, red nose, a furred tongue which is often tremulous, and a dry, harsh skin. The pulse becomes weaker, and when the disease is fatal its end is usually by cardiac failure. In about one-third of the cases that are ill enough to come into the hospital, the temperature is raised a little every evening (*Fig.* 167). Jaundice is or has been present in about one-third of the cases; it rarely if ever becomes as deep as that seen in cancer of the liver. Ascites occurs in 50 per cent of all cases of cirrhosis, but generally in the latest stages only; if it is abundant the enlarged liver can be felt only by dipping, which means pressing the hand down suddenly on the liver, and so, by dispersing the fluid which is over it, coming down on it. Tympanites is not uncommon in severe cases of cirrhosis, and it too may make it difficult to feel the liver. The urine is usually scanty, of high specific gravity, very acid, high-coloured, and full of urates; it generally contains urobilin and sometimes bile. Naturally sufferers from cirrhosis may have delirium tremens, but apart from this, cirrhosis towards the end is often accompanied by nervous symptoms, especially coma, and this may be so even in those who have not recently taken alcohol and who are not jaundiced. In severe cases the ankles swell, even when there is no disease of the heart, lungs, or kidneys, or pressure on the vena cava, to account for it. Lastly, it should be remembered that cirrhosis may exist without any symptoms; in between a third and a half of all cases of cirrhosis found in the post-mortem room the patient has died of something else, and in many of these cases, although he has been under observation in the wards, no symptoms of cirrhosis have been observed.

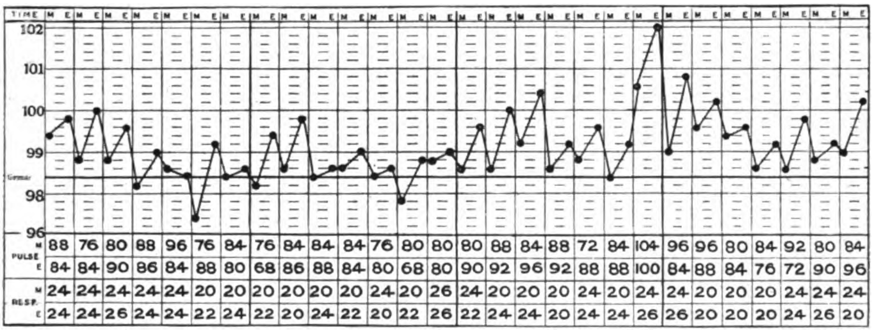


Fig. 167.—Temperature chart (morning and evening) from a case of cirrhosis of the liver, showing the tendency to slight pyrexia, especially in the evening.

The difficulties of diagnosis fall into one of two classes: the cause of Ascites (p. 45), and the cause of enlargement of the liver. If we have made out that the liver is undoubtedly enlarged, it is often a matter of great difficulty to tell whether this is due to cancer or to cirrhosis: this will be referred to in speaking of cancer. Sometimes cancer and cirrhosis are present in the same liver, but this is rare. Syphilis of the liver does not cause much difficulty, for it is uncommon at the bedside; the irregularities of the liver are much larger than the hobnails of cirrhosis; the patient who has a syphilitic liver is rarely jaundiced, and hardly ever has ascites. The symptoms of a syphilitic liver are nearly always entirely local; syphilitic disease of the liver rarely produces general symptoms. Obstruction of the common bile-duct leads to a large smooth liver ; when this is due to a gall-stone there is usually deeper jaundice than in cirrhosis, but no ascites ; the stools are quite pallid, which is very unusual in cirrhosis, and there is commonly a history of gall-stones. There is no real difficulty of diagnosis between the enlargement of malaria and ordinary cirrhosis, for so-called malarial cirrhosis occurs only in those who have drunk to excess, and is then to be ascribed to alcohol.

Hânot's Cirrhosis—often called hypertrophic biliary cirrhosis, an extremely bad name—is a very rare disease, of which the distinguishing features are : Most of the sufferers are children ; few reach the age of thirty ; it is commoner in males than females ; it lasts many years ; the liver is firm, enlarged, and smooth ; long-standing jaundice is present ; the spleen is very much enlarged. The patients are usually children of stunted growth, and therefore the liver appears very large, but the spleen is proportionately more enlarged. The liver usually remains smooth throughout, and even when towards the end of a long case it becomes a little granular, it never proceeds to anything like the irregularity of ordinary cirrhosis. Jaundice is an early symptom and lasts till the end, so it may be present many years ; very, very slowly it becomes darker. From time to time the patient has periods during which he feels ill and his temperature is raised ; but it is strange that in spite of their jaundice the children afflicted with this disease do not for years appear ill ; they may be seen running about enjoying life, with a clean tongue and a good appetite. Ascites is rare, and if present means that the end is near. In many cases the fingers become clubbed ; the clubbing is exactly like that seen in chronic fibrosis of the lung. As growth is stunted—for example, whereas the average height at 13 years is 4 ft. 9 in., a patient with this disease was only 4 ft. 1 in.—the size of the liver and spleen makes the abdomen very prominent. At the later stages there may be purpura and other hæmorrhages.

Splenic Anæmia.—This is a disease in which there are progressive enlargement of the spleen, secondary anæmia, leucopenia, a marked tendency to hæmorrhage, especially from the stomach, and in many cases a terminal stage of cirrhosis of the liver, jaundice, and ascites. The disease is often called *splenomegalic cirrhosis*, and its terminal stage of cirrhosis of the liver is frequently designated *Banti's disease*. When in this terminal stage the liver is enlarged from cirrhosis, it may be almost impossible to distinguish the condition from ordinary cirrhosis of the liver unless we know from the medical history of the case that the spleen has been enlarged for some time. Other points that may help are : on the average the spleen is much larger in splenic anæmia than in ordinary cirrhosis, so that an excessively large spleen is somewhat in favour of splenic anæmia ; hæmatemesis is an early symptom, usually present long before the stage of cirrhosis of the liver. The disease is very slow, but the patient may die before the supervention of either ascites or jaundice ; he does not often do this in ordinary cirrhosis. Anæmia is present in both conditions, but is, on the whole, severer in splenic anæmia.

Bronzed Diabetes.—In this disorder, which is very seldom seen, the liver is enlarged, hard, and cirrhotic, exactly like that of an ordinary cirrhosis : the pigmentation of the skin, which is like the discoloration due to arsenic, the absence of jaundice, and the presence of sugar in the urine, sufficiently distinguish the disease.

Syphilis of the Liver.—Syphilis when it affects the liver produces gummata in it, and leads to increased growth of fibrous tissue. Much of this is in the form of hard bands traversing the liver irregularly and leaving large areas of healthy liver substance, so that what with the presence of recent gummata, gummata that have begun to shrink, bands of fibrous tissue that have begun to contract, and pieces of normal liver, a syphilitic liver is very lumpy and irregular. It may be enlarged, and even during life this lumpiness may be felt, but the syphilitic liver does not become so large as a large cirrhotic liver, unless lardaceous disease be present ; it is much more irregular, and indeed usually resembles a cancerous more than a cirrhotic liver, but it seldom produces any clinical symptoms ; if detected during life the discovery is generally accidental ; it occurs at a younger age than cancer ; there are none of the other signs of cancer, but there may be some of syphilis, and the Wassermann reaction will be positive ; ascites and jaundice do not occur as signs of this disease unless an enlarged gland presses on the portal vein, which is so rarely the case as to be negligible ; and the liver is at most a little enlarged, never huge as in cancer.

In children, congenital syphilis may produce in the liver precisely the same effects as the acquired disease does in adults. Lardaceous disease may be due to syphilis ; it will be discussed presently.

Universal Chronic Perihepatitis may make the liver appear large, for the peritoneal coating of the whole organ is much thickened ; but as the liver itself is of normal size the apparent increase is not great, rarely exceeding an extra finger's breadth below the ribs. Such of the liver as can be felt is smooth ; the edge is uniform and thick. Usually, however, no apparent enlargement can be detected in universal chronic perihepatitis, and often the organ and its thickened capsule weigh the same as a normal liver, from which we may conclude that the liver itself is a little atrophied : in a few cases it appears actually smaller than natural, for the thin anterior edge is folded upwards under the thick peritoneal coat. There are no hepatic symptoms, e.g., jaundice, and the universal perihepatitis is only part of a general chronic peritonitis, symptoms of which, e.g., ascites and thickening of other parts of the peritoneum, may be detected on palpation.

Secondary Cancer of the Liver.—This is the commonest tumour of the liver. Generally there will be symptoms of the primary malignant disease, which in about 90 per cent of the cases is in the periphery of the portal area, but not infrequently none are present, and the patient does not know that he has anything serious the matter with him until he has symptoms of hepatic carcinoma. On the other hand, in about half the cases of hepatic carcinoma no symptoms of it are present, and it is not known to exist until a post-mortem examination is performed, for the primary disease kills while the hepatic disease is still in its early stages. Seventy-five per cent of all the patients are between 40 and 70 years old, and hepatic carcinoma is all but unknown under the age of 20. If the disease gives rise to clinical symptoms the liver can usually be made out to be enlarged both by percussion and palpation. There is no other disease in which such a huge liver may be found. I have known a cancerous liver to weigh 19 lb., and I have read of one which weighed 33½ lb. ; weights of 6 or 7 lb. are quite common. In rare cases the increase in the weight of the liver may be so great that the patient actually gains a little weight in spite of the general wasting caused by the cancer. The organ may be felt well below the ribs, even far below the umbilicus. Often it is so big that it can be seen to go up and down with each breath. Upward increase of the hepatic dullness is rare, and when present, slight. The edge of the enlarged organ can be felt to move up and down with respiration, unless it is fixed by adhesions, which is unusual. The edge is hard, and often irregular ; when the secondary nodules are numerous the whole organ feels uneven, knobby, and hard, and sometimes the lumps on it feel umbilicated ; this is absolutely diagnostic of cancer. If much softening has occurred a faint sense of fluctuation may be detected ; in a few instances local peritonitis causes a rub. Sometimes the nodules can be appreciated by the hand only when the patient takes a deep breath, for then those under the ribs come far enough down to be felt. Occasionally the cancer grows so fast that the liver obviously increases in size in a week ; very rarely a nodule may enlarge suddenly from hæmorrhage into it. Either or both these points are almost proof that the enlargement is due to carcinoma. It must not be forgotten that not all livers enlarged from malignant disease have palpable nodules, for they may be in such a situation that they cannot be felt, they may be too small to be felt, or the growth may be diffused through the whole liver. About half the patients have pain in the hepatic region, and may have it near the right shoulder and down the right arm. If the liver is very large there is a sense of dragging and fullness in the right hypochondrium. About half the patients are jaundiced. It is extremely important to remember that by far the most frequent cause of long-standing jaundice is cancer of the liver, which produces a deeper yellow of the skin than any other disease ; as time goes on this yellow changes to deep olive-green. The wasting becomes extreme, the skin dry and shrivelled, the patient becomes weaker and weaker, his pulse feebler, his respiration shallow, and finally he dies comatose. The usual symptoms of JAUNDICE (p. 324) are present. ASCITES (p. 43) is rather less frequent than jaundice, and the patient generally dies before tapping is necessary, for ascites is a late symptom. The urine usually contains much bile and lithates. Rapidly growing carcinoma of the liver is often associated with an evening rise of temperature to 99° F. or 101° F. (*Fig.* 145. p. 326). I have known it to be 102° F. every evening for weeks.

The chief difficulty of diagnosis is from cirrhosis. The large cirrhotic liver is uniformly large, and the palpable nodules are small ; if they feel bigger than small cherries the case cannot be one of cirrhosis, for hobnails are never bigger than this ; hobnails are never umbilicated, and never increase rapidly in size ; if jaundice is present and the patient has a large cirrhotic liver, the jaundice is never very deep, and remains yellow ; it never becomes the dark olive-green seen in cancer. In cirrhosis we do not get clay-coloured motions nor dilatation of the gall-bladder, but we often find a large spleen. Extreme wasting and dryness of the skin are more common in cancer. A moderate leucocytosis is often found in both diseases in the late stages. The discovery of cancer elsewhere is of course conclusive, and the history is of great help. Syphilis of the liver has already been described sufficiently to indicate the points of difference. Cases in which, owing to non-malignant obstruction of the bile-duct, usually by a gall-stone, there are enlargement of the liver and jaundice, may give rise to difficulty of diagnosis ; but these patients rarely have the extreme wasted look, with dry shrivelled skin, so frequently seen in cancer ; the hepatic enlargement is uniform and never so great as it may be in cancer ; the jaundice does not become green ; if it disappears for a time, it means that the gall-stone has shifted ; that the jaundice due to cancer should disappear is almost unknown. Rigors are common in cases of gall-stones. The age, history, and detection of growths elsewhere will be of help. As far as my experience goes, when we are in considerable doubt as to whether a patient has an impacted gall-stone or a malignant growth, exploration, if done, almost always reveals a growth. Hydatid tumours of the liver are seldom confused with cancer, for almost always these are only one or two in number, the liver is smooth and regular, and is not tender ; the hydatid tumour causes neither pain, jaundice, ascites, nor general emaciation, and it may give a thrill. There is no ordinary leucocytosis, but the patient may have eosinophilia.

Primary Carcinoma of the Liver.—This is very rare ; the liver has the same character as in the secondary form, but there are no symptoms of a primary growth elsewhere. It is almost always a disease of adult life. It is usually more rapid than secondary cancer ; most of the patients are dead within three months from the onset of symptoms, and therefore the jaundice has not time to become dark green. Wasting, and other general signs, including slight pyrexia, are present. During life, primary can hardly ever be diagnosed from secondary cancer of the liver, for even when the liver appears clinically to be the only organ affected, it often turns out that there has been primary disease elsewhere, giving no symptoms, and not detected till after death.

Secondary Sarcoma and **Embryomata of the Liver.**—These do not produce enlargement enough to be detected during life—except perhaps in the case of melanotic sarcoma secondary to a tumour of the eye, when the liver may enlarge very rapidly and to an extreme degree,—for the primary disease and the numerous secondary deposits elsewhere than in the liver soon kill the patient. If melanotic growth is suspected, the urine should be tested for melanin (p. 745).

Primary Sarcoma of the Liver is very rare, and during life cannot be distinguished from primary carcinoma.

Adenomata of the Liver are also very rare ; they are hardly ever of sufficient size to be detected during life. They are single, and I know of an instance in which a large one was operated on under the impression that it was a hydatid.

Lymphadenomata of the Liver.—New formations consisting of lymphoid tissue, generally diffused through the whole liver but sometimes occurring in nodules, may be seen in those dying from Hodgkin's disease or from lymphatic leukæmia. The nodules cannot be detected during life, but in a few cases the diffuse variety makes the liver uniformly enlarged ; it is smooth, its surface and edge are firm, it is painless, not tender, never of great size, and there is no jaundice. Leukæmic cases will be detected by the blood-count (p. 24).

Angiomata.—It is not uncommon to find small angiomata in the liver in the postmortem room, but they cannot be detected during life unless they are large enough to give symptoms which result from their size, and this is very rare. Sometimes when a large tumour of the liver has been thought to be a carcinoma, and yet the patient has seemed well enough to be suitable for operation, the growth has turned out to be a cavernous angioma, and these tumours have been excised. About fifteen of such cases are on record, and the patient was usually under fifty years of age.

Fatty Liver.—This is very common, but the enlargement of a fatty liver is usually not sufficient to be detected during life, sometimes because the patients are so obese that palpation of the liver is difficult. A fatty liver, if increased in size, is uniformly enlarged, has a rounded edge, feels a little softer than natural, with a smooth surface ; there is neither pain nor tenderness. The causes are so numerous that often they hardly help the diagnosis. The largest fatty livers are met with in phosphorus poisoning ; they then may weigh 10 or

12 lb. Severe anæmia, wasting disease, especially tubercle, and alcoholic excess, are perhaps the commonest causes. There is neither jaundice nor any other symptom that can be attributed to the disease of the liver.

Lardaceous Liver.—The liver is uniformly enlarged; the increase in size may be considerable; indeed, next to cancer, lardaceous disease causes the largest livers with which we meet. A lardaceous liver has been known to weigh 14 lb. It is so smooth that even through the skin it feels strikingly so; it is firm, and the edge is sharp and hard; it causes no pain, and is not tender. The diagnosis of this disease is much facilitated by finding lardaceous disease of other organs; thus the spleen may be enlarged considerably and uniformly, there may be albuminuria from lardaceous disease of the kidneys, or diarrhœa from lardaceous disease of the intestine. Only two causes for lardaceous disease are known, viz., long-continued suppuration, e.g., psoas abscess, bronchiectasis, chronic phthisis with cavitation, chronic hip-joint disease; and long-standing syphilis, even if this has not caused any suppuration. I have known it occur in a small child as a result of congenital syphilis. In a very few instances no cause for lardaceous disease can be discovered, but this is so exceptional that we should be very cautious of diagnosing lardaceous disease in the absence of syphilis or suppuration.

Tuberculosis of the Liver.—It is excessively rare for a tuberculous deposit in the liver to form a mass sufficiently large to be detected clinically: indeed, so rare is it that the diagnosis could not be made before exploration unless it were known that the patient had tuberculous disease at the periphery of the portal vein. Judging by morbid anatomy, a tuberculous tumour of the liver would, if discovered during life, be a solitary tumour of the liver. At an exploratory operation an irregular shaggy abscess cavity would be found, the pus of which would contain tubercle bacilli. There may be some uniform enlargement of the liver in a child suffering from general tuberculosis.

Actinomycosis, or, as it is sometimes called, *streptotrichosis* of the liver, could hardly be diagnosed without laparotomy unless the patient were known to have actinomycosis elsewhere. It is very rare, and has seldom been recognized in the liver until after the patient's death. If detected during life, there would be a local enlargement of the liver. The pus in it would be in an irregular cavity with shaggy walls and trabeculæ, and the characteristic little sulphur-coloured granules would be seen in it with the naked eye, and the ray fungus on examination with the microscope (*Plate XXVIII, Fig. S*, p. 614).

Hydatid Disease of the Liver can hardly be recognized unless the cyst causes a discoverable tumour of the liver. This may be huge. Hydatid cysts of the liver may contain thirty pints or more. If the tumour can be felt, it is rounded, smooth, localized, and regular, and thus is distinguished by its feel from cancerous or syphilitic livers, for in these the tumours are irregular and rough, and often there are one or more in different parts of the liver. A hydatid tumour is neither tender nor painful, and thus differs from an abscess. If the tumour projects from the lower part of the liver it may resemble a gall-bladder. A large hydatid cyst of the lower part of the right lobe of the liver causes considerable intra-abdominal enlargement of that lobe; on the other hand, if, as is frequently the case, it grows upwards between the layers of the coronary ligament, it pushes up the diaphragm, forming a rounded projection which may be percussed out in the chest as an addition to the top of the normal hepatic dullness: in exceptional cases the tumour may be so huge that the dome shape of the dullness is lost, and the case is apt to be regarded as one of pleuritic effusion. If a hydatid tumour is deep in the liver, the swelling feels hard; if it comes to the surface, the tumour feels tense—so tense that fluctuation is very rare. The so-called hydatid thrill perceptible in the finger lying on the tumour when it is struck by a finger of the other hand, is not often felt; it may be obtained over any tense collection of fluid, but if it be present it is of considerable diagnostic value, for other tense cysts are very unusual in the liver. Occasionally two or even three hydatid cysts are present in the same liver; each then has the characteristics of a single cyst, but the diagnosis of these cases may give much difficulty. It is excessively rare for hydatids to cause pressure symptoms; jaundice is hardly ever seen; if present, it is probably caused by rupture of the cyst into the bile-passages. A huge cyst may displace the heart. EOSINOPHILIA (p. 218), even to a considerable degree, is sometimes found when the parasitic cyst is living and active, but not when it is quiescent or obsolete. I have seen 10 per cent of eosinophiles, and even 50 per cent have been recorded. A more moderate increase is sometimes seen in cancer. Usually eosinophilia is absent in hydatid disease, but when present it is a considerable help in diagnosis. It decreases greatly after the cyst is drained. If the hydatid fluid becomes absorbed the patient may have urticaria. When the blood-serum of a patient with hydatid disease is mixed with some hydatid fluid, a precipitate may be formed after about twenty hours ; this reaction is not constant, but it does not occur when hydatid fluid is mixed with the serum of a patient who has not got hydatid disease. Hydatid fluid does not give an albuminous precipitate when heated, whereas the fluid of an ordinary pleuritic effusion does ; on the other hand, hydatid fluid gives an abundant white precipitate of silver chloride when silver nitrate is added to it. Hooklets (see *Fig.* 18, p. 49) may often be found in hydatid fluid, especially after it is centrifugalized. Hydatid cysts sometimes suppurate, and then they can hardly be distinguished from other forms of single solitary abscess. *Alveolar echinococcus disease* is very rare. No case has been recorded in England. The liver is enlarged, and there is jaundice, gradually deepening during the two or three years the patient lives. Pyrexia and gastro-intestinal symptoms are often present, and the patient dies from exhaustion.

Other cysts of the liver are very rare and very difficult to diagnose. Special textbooks dealing with the liver should be consulted about them. *W. Hale White.*

LIVIDITY.—(See CYANOSIS, p. 156.)

LOCK JAW.—(See TRISMUS, p. 729.)

LORDOSIS.—(See CURVATURE, SPINAL, p. 153.)

LUNG, HÆMORRHAGE FROM.—(See Hæmoptysis, p. 285.)

LYMPHATIC GLAND ENLARGEMENT.

A. GENERALIZED ENLARGEMENT.

There are certain diseases in which there is a tendency for all or nearly all the lymphatic glands in the body to be enlarged—generalized glandular enlargement, as distinct from enlargement of local groups of glands only. The distinction is not absolute, however, for in some patients suffering from a malady which usually causes general lymphatic glandular enlargement the changes may be confined to local groups instead of being as widespread as usual. It may be said, however, that if there is generalized enlargement of the lymphatic glands, the patient is probably suffering from one or other of the following diseases :—

Lymphatic leukæmia
Hodgkin's disease
Lymphadenoma
Lymphoma
Lymphosarcoma
Secondary syphilis
German measles
Still's disease
Plague
Tubercle, rare type.

It is of course important to be quite sure that the glands are really enlarged, and not merely palpable with greater ease than usual ; experience alone will decide this question. There are many conditions in which wasting affects the subcutaneous fat and not the lymphatic glands, so that the latter are felt with considerable ease, especially in the groins. General glandular enlargement usually implies affection of the cervical, axillary, and inguinal glands at the same time ; those in the popliteal space or above the internal condyle of the humerus are less often affected ; the various groups within the abdomen can seldom be palpated, unless perhaps in the iliac region or pelvis, whilst enlargement of the mediastinal and bronchial groups can only be surmised when there is evidence of obstruction to one or other bronchus, or when they can be demonstrated by the x-rays (*Fig.* 61, p. 149).

When a case of generalized lymphatic glandular enlargement presents itself, it is important to make a blood-count ; the blood-changes will either indicate *lymphatic leukæmia* (see ANÆMIA, p. 25), or else, if the characteristic leucocyte counts of the latter are not found, lymphatic leukæmia will be excluded. None of the other conditions exhibit pathognomonic blood-changes, although there will very often be a considerable degree of anæmia of the chlorotic type.

Hodgkin's disease nearly always starts with much swelling of one group of glands before the rest, especially those in the neck ; there is usually moderate enlargement of the spleen at the same time, and in the course of weeks or months, generalized swelling of the lymphatic glands occurs, especially those in the axillæ and within the thorax, the resultant masses sometimes being of considerable size (*Fig.* 168), though the individual glands remain distinct from one another, do not tend to break down and suppurate, and do not become fixed either to the skin or to the deeper parts, as they would do if they were tuberculous or due to secondary deposits of malignant disease. The blood-changes in Hodgkin's disease are for the most part negative (see ANÆMIA, p. 20), though in blood-films the occurrence of an occasional basophile corpuscle or myelocyte may help to clinch the diagnosis.

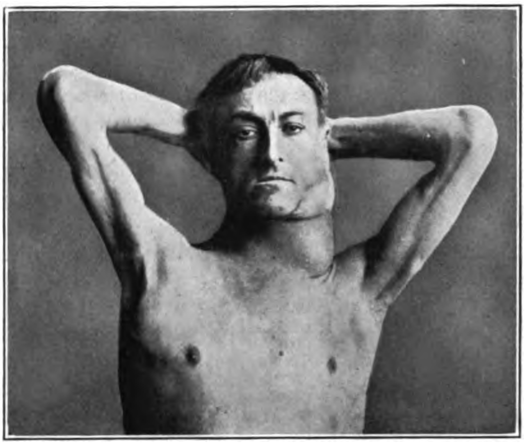


Fig. 168.—Hodgkin's disease. The lymphatic glands in the left side of the neck are very large; there is considerable overgrowth of the lymphatic glands in the right axilla, particularly along the outer border of the pectoralis major muscle; the glands in the left axilla are also enlarged, but to a less extent; even the left epitrochlear gland is visibly enlarged.

Lymphadenoma differs from Hodgkin's disease so little that some authorities use the two names as though they were synonymous; others reserve the term lymphadenoma for those cases in which splenic enlargement is not apparent whilst the affection of the lymphatic glands is very profound in one group and little marked elsewhere. *Lymphoma* is a term that has sometimes been used in the same sense.

Where *lymphosarcoma* ends and Hodgkin's disease, lymphadenoma, or lymphoma begins, it is difficult to say. If there is generalized enlargement of the lymphatic glands without much affection of the spleen, without any pathognomonic blood-changes, and with a rapidly fatal ending, the condition is spoken of as lymphosarcoma, but it might equally well be termed acute lymphadenoma.

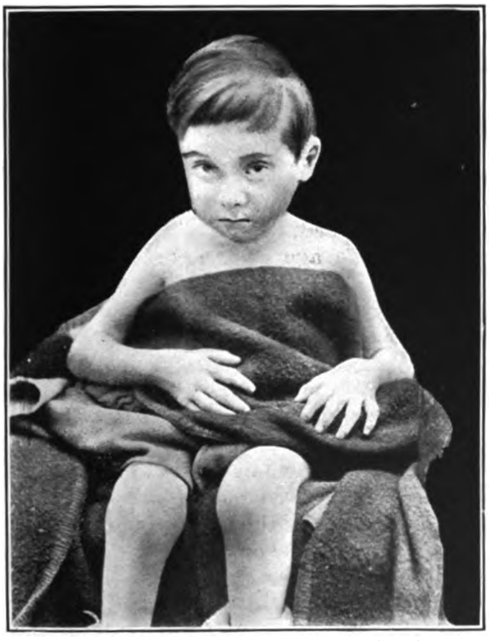


Fig. 169.—Still's disease: acute rheumatoid arthritis in childhood. The knees are swollen and their ordinary outlines lost; there is typical spindle-shaped enlargement of the first interphalangeal joints.

Syphilitic glands seldom reach any great size, only swelling, roughly speaking, to two or three times the normal; the first to be involved are those in the neighbourhood of the chancre, and therefore most often those in the groin, spreading later to all the glands in the body, including those in the occipital region, which are not as a rule affected except by syphilis, pediculosis capitis with sores, and German measles. Syphilitic glands are almond-shaped and firm, painless, or at most slightly tender, and they do not become adherent to the skin or to the deeper parts. They may remain palpable for years after all the signs of secondary syphilis have disappeared. The difficulty in their diagnosis does not arise when chancre or roseola is present: but later their nature may not be obvious unless there is a clear history of syphilis or Wassermann's serum test is positive.

German measles causes generalized enlargement of the lymphatic glands very similar to that of secondary syphilis, but the diagnosis is generally obvious from the nature of the skin eruption. The occurrence of enlarged occipital and other glands associated with a measles-like rash serves to distinguish German measles from ordinary measles, and also from scarlet fever and other erythemata.

Still's disease attracts attention primarily on account of the affection of the joints, and the enlargement of the lymphatic glands is a symptom of secondary importance. It is an affection of children (*Fig.* 169) precisely corresponding to acute rheumatoid arthritis of adults ; no joint in the body is exempt, and it is probable that the lymphatic glandular enlargement is secondary to absorption of micro-organisms from the infected joints. The patient becomes anæmic, with a tendency to pigmentation, and the spleen is enlarged as well as the lymphatic glands. The disease is unmistakeable. Similar lymphatic glandular enlargement occurs in the acute rheumatoid arthritis or infective synovitis or peri-arthritis of older persons, especially in that form which is characterized by spindle-shaped swelling of the first interphalangeal joints of the hands (*Fig.* 153, p. 342) ; but as a rule the enlargement is confined to those glands which are closest to the affected joints—epitrochlear glands, for instance, in the case of the fingers and hands, and so forth ; and the glandular enlargement disappears when the malady is in its quiescent phases, although the joint deformity remains.

Plague may be associated with very acute glandular enlargement all over the body ; the diagnosis depends largely on the history, and particularly upon the patient having been exposed to the risk of contracting plague in some infected town or port. The diagnosis may be confirmed bacteriologically.

Tuberculosis of glands is much more often local than general; occasionally, however, one meets with a case in which the inguinal and axillary as well as the cervical and internal glands are all enlarged as the result of tuberculous infection; the case then simulates lymphadenoma very closely, and it may be necessary to excise one or more of the affected glands and examine them histologically before one can be sure of the diagnosis.

B. LOCALIZED LYMPHATIC GLANDULAR ENLARGEMENT.

In all those diseases in which enlargement of the lymphatic glands may be general, it may sometimes be local, or may begin locally before it becomes general, so that in every case in which there is an affection of a local group of lymphatic glands, it is important to remember the possibility of the case being due to one of the diseases already discussed under heading *A*.

The following additional causes, however, have also to be considered, namely :—

Septic absorption, from sores, etc., on the skin or mucous membranes from which the lymphatics drain into the particular glands that are involved
Tuberculous disease
Secondary malignant disease.

Whenever there is any doubt, a blood-count should be made in order either to diagnose or exclude lymphatic leukæmia. When this can be excluded, the nature of the local glandular enlargement will generally be suggested by the age of the patient, by the characters of the glands themselves, and by their locality. We will here deal with the subject from the point of view of the particular group of glands involved.

Occipital Glands.—These seldom, if ever, become enlarged as the result of leukæmia, Hodgkin's disease, lymphadenoma, German measles, syphilis, or tuberculosis, unless there is obvious enlargement of other glands at the same time. When there is enlargement of the occipital glands and no others, by far the most likely cause is septic absorption from the posterior region of the scalp, particularly from *impetigo*, *seborrhœic dermatitis*, or most likely of all, *pediculosis capitis*. Nits should always be looked for in the hair with care, and they may sometimes be found even in ladies in whom the mode of infection may be quite inexplicable. The patients generally have much irritation of the skin at the back of the neck at the same time, and it may be attributed to the rubbing of a collar or the neck of a dress. There is generally considerable anæmia, the patient looks unwell, and often has some evening pyrexia.

Pre-auricular Glands.—The most common causes for enlargement of the pre-auricular glands are : *Septic infection* of the skin of the cheek, eyelid, ear, or temporal region of the scalp, or *epithelioma* of these regions. The occurrence of enlargement of this gland in association with an ulcer which may be rodent on the one hand, and an epithelioma on the other, does not necessarily indicate the latter, for without there being secondary deposits, the gland may become enlarged from absorption of bacteria and their products from the pus of rodent ulcer. In those very rare cases of *chancre of an eyelid* or other neighbouring part, enlargement of the pre-auricular gland may precede the generalized enlargement of the glands to which syphilis gives rise. The gland may also be the site of *melanotic sarcoma* in very rare cases, the primary growth being in the eye or a pigmented mole.

Submaxillary Glands.—The commonest cause for enlargement of these is *septic absorption* from the mouth ; tonsillitis and inflammation of the fauces are responsible for the great majority of cases in which a firm gland becomes palpable just beneath and behind the angle of the jaw ; generally the enlargement is greater upon one side than upon the other, and it may persist for days or even weeks after the causal inflammation in the tonsil has subsided. The glands are painful in the acute stages, and in a few cases the infection is so severe that the tissues break down, and suppurative adenitis with an abscess results. All kinds of inflammation of the throat may cause this glandular enlargement—ordinary simple tonsillitis, hospital sore throat, rheumatic tonsillitis, quinsy, diphtheria, scarlet fever, acute phlegmonous tonsillitis. The precise nature of the infecting organism is to be ascertained by taking swabbings from the tonsils or fauces for bacteriological cultivation. Vincent's angina less frequently produces glandular enlargement than do other severe forms of sore throat.

Inflammatory changes in glands further forward beneath the jaw are often secondary to caries of a tooth or to some variety of stomatitis, the diagnosis being ascertained by inspection of the mouth. Less acute enlargement, going on to much greater size than is the rule with inflammatory adenitis, may result from secondary deposits of *malignant disease* in the submaxillary glands when there is squamous-celled carcinoma (epithelioma) of the tongue, lip, gum, cheek, nose, palate, fauces, tonsil, pharynx, or larynx. The diagnosis in these cases depends upon the presence of an obvious primary epithelioma ; if there is any doubt as to this, a small portion of the ulcerating mass may be excised for microscopical examination. When a gummatous ulcer simulates epithelioma, the effect of iodide of potassium and mercury may point to the former, or Wassermann's serum test may be positive. A gumma of the tongue is likely to be median, an epithelioma lateral.

Cervical Glands.—Enlargement of the glands in the neck generally may be either unilateral or bilateral. If unilateral, if only a few glands are involved, and if the history is a short one, the changes are probably *inflammatory*, particularly if there has been any sore place on the skin of the neck, the buccal mucosa, or throat, or if there is evidence that the patient has been exposed recently to scarlet fever, or if there is otitis media. Acute cervical adenitis with sore throat is one of the chief features of a new epidemic malady, described by Kirkland, and referred to in detail on p. 616. Pediculosis capitis is a common cause of enlarged cervical glands in children of the poorer classes. It is sometimes difficult, however, to decide when the enlargement is merely inflammatory and when it is due to some more serious lesion, particularly *tuberculosis* on the one hand and *lymphadenoma* or *lymphosarcoma* upon the other. The longer the glandular swellings persist, the less likely is it that they are purely inflammatory. The younger the patient, and the more unsterilized cow's milk he has been drinking, the more likely are they to be tuberculous. If they are present on both sides of the neck ; if they show a tendency to become adherent to one another and to the skin ; if they are tender notwithstanding their having been present for some time, they are probably tuberculous, and the diagnosis will be settled by surgical measures, the affected glands being excised and examined microscopically. Spontaneous breaking down of the glands, with a red indolent condition of the skin around a discharging fistula, and very slow healing, are to be forestalled whenever possible ; but if they have occurred, the condition is almost certainly tuberculous in cases in which there is no question of a late stage of malignant disease. There may be confirmatory evidence in the shape of tuberculous lesions elsewhere, especially in a joint, the spine, or the peritoneum. It is noteworthy that cases of tuberculosis of the glands are even less likely than other individuals to develop ordinary phthisis, so that the absence of lung signs is no indication that the glands are not tuberculous. Lymphadenoma is sometimes so restricted in its earlier lesions as to affect the cervical lymphatic glands to a great extent, and long before any other groups are involved ; in such cases, previous to operation and microscopical examination, the nature of the glandular enlargement may be open to great doubt ; and even after an operation there may be differences of opinion, for there are some who hold that the large-celled hyperplasia exhibited microscopically by Hodgkin's-disease glands is an indication that they are only a chronic variety of tuberculosis. Clinically, the two are distinguished by the fact that tuberculous glands become matted together, while Hodgkin's-disease glands remain separate from one another, and do not soften or break down even when they have become of such great size that had they been tuberculous they almost certainly would have done so ; consequently, they do not become adherent to the skin, to one another, or to the deeper parts, and they do not cause a fistulous discharge. Enlargement of the spleen as well as of the lymphatic glands in the neck would indicate Hodgkin's disease rather than tubercle.

Secondary carcinoma of the glands in the neck is easy to diagnose when a primary growth is already known to exist ; it is generally either a squamous-celled carcinoma of the buccal cavity, especially of the tongue, lip, or palate, or else of the pharynx, larynx, or œsophagus. The cases which give rise to the greatest doubt are those in which an œsophageal growth has not caused stenosis, so that the occurrence of secondary deposits in the glands may be the first indication of anything being wrong. The patient's age will generally suffice to make tuberculosis unlikely, for tuberculous glands are far commoner in children than in adults, whilst carcinoma is a disease of the middle and later periods of life ; if there is any doubt to start with, the rapid enlargement of the glands, their extreme hardness, the way they become fixed to the deeper structures and ultimately to the skin, through which they finally ulcerate, will leave little or no doubt as to their character.

Sarcomatous glands in the neck are much rarer, the chief variety to be met with being that which has already been referred to above as acute lymphadenoma which, on account of its acuteness, is sometimes termed lymphosarcoma.

Supraclavicular Glands.—When the glands immediately above the clavicle, especially those on the left side in the region of the attachment of the sternomastoid muscle, are enlarged, without affection of any other lymphatic glands in the neck, it is highly suggestive of there being a *primary new-growth in the abdomen,* with secondary deposits ascending along the course of the thoracic duct, and exhibiting themselves in the glands close to where the thoracic duct enters the junction of the left jugular and left subclavian veins. There are, of course, many cases of abdominal malignant disease in which these glands do not become affected at all; but the value of the sign when it does occur can scarcely be exaggerated (*Fig.* 17, p. 49). No one variety of intra-abdominal carcinoma is more liable than another to produce secondary deposits here ; the primary seat may be the stomach, gall-bladder, pancreas, duodenum, colon, rectum, an ovary, or even a testicle or kidney; in not a few cases, excision and microscopical examination of the left supraclavicular gland has indicated the exact site of the primary growth. The right supraclavicular gland may be enlarged in a similar way, but far less often ; and generally not as the result of intra-abdominal but of *intrathoracic new-growth,* particularly squamous-celled carcinoma of the œsophagus. When the supraclavicular glands are affected at the same time as the axillary glands, in cases of cancer of the breast, the condition is very important as indicating that the disease has extended beyond the limits within which operative cure is likely to be possible.

Axillary Glands.—The three main causes for enlargement of the glands in one axilla without enlargement of the glands elsewhere are : *Septic absorption* from sore places upon the fingers, arm, breast, shoulder, or upper part of the back ; *secondary deposits* of carcinoma from the breast ; and *lymphadenoma. Tuberculous* axillary glands without obvious affection of those in the neck have been recorded, but they are by no means common. It is important to examine carefully for any possible source of septic absorption, for sometimes it is by no means obvious ; it may be no more than inflammation around a ragnail. Inflammatory glands are generally very painful, and they are associated with more or less pyrexia.

Lymphatic leukæmia will be excluded by the absence of pathognomonic blood-changes ; secondary malignant glands should be diagnosed when primary growth is found on careful palpation of the breast ; Hodgkin's disease will only suggest itself if inflammatory absorption, secondary growth, tubercle, and malignant disease can be excluded ; and it is probable that if the case is watched, if it is one of Hodgkin's disease other lymphatic glands will presently become enlarged also (see *Fig.* 168, p. 377), particularly those in the neck of the same or opposite side, and those in the other axilla. Enlargement of the spleen at the same time would be an argument in favour of Hodgkin's disease.

Epitrochlear Glands.—The only important cause of enlargement of the epitrochlear gland is *microbial absorption* from the fingers, hand, or forearm ; the site of primary infection may be in the skin—a whitlow, for example, or a post-mortem wound, or a dissecting-room sore; or it may be more deep-seated, as in cases of infective synovitis, arthritis, or peri-arthritis. It is important not to mistake for a simple whitlow such a lesion as a *digital chancre*, which may also cause enlargement of the epitrochlear gland before infection becomes general; if the history and the local appearance of the chancre do not suggest the diagnosis, its course and the associated secondary symptoms will indicate the nature of the case. Wassermann's serum reaction should be tried.

Mediastinal and Bronchial Glands.—These can never be palpated, and their enlargement can only be surmised when there are signs of something within the thorax obstructing one or other bronchus, or leading to laryngeal paralysis, or stenosis either of the innominate vein or of the superior or inferior vena cava. The diagnosis will be between *aortic aneurysm*, *chronic mediastinitis*, and *mediastinal new growth*. The x-rays may be of considerable value in confirming the diagnosis, and in distinguishing enlarged malignant glands from aneurysm of the aorta (*Fig.* 100, p. 209). Inflammatory or caseous bronchial or mediastinal glands seldom if ever obstruct a bronchus in the way that malignant glands do, possibly because, before they have reached a sufficient size, they have softened, and perhaps discharged their contents into the bronchial tube. When, as happens in rare cases, a caseous gland does obstruct a bronchus, it is important to remember that post-mortem evidence shows that it is very much less uncommon for a right bronchial gland to do this than a left.

Mesenteric Glands.—It is seldom possible to palpate enlarged mesenteric glands, although the diagnosis that they are swollen may often be made upon circumstantial evidence. Any *inflammatory* condition of the bowel may lead to their being enlarged, particularly if there is any breach of the mucous membrane, as in cases of *ulcerative colitis*, *dysentery*, *tuberculosis of the bowel*, or *typhoid fever*. They are greatly involved in most cases of *tuberculous peritonitis*; the masses that are felt in the abdomen, however, are hardly ever the glands themselves, but rather extensive inflammatory and caseous foci of which glands may form the nucleus. *Malignant new growth*, such as primary carcinoma of the stomach or colon, pelvic organs, or testes, may cause extensive secondary deposits in the mesenteric and retroperitoneal lymphatic glands, usually most marked in the immediate neighbourhood of the primary new growth, but extending thence in the direction of the liver until the portal glands are involved; however, without opening the abdomen, it is almost impossible to determine whether the masses felt in cases of this kind are really enlarged lymphatic glands.

Iliac and Pelvic Glands.—What has been said above in connection with mesenteric glands applies here also; but it is more often possible to determine by palpation whether or not the pelvic lymphatic glands are affected. In cases of suspected *malignant disease*, characteristic nodules of secondary deposits in lymphatic glands may be felt sometimes on careful palpation of the iliac fossa or upon making a rectal examination.

Inguinal and Femoral Lymphatic Glands.—The commonest cause by far of enlargement of the inguinal lymphatic glands and not of those elsewhere, is *septic absorption* from microbial foci in the regions whose lymphatic vessels drain into these glands; sore places should be looked for upon the toes, and between them, upon the feet, legs, thighs, buttocks, lower part of the back, scrotum, penis, perineal and vulval regions (see SORES, PENILE, ETC., p. 617); and a urethral discharge, gonorrhœal or otherwise, should also be sought for. Most of those cases will be associated with constitutional symptoms, especially pyrexia and loss of appetite, and with local pain and perhaps reddening of the skin over the inflamed glands. The latter may break down into abscesses—buboes.

Another, but far less common, cause for localized enlargement of the inguinal glands, is *secondary carcinoma*—secondary to squamous-celled carcinoma of the scrotum, prepuce, penis, perineal region, anus, clitoris, labium majus, vagina, leg, or foot. In such cases the diagnosis will become obvious when the primary growth is found, and if doubt exists as to the nature of any such ulcerating sore, the result of microscopical examination of a small portion excised will clinch the diagnosis.

Melanotic sarcoma is another rare but very important cause of enlargement of the inguinal lymphatic glands; sometimes, when the primary growth is hardly larger than a pea, arising in connection with the skin of one of the toes, or perhaps a mole, the inguinal glands may be as big as pigeon's eggs, rapidly growing and comparatively painless. The nature of this enlargement may be quite obscure unless the dark tinge of the growth can be seen through the skin, or there is melanuria (*Plate XXXIV*, *Fig.* 10, p. 748), or a careful examination reveals a small primary new growth of the skin, or unless surgical measures are adopted for their removal.

Popliteal Glands are seldom felt, and when palpable they are discovered as a rule rather because there are enlarged lymphatic glands elsewhere than from any symptoms which attract notice to the popliteal space itself. Almost the only cause for their enlargement is septic absorption either from joints or from the skin of the toes, feet, or legs, comparable to the conditions which produce enlargement of the epitrochlear glands of the arm.

Lymphatism or **Status Lymphaticus.**—Much attention has recently been attracted to the fact that in young persons under puberty who have died as the result of poisoning by anæsthetics, or of what under ordinary circumstances would be regarded as inadequate causes, such as operation for the removal of tonsils, circumcision, and so forth, the internal lymphatic glands and tissues, particularly the tonsils, thymus gland, bronchial and mesenteric glands, Peyer's patches, and the solitary follicles of the intestines, are considerably larger than is usually the case in post-mortem examinations upon patients of similar ages who have died of other diseases. It is generally stated that the condition referred to is pathological, and it has been described as the status lymphaticus or lymphatism. It is doubtful, however, whether this is not really the normal condition of the lymphatic tissues at this age, for very similar appearances are to be found in the bodies of children killed, not slowly by disease, but suddenly by accidents. In any case, it is almost impossible to diagnose the so-called status lymphaticus during life, for if it is an affection at all, it is one of the internal lymphatic tissues and not of the peripheral and easily palpable lymphatic glands. It is doubtful, however, if it is really a pathological state, though deaths produced by anæsthetics are being accredited to it. *Herbert French.*

MACROGLOSSIA.—(See SWELLING OF THE TONGUE, p. 698.)

MACULES are circumscribed discolorations or decolorizations of the skin, without noticeable elevation or depression. They may be due : (1) To the passage of blood, or of the colouring matter of the blood, into limited areas of the skin, as in purpura ; (2) To hyperæmia, either arterial or venous, as in erythema ; (3) To dilatation of the vessels of the skin, or the formation of new vessels, as in capillary nævus and telangiectases ; (4) To changes in the pigmentation of the skin, whether of the rete or of the corium—on the side of excess as in chloasma, or on that of deficiency as in leucodermia ; and such changes may result from the administration of drugs such as arsenic and chloral, or may be an expression of trophoneurosis, as in glossy skin.

Macules of the second and third groups are effaced temporarily by pressure ; those of the first and fourth remain unaltered.

Macules may be inflammatory as in the rose spots of enteric fever, or non-inflammatory as in purpura ; congenital as in moles, or acquired as in the exanthemata ; temporary as in drug rashes, or permanent as in leucodermia ; scanty as sometimes in leucodermia, or abundant as in roseola. They may be attended by subjective symptoms (e.g., itching) as in drug rashes ; but generally there are no such accompanying symptoms. Usually round or roundish, they may be oval, or irregular ; they also vary greatly in definition. In colour they may be red, brown, or yellow, in various shades. In size they vary from a mere speck to, say, the area of a man's hand ; if very widely diffused, as in malaria, the pigmentation is usually styled a discoloration. Most frequently a primary lesion, as in lentigo, the macule may also be secondary to burns, blisters, excoriations, and eruptions of various kinds, erythematous, vesicular, bullous, papular, pustular, and eczematous. The brown spots which follow traumatic or purpuric ecchymoses, hæmorrhagic urticaria, varicose eczema, etc., form a special group of macules in which the pigment is hæmosiderin. If a macule takes on a slight degree of elevation it is sometimes styled a maculo-papule.

The differential diagnosis of the erythemas, of which the lesions are for the most part too diffuse to be regarded as macules, is set out in the articles on ERYTHEMA (p. 222) and NODULES (p. 402) ; that of the purpuras in the article on PURPURA (p. 552) ; that of leucodermia, sclerodermia, morphœa, the various forms of chloasma, and the discolorations due to the use of drugs, in the article on PIGMENTATION OF THE SKIN (p. 527). Nor need the most familiar macule, that which occurs in lentigo (freckles), be described here, for the only affection with which it can be confused is xeroderma pigmentosum, the diagnosis from which is given under TUMOURS OF THE SKIN (p. 730), where also will be found the differential diagnosis of another macular affection, xanthoma in its various forms. Tinea versicolor has been dealt with under FUNGOUS AFFECTIONS OF THE SKIN (p. 250); pityriasis rosea under SCALES (p. 601); lichen planus and herpes, in both of which macules appear as secondary lesions, respectively under PAPULES (p. 487) and VESICLES (p. 753). Of nævi and of telangiectases the identification is self-evident, and it only remains to speak of the macules of leprosy and syphilis.

In *leprosy*, following the prodromal symptoms and the period of invasion, erythematous spots appear on the face, limbs, or trunk, varying in colour according to the natural pigmentation of the skin, but usually in white races of a light red. The colour is brightest at the edge; the centre may become white and atrophic. In size the macules vary from a pin's head to the palm of the hand, or larger; they are smooth and shining, with a well-defined outline. Some infiltration is usually present. Fresh crops continue to come out at irregular intervals, and each outburst is accompanied by an exacerbation of the constitutional symptoms. After a time the macules and the neighbouring areas of apparently normal skin become more or less anæsthetic. The macular stage of leprosy may possibly be confused with *erythema simplex*, but the macular areas are usually larger than in erythema, in which also there is little or no constitutional disturbance. As soon as anæsthesia arises the diagnosis is settled. This is indeed the crucial test in all cases of doubt as between leprosy and any other affection, for in leprosy it is almost invariably present, if not in the lesions themselves, then in some neighbouring area of the skin. Its commonest sites are towards the centre of the macule, in the pale patches left by macules that have disappeared, and in the hands and feet. Another distinctive feature of leprous spots is that they rarely perspire. In *syringomyelia* the sensory and trophic lesions may suggest leprosy, but the macules will be absent, nor is there enlargement of lymphatic glands or thickening of nerve-trunks. (For the diagnosis of nodular leprosy from lupus vulgaris, see under NODULES, p. 402.) Whenever doubt exists, the lepra bacillus should be sought in the lesions or in the nasal discharge.

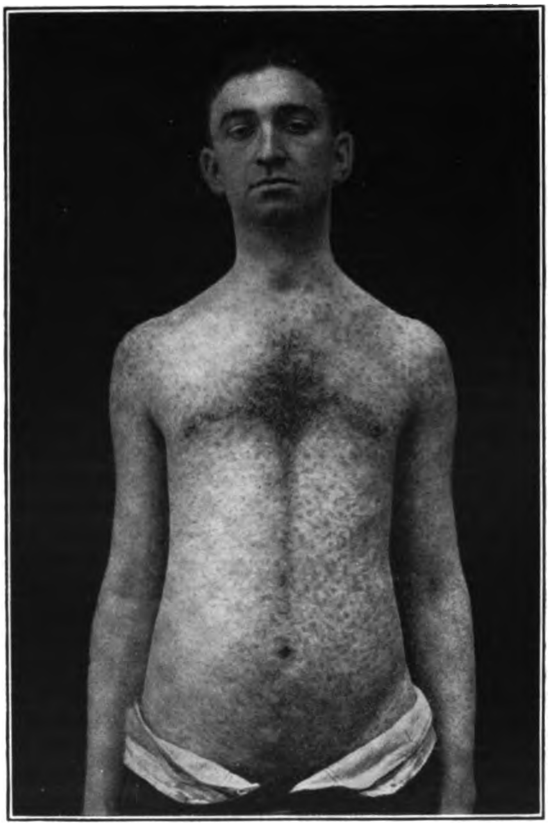


Fig. 170.—Macular syphilides.

The *macular syphilide* is one of the most characteristic lesions of secondary syphilis. The eruption (*Fig.* 170), erythematous in character and styled syphilitic roseola, begins as a macular mottling, resembling measles but rather more dusky, distributed over the chest and abdomen. It is extremely evanescent, often disappearing in a few hours and coming out again as suddenly. The mucous membrane of the throat is the seat of a similar eruption, and superficial ulcers may form on the tonsils. Generally about a fortnight from its appearance the rash begins to fade, giving place to a papular or follicular eruption on the trunk, limbs, face, and neck. Hyperæmia of the papillæ here and there gives rise on the chest and abdomen, and often on the flexor aspects of the limbs, to red patches which may persist for a longer or shorter time as isolated blotches, varying in colour from a delicate rose to a pale violet or dusky-bluish or even brownish-red. Scattered about among these macular syphilides may often be seen papules (maculo-papular syphilides), which leave stains of varying depth.

From the macular syphilide both *tinea versicolor* and *tinea circinata* may be distinguished by the fungous parasites present in the lesions of those affections, and, in the case of tinea versicolor, by the ease with which the scaly patches can be detached by the finger-nail; the erythematous *drug rashes*, such as those produced by copaiba, etc. (p. 222), by their more vivid redness and the presence of itching and burning; *seborrhœa corporis* by its more limited distribution; *measles* by the crescentic character of the eruption, the coryza, cough, and the different distribution. A peculiarity of this syphilide, which should always be watched for in doubtful cases, is that it varies in colour with the temperature; a cool atmosphere will bring it out in vivid colours, even when almost completely faded.

Malcolm Morris.

MAIN-EN-GRIFFE.—(See Claw-Hand, p. 109.)

MARASMUS literally means 'wasting,' and therefore signifies much the same as loss of weight. By common consent, however, when speaking simply of marasmus, one generally has in mind an infant or young child, so that lesions which cause loss of weight in tender years will be considered under the present heading, whilst wasting in older patients is discussed under the heading Weight, Loss of, p. 768.

The bodies of infants and young children consist so largely of water that great variations may occur within a comparatively short time, particularly in association with a disease which causes loss of fluid. The most rapid loss of weight occurs as the result of *acute diarrhœa*, with or without vomiting; in the summer zymotic diarrhœa of infants the subcutaneous tissues may be seen to shrivel in twenty-four hours or less, the eyes become sunken, the fontanelle depressed, and the patient loses weight rapidly. There are probably various micro-organisms producing these acute symptoms, of which the best known are the *Bacillus enteritidis* of Gaertner and Morgan's bacillus I., but the exact bacteriological diagnosis of the symptoms can only be arrived at by investigation of the stools and perhaps of the patient's serum in special laboratories. Acute vomiting without diarrhœa generally causes loss of weight, but less markedly than does severe diarrhœa; it sometimes does so to a considerable extent, nevertheless, particularly in that periodic type of the malady known as *cyclical vomiting of infants.* Without apparent cause, a child of tender years who is subject to this complaint is seized, without any preceding irregularity in diet and apparently without anything definite to account for the mischief, with most severe and recurrent vomiting, lasting for twenty-four, thirty-six, or forty-eight hours, or even longer, nothing whatever being kept down, and the urine at the same time abounding as a rule with diacetic acid and acetone, the evidence of acidosis (p. 3). Severe though the loss of weight may temporarily be, the symptoms generally subside as rapidly as they come on, and the patient remains in apparently normal health until the next period of similar vomiting with acidosis comes on. Besides cyclical vomiting, severe attacks of vomiting may be caused by errors of diet of various kinds, though it is remarkable how children escape the disorders of injudicious feeding if only virulent organisms are not administered in the food at the same time. *Congenital hypertrophic stenosis of the pylorus* is nowadays spoken of as though it were itself a disease; it is associated with persistent vomiting of all foodstuffs, the symptoms coming on either immediately after birth or within a few days or weeks, and in not a few instances resulting in death from sheer inanition. At the post-mortem examination in such cases there is undoubtedly both more muscle than there should be in the pylorus, and undue tightness of its constriction, but it is very doubtful whether this is really a condition of congenital malformation, and not the result of spasm of the pylorus produced by injudicious feeding, especially the giving of food before the mother has milk in her breasts; the pyloric hypertrophy being, not congenital, but the result of the muscular contractions so induced.

Simple *starvation* owing to inability of the parents to provide food will naturally cause acute wasting, though the nature of the case may not be obvious to the doctor unless the conditions of home life are known.

Defective feeding is one of the commonest causes of lack of progress and of actual marasmus amongst the children of the lower classes. The amount of dirt that reaches the child's mouth from its own fingers, from its mother's breasts, and from the utensils in which the food is given, is by itself enough very often to upset the digestion, even if the right food were given in the proper amounts and at the right intervals ; when, in addition to the dirt, the food supply is of the wrong kind and the intervals are irregular, it is not at all surprising that the child does not thrive.

Rickets is not so much a cause of marasmus as a concomitant effect of the injudicious feeding—many rickety children being, indeed, unduly fat and heavy.

Congenital syphilis, on the other hand, is a very potent cause for marasmus. The diagnosis may sometimes be guessed at ; it may sometimes be obvious from the snuffles, skin lesions, Parrot's nodes, condylomata, and so forth ; it may be known of in the parents ; and it may be confirmed by Wassermann's serum test. Many congenital syphilitic children, without developing any of the better known evidences of syphilis, fail from simple inability to thrive, and, although born fine, healthy-looking infants, presently waste and pine, and they may be said rather to cease to live than in the ordinary sense to die of a disease.

Tuberculosis is a very important and common cause for loss of weight in infants, though it is generally very difficult indeed to be certain that a tuberculous lesion is present. No obvious foci such as tuberculous glands in the neck, kyphosis from spinal caries, ascites, or abdominal lumps from tuberculous peritonitis or tabes mesenterica, tuberculous joints such as the hip or knee, tuberculous dactylitis, and so forth, may be present, and yet there may be some deep-seated lesion, of which the commonest by far is caseation of the bronchial glands. Phthisis pulmonalis is almost unknown in infancy and childhood; in phthisical patients there is practically never caseation of the bronchial glands ; in infants and young children caseous bronchial glands are very common, and they occur almost entirely in those who have drunk any large quantity of milk. The danger in such cases is that the bacilli will not remain localized to the bronchial glands : many a child is quite unsuspected of having such a lesion until some intercurrent malady such as measles causes the mischief to light up and become generalized in the form of general tuberculosis and meningitis, and at the post-mortem examination caseous bronchial glands, obviously of long standing, are found. Many children recover completely, and the nature of the case at the time when there were loss of weight and general ill-health may never be determined. The child outgrows its delicacy. It is always possible, or even probable, when obscure wasting occurs in a child who objectively presents no particular abnormality except ill-health, that the lesion is tuberculous absorption from infected milk, with accumulation of the bacilli in the mesenteric and bronchial glands. The fæces may be examined for the bacilli after antiformin treatment in the laboratory. It may often help the diagnosis to eliminate milk from the dietary, and, if need be, butter also, and watch the effects of giving the patient such foods as are known to contain no tubercle bacilli. Von Pirquet's skin reaction might also be tried, though its results are by no means pathognomonic (p. 768), especially if the tuberculin used is not prepared exactly as it was in von Pirquet's original method.

Herbert French.

MELÆNA.—This term is correctly applied to black motions containing altered blood. The colour is due to the action of the digestive juices upon hæmoglobin, and the condition is usually associated with some ulcerative lesion of the stomach or duodenum. It may be simulated by the presence of sulphide of iron or of bismuth in the stools of patients taking either of these metals. The slatey-black colour of these sulphides does not very closely resemble the tarry motions of hæmorrhage. The motions may also be black after consumption of charcoal, in the form of biscuits or otherwise, or of bilberries. In case of doubt, the microscope may be used to reveal blood-corpuscles, or the chemical tests for blood may be employed. (See BLOOD PER ANUM, p. 75.) *W. Cecil Bosanquet.*

MELANURIA.—(See URINE, ABNORMAL COLORATION OF, p. 745.)

MEMORY, LOSS OF.—(See AMNESIA, p. 19.)

MENORRHAGIA.—By this symptom is meant an excessive amount of the menstrual flow, or an undue prolongation of the time during which it takes place. It is important to remember that in this condition the patient is free from bleeding during the intermenstrual periods, the term METRORRHAGIA (p. 390) being reserved for bleeding which occurs between the periods. The careful distinction between these symptoms often serves to distinguish very important conditions, and they should on no account be confounded with one another or considered as the same entity. Pure menorrhagia is an important symptom of many well-defined conditions which do not, as a rule, give rise to irregular bleeding. Both these terms must be limited carefully to patients who menstruate, and must not be used for bleeding after the menopause. The term METROSTAXIS (p. 392) is the best for bleeding occurring after menstrual life has passed.

CAUSES OF MENORRHAGIA.

1. GENERATIVE SYSTEM

Uterine congestion
Endometritis
Retroversion and flexion
Fibromyoma
Salpingo-oöphoritis
Sub-involution

Acute Infectious Diseases
Influenza
Enteric
Cholera
Scarlatina
Variola
Rheumatism
Malaria
Diphtheria
Measles

2. CIRCULATORY SYSTEM

Uncompensated valvular disease of the heart
Cirrhosis of the liver
Emphysema of the lungs

The Circulation
Passive hyperæmia from :
Constipation
Tight-lacing
Sewing machine

The Blood Itself
Deficient coagulability
Scorbutus
Purpura
Hæmophilia

High Blood-pressure
Arteriosclerosis
Disturbance of internal secretions

3. NERVOUS SYSTEM

Excessive coitus
Prevention of conception

A Single Excessive Period
Fright
Violent emotion
Sudden changes of temperature
Cold bath
Dancing
Gymnastics
Bicycling, etc.

Perusal of the above table will make it clear that the causes of pure menorrhagia can be grouped under the three headings of diseases of the generative organs, circulatory organs, and the nervous system. In attempting to differentiate these causes from one another, the first point to ascertain is whether there is any disease of the generative system, and failing this, to make such systematic examinations as will place the cause under one of the other two headings.

In considering the **Generative System,** it is clear that some diseases will be easy to discover, others will require some special method of examination.

For instance, of all the causes of pure menorrhagia, *fibromyoma* of the uterus stands out by itself as the only important growth associated with this symptom, and a simple bimanual examination, as a rule, suffices to show that such a tumour exists, the chief characteristics of a fibromyoma of the uterus being these : the uterus itself is enlarged, and in almost every instance the enlargement is asymmetrical, the typical shape of the organ being altered ; as there may be more than one tumour in the uterus, its shape may be exceedingly irregular ; the consistence of the tumour is hard and unyielding as a rule, but pathological changes in these tumours are common, some of them leading to softening, others to cystic changes which may give a fluid thrill. The tumour and cervix always move together if the organ can be moved at all. The only difficulty in diagnosis, as a rule, lies in distinguishing a fibromyoma of the uterus from an ovarian cyst, and sometimes this is exceedingly difficult, for it is not always possible to say that a given tumour is actually the enlarged uterus. It must be remembered, however, that the symptom which has led to this difficulty is menorrhagia, and ovarian tumours almost never give rise to it. Ovarian tumours usually cause no disturbance of menstruation at all, unless they are double and destroy completely both ovaries, in which case they cause amenorrhœa. If the tumour cannot be diagnosed by simple examination, there still remains examination by the uterine sound. If no possibility of pregnancy exists—and with pure menorrhagia pregnancy *is* impossible—the sound may be passed into the uterus with every precaution against sepsis.

In all cases of fibromyoma, the sound passes beyond the normal 2½ inches, and it may pass as much as 6 inches, or even more. In cases of subperitoneal fibroids, the uterus may not be much enlarged, but in such cases menorrhagia is not usually present. In ovarian tumours the length of the uterine cavity is not increased unless a condition of endometritis co-exists, which is very uncommon ; and if it did not exist, the amount of elongation of the uterine cavity would be small. In general, however, it is quite unnecessary to use the sound for the diagnosis of a fibromyoma.

Uterine Congestion and Endometritis.—These lesions can only be inferred in cases of pure menorrhagia when the uterus is not enlarged to any appreciable extent, and when, in addition, there are leucorrhœa and backache. These three cardinal symptoms, Pozzi's syndrome, point always to endometritis, whatever other lesion of the generative system may be present. As a rule the subjects of these are married and have had pregnancies or abortions, but endometritis may occur in a virgin, the result of infection, without any pregnancy having taken place. The presence of endometritis cannot be proved without the removal of the endometrium and microscopical examination of sections of the material removed by curettage.

Retroversion and flexion of the uterus and *salpingo-oöphoritis* are very definite and obvious lesions which are associated with menorrhagia, but the actual prime cause is again endometritis and uterine congestion.

So also with *sub-involution*, which necessarily can only follow labour or abortion ; though a relaxed uterine muscle and a dilated uterine cavity are present, endometritis and congestion are present, too, and are the real causes of menorrhagia.

Exanthemata.—The various exanthems are liable to cause menorrhagia except in those instances where they give rise to anæmia. It has been shown bacteriologically that an acute endometritis may be set up by various zymotic diseases, and therefore it is not surprising that in some instances this condition becomes chronic and causes a lasting menorrhagia.

Circulatory System.—Under this heading there can be no doubt that definite causes of menorrhagia exist, but in the absence of well-defined lesions of heart, liver, or lungs it may be a matter of considerable difficulty to make a differential diagnosis. Any lesion of the heart, liver, or lungs which leads to back-pressure in the venous system may cause hyperæmia of the pelvic organs and consequent excessive menstrual losses. It does not follow, however, that this will be the case, because the sufferers from these diseases are sometimes anæmic as far as the *quality* of the blood goes, and consequently may lack the stimulus to menstruate at all. However, it happens not uncommonly that menorrhagia is caused by uncompensated valvular lesions of the heart, cirrhosis of the liver, or emphysema of the lungs. *Passive hyperæmia* of the pelvic organs may result from constipation, tight lacing, or certain occupations such as the working of a treadle sewing-machine : but endometritis may also be present and be the real underlying cause of excessive flow.

Anæmia.—That the quality of the blood itself may be a cause of menorrhagia is undoubted, and particularly if it be deficient in calcium salts, leading to retardation of the coagulation-time. Modern methods of estimating coagulation-time enable us to distinguish these cases with some certainty, and thus point out a line of treatment. Unfortunately there is no simple clinical method. Doubt also has recently been thrown on the view that the calcium salts have any effect on coagulation-time. The well-known signs of *scorbutus* in its minor degrees, *purpura*, and *hæmophilia* may draw attention to cases of this class.

Menorrhagia in young girls at the time of puberty and commencement of menstruation depends upon *excessive ovarian activity*, and we must conclude, therefore, a disturbance of the balance between the internal secretions. It is often associated, too, with retardation of the coagulation-time of the blood, especially in the subjects of chilblains, cold hands and feet, 'dead fingers,' etc. It must not be forgotten that young girls may have a malignant growth of the uterus, such as sarcoma, but this is more likely to cause irregular bleeding as well as menorrhagia.

Finally, *high blood-pressure* must be reckoned with as a cause of menorrhagia at any period of life, but particularly when nearing the onset of the menopause. Menopause menorrhagia much more often depends upon one of the well-defined lesions of the uterus described above than on high blood-pressure, but cases occur in which the blood-pressure is alone responsible. The arteriosclerosis which is likely to affect the uterine vessels about this period of life may contribute to the causation of menorrhagia. The high blood-pressure, and possibly the arteriosclerosis also, may eventually prove to be connected with the internal secretions of the ductless glands. Though still mainly a matter of theory, normal menstruation depends in part at least on the normal balance being preserved between the various internal secretions, the ovarian and thyroid on the one hand being balanced by the suprarenal and pituitary on the other, and any disturbance of this balance may result in amenorrhœa (as in myxœdema), or in menorrhagia, as sometimes occurs in *exophthalmic goitre* and at the menopause. It is very fascinating to believe that high blood-pressure may be due to the unbalanced action of the suprarenal and pituitary secretions, and to suggest a remedy in consequence. In the absence, however, of collateral signs of definite lesions of ductless glands, we have at present no ready means of telling which gland is at fault.

The Nervous System alone is never likely to be a cause of lasting menorrhagia, but that a single profuse period may result from such disturbance of the nerve mechanism of menstruation has long been believed. There certainly are cases in which no other causation can be recognized, and in which the excessive flow is not repeated. The effect of sexual intercourse upon the menstrual flow is difficult to determine, but cases do occur in which excessive menstruation has been cured by abstention, and we cannot but believe that excesses in this direction must therefore have been the cause. Such cases occur chiefly in the newly married. The part played by incomplete coitus, coitus interruptus, or prevention of conception by other means, is still difficult to determine, but we have no *real* evidence to hand which proves that any menstrual disturbances arise on these accounts. In any case, however, we are not justified in assuming that the nervous system is to blame for a menorrhagia until, by careful examination, we have eliminated the other more important causes. *T. G. Stevens.*

MENSTRUATION, ABNORMALITIES OF.—(See AMENORRHŒA, p. 17 ; MENORRHAGIA, p. 385 ; and METRORRHAGIA, p. 390.)

MERYCISM is equivalent to cud-chewing or rumination; it is very rare in man ; even when it does occur it is no evidence of disease. It has to be distinguished from pyrosis and from flatulence ; in typical cases there is no difficulty, for with merycism the act may be voluntary to some extent : actual food returns to the mouth instead of merely acrid fluid as in pyrosis, and there is none of the belching of flatulence. It sometimes develops in several members of the same family ; this may be a question of imitation, but it is due quite as likely to congenital peculiarity. The diagnosis depends mainly upon the patient's own account of what he feels taking place inside him, upon the history of a similar condition affecting other members of the family, and upon the absence of objective evidence of gastric, intestinal, intracranial, or renal disease. *Herbert French.*

METEORISM, or tympanites, is the term used to denote enormous distention of the abdomen with gas, the latter generally being within the alimentary canal, though it may be free in the peritoneal cavity. It is seldom a symptom in itself of diagnostic importance, the nature of the case being determined usually on other grounds. It is apt to be very troublesome in cases of *general peritonitis* ; the diagnosis will depend upon the history, which may suggest a cause for peritonitis, such as gastric or duodenal ulcer, appendicitis, typhoid fever ; and upon the persistent vomiting, the dry furred tongue, the motionless rigid abdomen, the rising rapidity of pulse, the facies Hippocratica, the impairment of note in the flanks, the rub over the liver or spleen, and the absence of borborygmi.

Intestinal obstruction, whether acute, subacute, or chronic, and whether due to strangulated hernia, peritoneal band, volvulus, new growth, intussusception, or other cause, often leads to extreme meteorism, with visible peristalsis, the passage of neither fæces nor flatus, and persistent vomiting which will become fæculent if the case is not operated upon. Peritonitis ultimately supervenes ; but previous to this, intestinal obstruction is differentiated from general peritonitis by the absence of rigidity of the abdominal wall, by the presence of borborygmi and visible peristalsis, the absolute constipation in spite of enemata, the slower pulse, and the relatively better condition of the patient.

Acute pancreatitis, whether hæmorrhagic or not, may cause acute meteorism. The symptoms are variable, but they nearly always suggest an acute abdominal condition requiring immediate laparotomy, the diagnosis being then suggested directly the areas of fat necrosis are seen in the omental fat. Previous to laparotomy, the symptoms are rather those of acute intestinal obstruction than of general peritonitis ; the usual history of acute pain in the epigastrium may at first suggest perforated gastric ulcer, but the abdomen remains supple as in obstruction more often than it becomes rigid as in peritonitis.

Meteorism in cases of *typhoid fever*, *dysentery*, *dengue*, and other severe illnesses in which the bowel is affected, is chiefly of importance in that it may lead to a suspicion of perforation and general peritonitis. The diagnosis is often very difficult, and there may be grave anxiety and doubt as to whether the abdomen should be opened or not. One important point in typhoid fever is that perforation is generally accompanied by a sudden drop in the temperature and an equally sudden rise in the pulse-rate, whereas meteorism by itself would not cause this.

When the vessels in the mesentery are affected by *thrombosis* or *embolism*, acute meteorism results, with all the signs of intestinal obstruction, rapidly followed by peritonitis. The nature of the case may be quite obscure until laparotomy is performed, unless the existence of a cause is known, such as fungating endocarditis.

Interference with the *solar* and *mesenteric plexuses of nerves* has sometimes led to severe meteorism in cases of *tabes mesenterica*, or infiltrating intra-abdominal *new growth*. The symptom occurs late, and the diagnosis will generally have been made on other grounds.

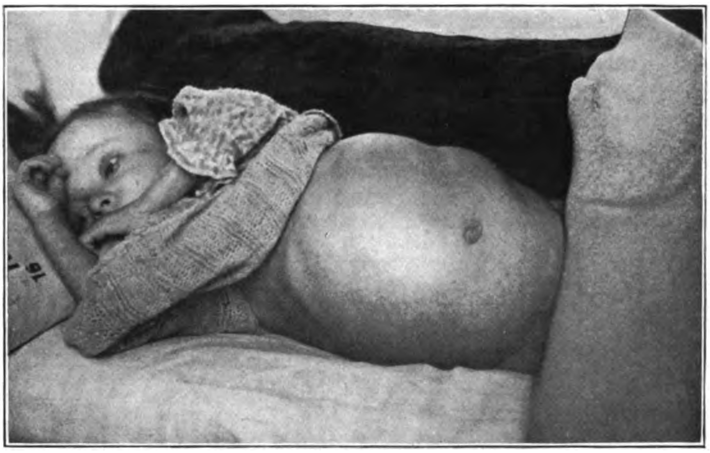


Fig. 171.—Hirschsprung's disease; or idiopathic dilatation of the colon. Note the distended coils of intestine. (*From Professor Rutherford Morison's Introduction to Surgery.*)

Affections of the *spinal cord* may lead to paralysis of the bowel and tympanites. This may result from transverse ' myelitis,' whether due to primary softening of the cord from syphilitic or other spinal arterial thrombosis, from compression by spinal caries, new growth, aneurysm, or from destruction of the dorsal region of the cord by a stab, a crushing, or a bullet wound. There will generally be Paraplegia (p. 510) to indicate the nature of the case.

Diabetes mellitus often indicates its impending termination in coma by the onset of abdominal pains, with more or less meteorism. The diagnosis will be known already on account of the glycosuria. Meteorism is also common in the late stages of *cirrhosis of the liver*.

Particular mention may be made of *Hirschsprung's disease*—idiopathic enormous distention of the sigmoid colon in children and young people (*Fig.* 171). Careful examination indicates that the enormous gaseous distention of the abdomen is not due to general tympanites, but to ballooning of what may seem at first to be stomach, but which is proved not to be this by the absence of immediate effect on the gas-containing cavity when fluid or gas is given by the mouth, by the swelling appearing to arise from the left iliac fossa, and if need be by the x-ray shadows after a bismuth meal. Obstinate constipation, or symptoms of recurrent intestinal obstruction, are usual in these cases, and the diagnosis is confirmed by the laparotomy that is generally required, in the end, to relieve the patient.

Hysteria, or rather functional derangement of the nervous system, can lead to almost any symptom (p. 465), including meteorism. Two difficulties arise in the diagnosis: namely, to be sure: (1) That the condition is meteorism at all, and not pregnancy, ascites, ovarian cyst or other tumour—phantom tumours are difficult to diagnose without examination under an anæsthetic, and even laparotomy may be undertaken before the absence of a tumour is established certainly; and (2) That the meteorism has no organic basis—the circumstances may sometimes suggest this at once, but in some cases the exclusion of an organic cause for the tympanites may take much time, careful enquiry into symptoms and physical signs, and considerable anxiety meanwhile. *Herbert French.*

METHÆMOGLOBINURIA.—(See HÆMOGLOBINURIA, p. 284.)

METRORRHAGIA means loss of blood from the uterus in the intermenstrual periods, and the term should be applied only to irregular hæmorrhages during menstrual life. It is not correct to apply it to hæmorrhages connected with pregnancy, for menstruation is then in abeyance. It may, however, be used with propriety in those cases remotely connected with pregnancy in which menstruation has been re-established. The term may be used for losses of actual blood, or for blood-stained discharges in which mucus is mixed with the blood.

CAUSES OF METRORRHAGIA.

1. GENERATIVE SYSTEM	2. CIRCULATORY SYSTEM	3. NERVOUS SYSTEM
Malignant Growths: Carcinoma Squamous epithelioma Sarcoma Chorion-epithelioma	*High blood-pressure due to:* Internal secretions Arteriosclerosis	Sexual excess
Benign Growths: Submucous fibroid Fibroid polypus Mucous polypus	*At the menopause:* Undue congestion due to: Internal secretions Deficiency of calcium at the onset	
Inflammatory Lesions: Erosion of cervix Endometritis Tuberculosis of the uterus	*Blood Changes:* Purpura Scorbutus Hæmophilia Leukæmia	

The **Lesions of the Generative Organs** which give rise to metrorrhagia are well defined as a rule, and in the case of growths of the cervix uteri are often self-evident. Where growths of the body of the uterus are present, differential diagnosis is often a matter of great difficulty, and in many instances cannot be made without a preliminary curettage and microscopical examination of the material removed. In fact, with the exception of obvious mucous polypi, fibroid polypi, and advanced growths of the cervix, all the growths of the uterus require a preliminary histological examination for their exact diagnosis unless the symptoms demand a radical operation. In such cases it is sufficient to diagnose the actual nature of the growth after removal. It is not out of place here to suggest the best way to make histological preparations from curetted material, a matter of great importance to the patient, because it is often difficult to distinguish between cancer and endometritis unless the very best microscope sections can be secured. The curetted material must be obtained after dilatation, with a sharp curette, and the larger the fragments removed the more easy will the histologist's work be. Anæsthesia is always essential except in the case of cervical growths. In doubtful cervical growths a wedge should be cut out, including some normal tissue if possible. Curetted fragments should be washed free from blood for a minute or two, but should not be left to soak in water. They should then be placed immediately in an efficient fixing fluid, and the best all-round fluid for this purpose is formalin 10 c.c., 0·75 per cent salt solution 90 c.c. Twenty-four hours in this fluid lead to good fixation, after which the tissues can be dehydrated in successive strengths of alcohol, cleared in xylol, and finally embedded and infiltrated with paraffin wax. Sections cut from these paraffin blocks are the best obtainable, far superior to any freezing method or celloidin infiltration. If the stained sections are submitted to a histologist who has experience of uterine growths, there should not be two per cent of doubtful specimens. If, however, the tissues are fixed improperly, thick sections are cut, and stained badly, then the most skilled histologist will be unable to give a definite and reliable diagnosis.

Cancer of the body of the uterus, cancer of the cervical canal, early cancer of the cervix, sarcoma of the uterus, chorion-epithelioma, some sloughing fibroids, tubercle, and endometritis can be distinguished from one another only by investigations carried out on these lines. The fact that all these lesions produce metrorrhagia, and may give rise to hæmorrhage on coitus, walking, straining at stool, and bimanual manipulation of the uterus, makes it imperative that we should have histological confirmation of the nature of the lesion before making an exact diagnosis.

The relation of *fibromyoma* to metrorrhagia as opposed to pure menorrhagia, which is the rule with these tumours, is interesting. Fibroids only produce irregular bleeding when they are submucous and in process of extrusion, when they are infected and sloughing, or when they are actually polypoid. The reason for this is that in these conditions the tumours are always partly strangulated by uterine contractions, and therefore in a state of gross venous congestion ; hence they bleed more or less constantly, without provocation. The occurrence of irregular bleeding in a person who is known to have fibroids almost always means one of these conditions, and, commonly, extrusion of the tumour from the uterus. On the other hand, it must not be overlooked that carcinoma may develop in the endometrium with a fibroid also present, or that a fibroid may become sarcomatous, or that a sarcoma may arise *de novo* in the uterus and attack a pre-existing fibroid. Rapid enlargement of a uterus, with irregular hæmorrhage, is very suspicious of a *sarcoma*, but as it is not uncommon for several fibroids to be present in the same uterus, it is also common for rapid enlargement to occur as a result of cystic changes in one of them, whilst hæmorrhage may take place due to extrusion of another.

Pure *carcinoma of the body of the uterus* rarely produces much enlargement of the organ, and any increase in size is not very rapid.

Chorion-epithelioma follows hydatidiform mole in about 50 per cent of the recorded cases, and it always follows pregnancy, never having been seen in the uterus in a case where pregnancy could be excluded. It is associated especially with profuse bleeding and the rapid development of a fœtid discharge due to decomposition of blood and necrosing tissues in utero. Carcinoma of the body of the uterus rarely produces foul discharges until the condition is very advanced and has become exposed to the air.

The differential diagnosis of bleeding due to *cancer*, *erosion*, and *tubercle of the cervix* is often difficult in the early stages. In advanced cancer the friable hardness of the growth distinguishes it at once from the tough leathery hardness present in erosions. In the former, the growth can be broken down with the finger ; in the latter, the soft velvety erosion can be scraped off the tough leathery and fibrous cervix beneath. Nothing, however, but sections made from wedges removed from the cervix enable us to distinguish cancer or erosion from tubercle in the early stages. Tubercle of the cervix is usually mistaken for cancer, but the difference is clear enough in microscope sections.

Mucous polypi and *fibroid polypi* are common causes of intermenstrual bleeding, and are usually quite definite growths. The mucous polypus is soft, strawberry-red in colour, often pedunculated, and contains cystic spaces filled with glairy mucus. It almost never gives rise to a malignant growth. The fibroid polypus is hard, and shows the glistening whorled appearance so well known in fibromyomata on section. These growths are liable to infection and sloughing, and are then apt to be mistaken for cancer or sarcoma. The microscope alone will enable the difference to be made out.

Endometritis rarely causes severe metrorrhagia, but is often associated with a blood-stained watery discharge. In a doubtful case there is absolutely no way of distinguishing it except with the microscope.

The Circulatory System is sometimes responsible for metrorrhagia, just as it is for MENORRHAGIA (p. 385), and the actual causes are much the same. It is, however, especially at the onset and the decline of menstruation that irregular bleeding is likely to occur from this cause. The same disturbance of the internal secretions which may cause menorrhagia at these periods sometimes acts similarly in causing irregular bleeding. It is fairly common to find young girls at the onset of menstruation having menorrhagia and metrorrhagia, and it is often very difficult to be certain of a cause. It depends, however, very largely on two definite factors, namely : (1) Unusual uterine congestion, the result of an excess of the biochemical stimulus (internal secretions) of menstruation, and (2) deficient coagulation power, possibly due to a want of calcium in the blood. The former cannot be diagnosed by any defined investigation, but the latter is determined by estimating the coagulation-time. Purpura, scorbutus, and hæmophilia are diagnosed readily when they act as causal agents. Leukæmia is sometimes responsible for irregular uterine bleeding, and is diagnosed readily by making a total and differential leucocyte count (p. 24).

The Nervous System seldom causes metrorrhagia, but there is no doubt that sexual excess, often seen in the first months of married life, is a reflex cause of uterine congestion, and may cause metrorrhagia as well as menorrhagia. *T. G. Stevens.*

METROSTAXIS is the term applied to uterine hæmorrhage at any period of life, unconnected with menstruation, or at times when menstruation is in abeyance. It is convenient to keep this form of hæmorrhage separate from the other varieties, because in this way all the pregnancy hæmorrhages can be differentiated carefully. Its cause may be classified according to whether the uterus is pregnant or not. The bleeding which occurs from the vagina occasionally in new-born infants is usually thought to depend upon uterine congestion subsequent to the cessation of the placental circulation. It is usually trivial, but a fatal case has been reported.

CAUSES OF METROSTAXIS.

UTERUS NON-PREGNANT	UTERUS PREGNANT
Uterine bleeding in the new-born Malignant growths Polypi Senile endometritis Senile granular vaginitis Pyometra Secondary post-partum hæmorrhage Sub-involution	Threatened abortion Ante-partum hæmorrhage Extra-uterine gestation Malignant growths of cervix or vagina Erosions Polypi.

The differentiation of *malignant growths*, *polypi*, and *senile endometritis* can only be established in the same manner as in cases occurring during menstrual life (p. 391). *Senile adhesive vaginitis* must not be overlooked as a possible cause ; the vaginal walls at the fornices become inflamed and form granulation tissue which may bleed if the surfaces rub together. On examining such cases the surfaces may be partly adherent, and the separation brought about by the finger may cause bleeding. In any doubtful case, the routine dilatation and curettage of the uterus must never be omitted. An unsuspected *pyometra*, or distention of the uterus with pus, may cause hæmorrhage, along with a foul discharge, and although it is almost always accompanied by a malignant growth, may be only the result of infection and granulation-tissue formation.

In relation to a recent pregnancy, hæmorrhage may result from simple sub-involution, from retained products of conception, and from chorion-epithelioma. The differentiation of these conditions can be established only by exploration of the uterine cavity, with, if necessary, the assistance of the microscope. Such conditions may be termed secondary post-partum hæmorrhage in cases occurring within a few days of delivery.

Hæmorrhage from the pregnant uterus almost always means separation of the placenta or of the embryo from its attachments, but malignant growths of the cervix, erosions, and polypi may have to be considered. Hæmorrhage from a pregnant uterus is never due to malignant growths of the body of the organ, because pregnancy is practically impossible with such lesions. There are, however, two great difficulties in connection with pregnancy hæmorrhages: these are to differentiate (1) the uterine hæmorrhage which occurs along with *extra-uterine gestation* from that due to *threatened abortion* ; and

(2) the bleeding of *placenta prævia* from that due to the *separation of a normally situated placenta.*

In the first case, arising very early in pregnancy, the hæmorrhage occurs when the extra-uterine gestation is separated from its tubal or other attachments and is converted into a tubal mole, when it becomes extruded from the fimbriated extremity of the tube, or when the tube ruptures. Therefore, there may be history of acute abdominal pain, faintness, and possibly collapse from internal hæmorrhage. Along with this, the uterus will not be found obviously enlarged, whilst there is some sort of swelling in one or the other posterior quarter of the pelvis. Hæmorrhage due to threatened abortion cannot be diagnosed unless the presence of an intra-uterine pregnancy can be established. Therefore, in this case we must look for the definite signs of a normal pregnancy, which in the early months will be: amenorrhœa, morning sickness, breast changes, enlargement of the uterus, Hegar's sign, and Braun's sign. The former consists in the extreme softening of the upper part of the cervix and lower part of the uterine body, combined with the as yet unsoftened vaginal portion and globular tense fundus; it is found from the sixth to the eighth week. The latter consists in the irregular shape of the uterus from the eighth to the twelfth week. One side is larger than the other, and an ill-defined groove is found between them.

In the second case, occurring generally after the sixth month of pregnancy, it is of the greatest importance to be able to diagnose *placenta prævia.* The only definite sign is the feeling of the placenta through the cervix when it will admit of this method of investigation. The suggestive signs are those due to the filling up of the lower uterine segment by the placenta. The presenting part remains high up and movable, not engaged in the brim, and there is a sensation of great increase of thickness between the vaginal fornices and the presenting part. In any case of severe hæmorrhage, however, the cervix must be dilated so as to admit a finger, as treatment depends upon diagnosis, and no patient with a placenta prævia is safe until she is delivered and bleeding has ceased.

T. G. Stevens.

MICROPSIA.—(See VISION, DEFECTS OF, p. 763.)

MICTURITION, ABNORMALITIES OF.—A person in health micturates about five times during the twenty-four hours, the total amount of urine passed being about 1500 c.c., or 50 ounces. This varies according to the amount of fluid taken, the amount lost by perspiration, and so forth. The act of micturition is controlled by a nervous mechanism, a stimulus from the vesical mucous membrane starting an impulse which causes contraction of the detrusor muscle, and at the same time relaxation of the sphincter at the urethral orifice. The special centres controlling the motor functions of the bladder are in the spinal cord at the level of the third sacral nerve, whilst the brain controls these centres in response to sensory impulses received. The abnormalities of micturition which are met with in practice depend partly upon lesions of some portion of the urinary apparatus, and partly upon some change in the nervous mechanism controlling the act, and will be discussed from these points of view, and under ENURESIS (p. 218).

1. **Increased Frequency of Micturition.**—A large number of diseases of the genito-urinary tract are accompanied by increased frequency of micturition, and it is necessary to ascertain if the increased frequency of micturition depends upon an increased amount of urine to be passed. Thus in *diabetes* or *chronic interstitial nephritis*, the increased amount of urine will cause an increased frequency of desire to micturate, provided the capacity of the bladder is unaltered. If the total amount of urine remains normal, any increased frequency of micturition may be due to some lesion of the genito-urinary apparatus, and consideration of the other symptoms of a case will often point to a definite diagnosis. It must be remembered, however, that increased frequency does not necessarily imply that the bladder is the seat of the disease, as the symptom is present with any form of renal pyelitis—commonly calculous or tuberculous—or with prostatic enlargement.

It is important to ascertain the relationship between micturition during the day and during the night. Normally, a healthy person should not wake during the night to pass urine, unless an excess of fluid has been taken; but if any inflammatory condition is present in the bladder, micturition will be present during the night, as well as increased in frequency during the day. Any form of *cystitis* or acute inflammatory conditions of the prostate or neighbouring organs, will cause increased frequency both day and night. In patients with chronic nephritis who are passing normal quantities of urine during the day, frequent micturition at night is common.

With *vesical calculus* there is increased frequency during the day, but often no urination is necessary during the night. The frequency during the day is increased with activity or exercise, or by the jolting movements of travelling, but is absent during a period of rest. If the presence of a calculus has excited cystitis, increased frequency of micturition will be present both day and night.

With *prostatic enlargement*, whether simple or carcinomatous, the increased frequency is most marked at night, and is commonly the first symptom of the disease noticed by the patient, generally a man of about sixty. The bladder is not emptied completely, so that the addition of a relatively small amount of urine from the kidneys soon fills up the incompletely emptied viscus and sets up afresh the desire to micturate.

In *vesical carcinoma*, increased frequency of micturition is present during both the day and night, as the infiltration of the vesical wall prevents the bladder from being distended without pain, and it is frequently associated with cystitis.

In *renal colic* caused by *calculus* or *blood-clot*, or torsion of a *movable kidney*, there may be increased desire to micturate, and the symptom may be present in inflammatory diseases in the pelvis, such as *salpingitis*, *pyosalpinx*, or a low-placed *appendicitis*, or in the secondary infiltration of the bladder in *carcinoma* of the *uterus* or *rectum*.

Increased frequency of micturition may be produced by mechanical obstruction to the normal vesical distention by a tumour occupying the pelvis, and is seen commonly with *ovarian cyst*, *uterine fibroid*, or a *retroverted gravid uterus*; these tumours will be found upon vaginal examination.

In children, increased frequency of micturition may be due to *phimosis*, *balanitis*, a *small urinary meatus*, *worms*, *penile calculus* (p. 469), *oxaluria* (p. 423), *coli bacilluria* (p. 69), or to *hyperacidity* of the urine.

2. **Changes in the Stream of Urine.**—An abnormality of the stream of urine may be due to a congenital deficiency of the terminal urethra, as in *hypospadias* or *epispadias*, or to some lesion mechanically obstructing the stream. Most commonly this is due to a *stricture* of the urethra. If the stricture be situated in the penile portion, the stream of urine is of small calibre but of fair force, whilst if the stricture is in the bulbous urethra, the mechanical effect upon the stream of urine passing through the stricture into the urethra of wider calibre beyond the stricture is that the force is diminished, whilst the actual stream as it leaves the meatus is not thinned. A stricture at or near the urethral meatus forms a thin but forcible stream; but no reliance can be placed upon the complaint of a 'twisted stream.'

The obstruction to micturition by an *enlarged prostate* causes the stream of urine to be slow and forceless, so that it may fall vertically from the meatus instead of in the usual arched manner. This same dribbling of urine will be seen when a urethral stricture becomes much narrowed, or again when the bladder musculature has lost its contractile power, or in disease of the nervous system affecting the motor paths to the bladder.

In any case presenting an abnormality in the stream of urine, careful inquiry should be made to ascertain if the stream has become gradually and progressively narrowed, as in stricture, or if the alteration in the force of the stream is accompanied by increased frequency of urination, as in prostatic hypertrophy in an elderly patient, or by urethral discharge in a case suggestive of acute prostatitis. A stricture may be diagnosed with certainty by careful endoscopic examination under air-distention, or, failing this, by the obstruction offered to the passage of a catheter or bougie. Prostatic enlargement or inflammation will be suggested by the history of the case, and confirmed by a digital examination of the gland per rectum; in the absence of a mechanical obstruction in the urethra, examination should be conducted for any disease of the spinal cord by testing the knee-jerk and other reflexes.

Sudden stoppage of the flow of urine during micturition may be caused by a small, movable *vesical calculus*, if the latter happens to engage in the internal urethral orifice or becomes impacted in the urethra. The same sudden cessation of the flow is caused occasionally by a tuft of a vesical *villous tumour* blocking the urethral opening during micturition. Usually the flow will be resumed after a few seconds, unless the calculus has passed into the urethra, when it may be passed naturally or require to be removed by surgical means. If the symptom recurs, a cystoscopic examination of the bladder will distinguish readily between the two conditions.

The same sudden cessation of the stream may occur without any intravesical lesion as the result of *spasmodic contraction of the vesical sphincter.* Patients subject to this trouble (so-called *stammering bladder*) can at times pass urine quite normally, but at others the stream is interrupted frequently, or they may be unable to pass urine at all, especially in the presence of a second person.

3. **Difficulty in Micturition.**—Frequently associated with some change in the character of the stream of urine, a patient may complain of difficulty in micturition, either as a hesitation in commencing the flow or a need to strain to maintain it. This, again, is most common with *urethral stricture* or *prostatic enlargement,* or may be due to the impaction of a *calculus* in the urethra or to the formation of *blood-clot* in the bladder. A calculus may be passed into the urethra and become arrested in the canal, but not so that it wholly obstructs the passage of urine. It is not uncommon for a calculus to occupy the dilated portion of the urethra behind a stricture, or occasionally a prostatic calculus projects from the gland into the lumen of the posterior urethra. A calculus so placed may increase in size by the further deposition of urinary salts whilst in the urethra, and cause difficulty in micturition; it may be felt in the canal from the outside, upon rectal examination, or upon passing a soft bougie into the urethra. Even if placed behind a stricture it may be felt by a fine guide or bougie passed to dilate the stricture. Difficulty in micturition may also arise from prostatic inflammation or from tuberculous disease of the prostate.

Difficulty in micturition due to the presence of blood-clot in the bladder will usually be indicated by the previous passage of blood-stained urine and by the constant efforts to micturate.

Difficulty in micturition in the *female* may be caused by a *pelvic tumour* by the drag or direct pressure on the urethra or vesical neck. This may occur with a *uterine fibroid* or a *pregnant retroverted uterus.* Occasionally, difficulty is produced by the direct infiltration of the urethra by a *carcinoma* of the vaginal wall or vulva.

Difficulty in micturition is not uncommon in *disease of the nervous system,* causing paralysis or paresis of the detrusor muscle of the bladder. This may be due to *trauma* and pressure on the spinal cord by blood-clot, or to *myelitis* or *tabes.* It must be remembered that it is not uncommon for the early cord-changes of tabes to affect the urinary organs, and that difficulty in passing urine may be complained of when the urethra and bladder are normal.

Atony of the bladder wall without any affection of the nervous mechanism, from recurring over-distention of the bladder, may cause difficulty in micturition.

4. **Retention of Urine**—by which is implied the gradual accumulation of urine in the bladder, with inability to pass any per urethram—may arise from *mechanical causes* obstructing the urethra, or from *derangement of the nervous system.* Retention of urine must be distinguished from ANURIA (p. 39), or failure of the kidneys to secrete urine, for in retention the kidneys are still functioning, and the urine is collecting in the distended bladder. Retention of urine occurring suddenly produces very severe pain and strangury, but in cases of old-standing obstruction the bladder may be distended enormously before pain becomes severe. If the retention remains unrelieved, urine may continually dribble away per urethram, when a condition resembling incontinence of urine is produced; but it is most important to distinguish the condition from *true incontinence* of urine due to injury or paralysis of the vesical sphincter muscle. In true incontinence the bladder remains empty, urine flows away as soon as it passes down into the bladder, and there is no obstruction in the urethra; whereas, in the condition of involuntary passage of urine from an unrelieved distended bladder—*incontinence from overflow,* or *false incontinence*—the bladder may be felt distended in the suprapubic region, and there exists some mechanical obstruction in the urethra, or at the internal urethral orifice.

The common causes of retention of urine are *urethral stricture* and *prostatic enlargement.* In stricture, it does not necessarily follow that the urethra is entirely occluded by the fibrosis, but rather that some spasm or congestion is present at the stricture, from exposure to cold or indulgence in alcohol, when a small catheter may be passed. In elderly men with *prostatic hypertrophy,* acute retention may occur early in the disease from a congested condition of the enlarged gland, or in the later stages be due to actual obstruction of the urethra by a localized enlargement from either lateral lobe or the so-called third lobe which acts as a ball-valve to the internal urethral orifice in such a manner that each forced attempt at urination closes the orifice more securely. A large coudé catheter can usually be passed readily; but in cases of acute retention, especially in those of old-standing obstruction in which the kidneys are probably affected by the backward pressure, *the urine must be drawn off very slowly*, otherwise fatal anuria may be induced.

A case of acute retention of urine from *stricture of the urethra* will generally be that of a comparatively young patient, who will give a history of former gonorrhœa, gradually increasing difficulty in micturition, narrowing of the stream, and inability to finish the flow completely without some dribbling of urine. Examination of the urethra by an endoscope, or by the passage of olivary-pointed flexible bougies, will reveal the presence of a stricture.

In *prostatic enlargement* the patient is usually above the age of fifty-five years, has been troubled with increasing frequency in micturition, especially at night, with straining and loss of force in the stream of urine. Per rectum, the prostate may be found to be enlarged both from above downwards and laterally; it may be smooth, elastic, and movable in the pelvic space in the case of adenomatous enlargement, or nodular, hard, irregular, and fixed in the case of carcinoma; the subjective symptoms of both are very similar. In some cases the prostate may not appear to be much enlarged upon rectal examination, though it is causing an intravesical tumour which obstructs urination, or a firm fibrous collar around the internal urethral orifice which gives rise to marked prostatic symptoms. In prostatic cases, even a large catheter of coudé form can usually be passed into the bladder readily. Retention of urine may also be present in cases of acute prostatitis or of prostatic abscess.

Acute retention of urine may be produced by other causes than the above. A *small calculus* may be passed into the urethra and totally obstruct the passage of urine. This may occur at any age, and the calculus become arrested at some narrow portion of the canal—usually at the meatus or at the membranous urethra. The urethra may lodge a calculus for some time with comparatively little pain; but more often the stone passes into the canal during micturition, causing a sudden pain, with cessation of the flow of urine and dribbling of a few drops of blood. The calculus may be palpated if it lies in the penile urethra or in the perineum, or will be felt on passing a metal instrument into the urethra.

Retention may be caused by the blockage of the internal urethral orifice by the free portion of a *pedunculated vesical tumour*. On any attempt at micturition the growth is forced into the orifice and obstructs it. These cases are rare, but in one under the care of the writer, a man, owing to his inability to pass any urine, had been condemned to catheter life on the assumption that he had prostatic enlargement. No enlargement could be felt per rectum, but upon cystoscopic examination a papilloma was found in the bladder, attached by its pedicle just above the urethral orifice and obstructing the flow of urine.

Retention of urine may also occur with *paralysis* of the *motor nerves* of the detrusor muscle of the bladder, or interference with the spinal centres by compression paraplegia, tabes dorsalis, or myelitis, each being diagnosed on examination of the nervous system; or as a reflex *spasm of the vesical sphincter* after operations upon the rectum or neighbouring organs.

In other cases, retention of urine is present in association with other symptoms of *hysteria*; but care must be taken not to give a diagnosis of hysteria until all other causes of retention are excluded. These cases usually occur in children or in young women.

Retention of urine occurring after operations about the anal or rectal areas or for hernia, etc., will be diagnosed readily.

5. **Pain during Micturition.**—Pain may be present *during* or *immediately after* micturition, and it is important to ascertain not only the period at which it is present, but also the actual *location* of the pain. If pain is present in the urethra during micturition, it usually indicates that a stricture or some inflammatory process is present, the latter being evidenced by a urethral discharge (see DISCHARGE, URETHRAL, p. 181). If pain is experienced *immediately after* micturition, and felt as a tingling or pricking sensation in the glans penis, there is some inflammatory or irritant process at the trigonal region of

the bladder. Formerly this symptom was looked upon as diagnostic of vesical calculus, and though it is almost a constant symptom of the latter, provided the calculus is not trapped in a post-prostatic pouch, it is also present in cystitis, tuberculous or otherwise, in vesical carcinoma which is infiltrating the bladder base, and in acute or subacute prostatic infections. Prostatic infection can be diagnosed by the history of the case, usually following an acute urethritis, and by a rectal examination. Tuberculous cystitis usually occurs in young adults, and frequently other tuberculous lesions are present in the genito-urinary organs, such as the epididymis, vas deferens, seminal vesicles, or prostate, whilst the urine contains not only blood and pus, but tubercle bacilli. Cystitis from other causes, and vesical growth or calculus, can be ascertained upon cystoscopic examination.

Pain may be felt in the *perineum* during and after micturition in cases of prostatic disease, especially if much straining occurs during micturition, or may be felt in both the perineum and the anal area in vesical carcinoma.

In the female, pain is felt at the urethral orifice and in the vulva after micturition in cases of cystitis or vesical carcinoma.

It should be noted that in either sex, severe pain may be present at the termination of the urethra after micturition when a *calculus* is impacted in the *vesical end of a ureter*, (*Fig.* 192, p. 455), especially if the latter is partially prolapsed into the bladder. In one such case the patient would hold her urine for hours rather than pass it, owing to the pain that followed micturition.

6. Micturition through Fistulæ.—Urine may pass, either wholly or in part, through a fistulous track communicating with the urinary organs, such opening being the result of preceding disease or injury. Occasionally, owing to congenital malformation of the urethra or bladder, urine passes by an opening in the perineum, pubes, or into the vagina ; but these cannot be regarded as fistulæ.

Urinary fistulæ in connection with the urethra are most common as the result of periurethral abscess, stricture, or some operation ; and in a case in which a penile fistula is present, it is necessary to ascertain if the calibre of the urethra is in any way narrowed by cicatricial inflammation. A fistula may open in the perineum as the result of inflammation and extravasation behind a stricture, following an operation upon the lower urinary organs, or in the female into the vagina from damage during parturition or some vaginal operation. In cases in which a fistula opens into the vaginal fornix, the urine may leak from the bladder or from the lower end of the ureter. The opening is usually small and embedded in an area of cicatricial tissue, so that it is very difficult to pass a probe along the track. In these cases, evidence of the nature of the fistula may be obtained by filling the bladder with some sterile coloured solution, such as weak methylene blue ; if the opening is in communication with the bladder, coloured solution will appear in the vagina, but if the urine comes from the ureter, no stain will be found. Evidence may also be obtained by means of the cystoscope, when a cicatricial area may be found in the bladder surrounding a retracted fistulous opening, or the ureteric orifice of one side may be found displaced from its normal situation by the scar contraction when the ureter is at fault. In these cases it may be impossible to pass a bougie into the ureter more than a very short distance, the tip being arrested by the scar tissue.

A urinary fistula may be present in the suprapubic area in connection with the bladder, or in the lumbar area communicating with the kidney, as the result of operations on these two organs. A fistula has been seen in the iliac fossa as the result of an operation on the ureter, and after the opening of an abscess formed around the ureter from the ulceration caused by a ureteric calculus.

7. Disorders of Micturition from Diseases of the Nervous System.—In most of the foregoing paragraphs it will be noticed that symptoms referable to the urinary organs have been stated to be due in some cases to disease of the nervous system, such as myelitis, tabes dorsalis, or hemiplegia ; in spite of repetition it is advisable to gather these under one heading. The control of the act of micturition depends upon the integrity of the nervous system ; for although special centres exist in the lower segments of the spinal area presiding over the motor functions of the bladder, the impulse calling for action of these centres is supplied by the brain after a stimulus has been conveyed to the latter by the sensory nerve fibres from the bladder. There are two centres in the lower spinal segment, by one of which the detrusor muscle of the bladder is brought into action, and by the other the sphincter muscle surrounding the vesical outlet is maintained in tonic contraction until inhibited by the same stimulus which produces contraction of the detrusor. The two vesical muscles are thus antagonistic in their action, the detrusor contracting and the sphincter relaxing in answer to the stimulus to micturition. In the diagnosis of all neuroses of the bladder it is most important to exclude all lesions of the urinary apparatus, and not to overlook the fact that vesical symptoms are often produced by some lesion in the kidney when the bladder on careful examination appears quite normal.

(*a*). *Irritability of the Sensory Nerves of the Bladder.*—Some patients experience an urgent and frequent desire to pass urine, often every half-hour, though no objective symptoms of disease can be found, and all inflammatory lesions can be excluded ; there is no pain and no increased frequency of micturition during the night. The cases have received the name of *cystalgia*, *hyperæsthesia vesicæ*, and *irritable bladder*, and they must be distinguished carefully from those in which there is some lesion of the urinary organs, the rectum, and of the female pelvic organs.

(*b*). *Irritability of the Motor Nerves of the Bladder.*—In this condition there is a spasmodic contraction of the sphincter muscle of the bladder, with resulting retention of urine or great difficulty in micturition. There is no stricture or urethral obstruction, as shown by the ease with which a catheter is passed, nor is there any prostatic enlargement. The neurosis is not confined to the male sex, and is seen in hysteria as well as in those nervous affections which affect the spinal centres, such as myelitis, lateral sclerosis, and tabes dorsalis.

(*c*). *Paralysis of the Motor Nerves of the Bladder* may affect the peripheral nerves or spinal elements, but the results as regards the bladder are the same. If the nerves supplying the detrusor muscle or its spinal centre be paralyzed, retention of urine occurs, and the patient can expel urine only by the force of the abdominal wall. If the sphincter muscle is affected, it becomes relaxed, and urine dribbles away. In many cases only part of the motor tract is affected, so that the power of the bladder is not abolished but diminished, and a portion of the urine is retained in the bladder after micturition. The bladder may be affected thus in compression of the spinal cord by fracture, or hæmorrhage into the membranes, in myelitis, paraplegia, and tabes.

(*d*). *Destruction of the Spinal Centres for Micturition*, by injury, softening, or compression, gives rise to incontinence without distention of the bladder. The urine dribbles from the urethra as fast as it enters the bladder. *R. H. Jocelyn Swan.*

MOUTH, PIGMENTATION IN.—(See PIGMENTATION IN THE MOUTH, p. 526.)

MUCUS IN THE STOOLS.—This occurs in such a variety of conditions that it is impossible to give a complete differential diagnosis of them here. Its presence always indicates organic disease, usually of the large bowel, for if it comes from the small bowel it will, unless the motions are very fluid, be so incorporated with them that it cannot be seen. It occurs in *malignant disease of the colon* as a clear glairy mucus, often blood-stained, and it has the same characters in *intussusception*, for the obstruction in both these cases accounts for the absence of fæcal colouring. It is often seen in *constipated motions*, the hard fæces having led to irritation of the large bowel, with consequent excessive secretion of mucus ; if this has lain some time in the bowel it has become coagulated into white shreds, which can be seen attached to the motions and look like parasitic worms. In severe

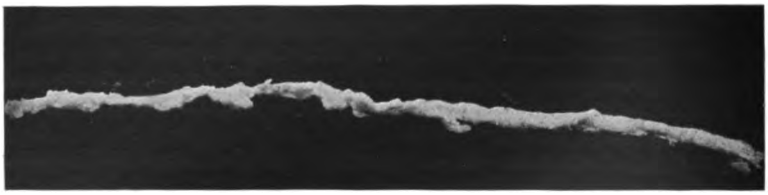


Fig. 172.—Tubular mucous cast of large intestine, much reduced in size, from a case of muco-membranous colitis. The cast measured 25½ inches in length.

cases a motion may consist almost entirely of these shreds; there may be little fæcal matter. If the mucus has not lain so long in the bowel, it appears like a jelly outside the motion. Sometimes, especially in adult women who are constipated, complete casts of the bowel formed of coagulated mucus are passed; they may be a foot or more in length (*Fig.* 172). Often, however, by the time they are passed, they have become broken into fragments which the patient describes as skins, and which look not unlike segments of tape-worm. Patients passing this variety of mucus are said to have *membranous colitis*. In the more acute varieties of inflammation of the bowel the mucus passed is jelly-like and semi-fluid, of varying colour according to the amount of fæcal staining. In severe cases of *enteritis* the motions consist of nothing but mucus and blood. It is impossible to attempt to differentiate here between all the numerous varieties of enteritis.

W. Hale White.

MUCUS IN THE URINE is generally of little clinical significance. Many normal urines, particularly those of women, develop a faint or even a more definite deposit of mucus, which may remain in suspension or may accumulate as a light floccular deposit at the bottom of the specimen-glass. Such mucus is a normal product of the epithelial cells of the urinary passages. It is not possible by merely looking to say whether it is in excess or not. It may indicate catarrh of the mucous membranes; but such catarrh will be shown more decisively by the occurrence of epithelial cells or actual pus corpuscles, or by a cause for catarrh such as OXALURIA (p. 423); diagnosis depending not upon the mucus but upon the other substances present with it. It is important not to mistake elongated strands of mucus for tube-casts; the error is particularly apt to occur if the cover-glass, on being pressed down on a specimen stained with methylene blue, slips slightly and draws out the mucus into long narrow strands. When large numbers of these are seen all parallel with one another, they are not likely to be mistaken for casts. Mucus stains readily either with methylene blue or with eosin, but exhibits no structure beyond granular particles, or cells that may have become entangled in its meshes.

If a male patient has formerly suffered from gonorrhœa, a residual catarrh of the glands in the prostate often persists long after the cure may have seemed to be complete. Urine from such a case, looked at in a tall glass vessel, often exhibits numerous filaments or 'prostatic threads,' consisting for the most part of mucus coming in the form of casts from the prostatic tubules.

Herbert French.

MUSCÆ VOLITANTES.—(See BLACK SPECKS BEFORE THE EYES, p. 71.)

MUSCULAR ATROPHY.—(See ATROPHY, MUSCULAR, p. 59.)

MYDRIASIS.—(See PUPIL, ABNORMALITIES OF THE, p. 551.)

MYOSIS.—(See PUPIL, ABNORMALITIES OF THE, p. 551.)

NAILS, AFFECTIONS OF THE.—Various pigmentary and degenerative changes may occur in the nails as the result of occupation, as in dyers, washerwomen, jewellers, confectioners, and others; or the condition known as *pterygium* may arise, the fold of skin at the proximal end of the nail adhering and growing over the nail, like a 'wing.' The nails are liable to attack also in such cutaneous affections as ringworm, favus, eczema, psoriasis, and epidermolysis bullosa. The differences between the onychomycosis due to *ringworm* and that due to *favus* are described in the article on FUNGOUS AFFECTIONS OF THE SKIN (p. 246). In *eczema*, usually the first sign of involvement of the nails is pitting, which gives them an appearance somewhat resembling orange-rind. They become discoloured and thinned, transverse and longitudinal splitting follows, and finally exfoliation may occur. In long-standing cases they may be thickened to the extent of deformity. In *psoriasis*, if the matrix of the nails is attacked, they become furrowed transversely, and dull in colour; later the nails split and may be shed, but not permanently. In other cases, instead of the matrix being affected, the nails are discoloured about the free border, and they become thickened as the discoloration extends downwards to the root. In *epidermolysis bullosa* there may be repeated bleb-formation at the finger-ends, causing atrophy of the skin and loss of nails. The signs of nail involvement in these three conditions are sufficiently distinctive to obviate confusion between them ; and the lesions elsewhere will aid the diagnosis.

Trophic changes in the nails may also be consequent on *acute illness* or *senile decay*, or they may occur without any apparent cause : the longitudinal striæ may be exaggerated, transverse furrows may appear, or white spots may develop, and a large part or the whole of the nail may become white (*leuconychia*). With this condition *spoon-nails* may be associated ; the nail becomes thin and hollowed, either from side to side or antero-posteriorly. Shedding of the nails may occur not only in distinctively cutaneous affections, but also in diabetes mellitus and syphilis, in locomotor ataxy and other nervous disorders. Either without definite etiology, or in connection with inflammation of the finger-tips, the nail may be separated from its bed without being actually shed. *Onychia*, or inflammation of the nail, is in some instances due to syphilitic or tuberculous infection ; in the latter case associated scrofulous lesions will often be found in the eyelid and elsewhere. Onychia, however, may also be due to trauma, or to contact with irritants used in industries, or may be idiopathic. Whatever the cause, the condition cannot be mistaken. If the process is acute there is great pain, with redness ; suppuration takes place beneath the nail, which becomes thickened and discoloured, and is ultimately shed, leaving an unhealthy sore. If this should fail to heal, the lymphatics may be involved, and the case becomes one of *paronychia*, or whitlow. This condition is sometimes caused by the pressure of tightly-fitting boots, or by irritation set up by the edge of a badly-cut nail—usually that of the big toe. *Onychorrhexis*, brittleness of nails, may be either congenital or acquired. It is sometimes present in cheiropompholyx, and in other cases is associated with nervous affections and anomalies of development. In *onychauxis*, hypertrophy of the nail, there may be overgrowth in one or in all directions, accompanied by distortion or discoloration, and sometimes by inflammation. In some cases the free end may grow to a great length, and may become twisted like a ram's horn (*onychogryphosis*). This curious distortion is often found in connection with congenital ichthyosis. A rarer condition of modified nutrition is that known as *egg-shell nail*, which is intimately associated with hyperidrosis ; it has been met with in debilitated young women ; the nail tends to grow upwards rather than forwards ; its connection with the distal portion of the bed is enfeebled ; and in typical cases the colour is precisely that of the inner face of the shell of a hen's egg—a delicate combination of white and purple. It has been suggested that, owing to the maceration of the distal portion of the nail-bed due to hyperidrosis, there is interference with the normal cornification of the nail-plate. Whatever the process, the diagnosis is clear. Transverse ridging of the nails due to previous illness (*Fig.* 306, p. 769) is described on p. 769. (See also FINGER, SORE, p. 239.) *Malcolm Morris.*

NAPKIN-REGION ERUPTIONS.—Infantile eruptions in this region, when they are a manifestation of *congenital syphilis*, are usually erythematous or papular, but they may also be pustular, bullous, squamous, or polymorphic ; in all cases alike they are distributed symmetrically on the buttocks. Frequently, around the anus and the genital organs the papules are moist and coalescent, and form flattish condylomata. Similar lesions are also found on the soles, palms, forehead, and around the mouth, and in these regions also the distribution is symmetrical. The eruption is as a rule transitory. The other symptoms of hereditary syphilis are so characteristic that the lesions here described are seldom liable to misinterpretation. The skin eruption is usually preceded by a chronic coryza (' snuffles ') and laryngitis. Often the nails are severely affected coincidently with the skin. The colour of the lesions, approximating to the characteristic raw-ham tint, the loose, dry, *café-au-lait* skin, the senile aspect of the face, and the accompanying cachexia, form a distinctive clinical picture.

A napkin-area eruption which was often mistaken for congenital syphilis is the *infantile erythema of Jacquet*. It is a process which manifests itself in : (1) Simple erythematous, (2) Erythemato-vesicular, (3) Papular, (4) Ulcerating forms. These may develop consecutively or coincidently. The most common are the erythematous and the papular. All alike are probably due in part to the irritation set up by moist or soiled napkins, but vasomotor irregularities and gastro-intestinal toxæmia may also be concerned in the etiology. The preference sites of all four forms of the eruption are the *convex* surfaces of the buttocks, of the thighs, and of the scrotum or vulva. In the simple erythemas, of which the usual subjects are quite young infants, the rash may be limited in mild cases to the genitalia, the inner sides of the thighs, and the perineum, while in severer cases it may extend to the lumbar region, the lower abdomen, and the calves and heels. In the erythemato-vesicular form there appear on the convex surfaces towards the centre of the erythematous areas small bright-red erosions which, forming groups of from two or three to a dozen or more, may become confluent; the erosions are preceded by vesicles, which may usually be found near the borders of the reddened area. The erythemato-papular form of the eruption is met with when the erosions just described have thrown up flattened granulations, which give to the lesions the appearance of flat, reddish papules. In this stage the heels and the lower abdomen may be involved in the erythema. In the fourth form of the eruption the erosions, failing to granulate, develop into ulcers, with sharply defined borders or coalescing into vermicular lesions. They are confined to the convex surfaces, the folds always escaping. Attention to the appearance and distribution of the lesions, and the course they run, together with the absence of the more familiar signs and symptoms of congenital syphilis, will prevent confusion with that disease, or with the condition which Colcott Fox has styled vacciniform erythema of infants.

Pemphigus neonatorum consists of an eruption of bullæ on the thighs and buttocks in new-born infants. It is not, however, confined to this region, but attacks other parts, including the face, and this is true also of the bullous impetigo of older babies, which Adamson believes to be, like pemphigus neonatorum, a form of the impetigo contagiosa of Tilbury Fox. The diagnosis of these affections has been given under BULLÆ, p. 96).

The 'seborrhœic eczema of infants' has been styled 'seborrhœic dermatitis of infancy' by Adamson, who was the first to lay stress upon its special incidence upon the napkin region, and who does not regard it as a form of eczema. The whole napkin region is occupied by a uniform bright-red rash, for the most part covered with moist or greasy yellowish scales, though in prominent parts the surface may be smooth and polished. The margins of the area are sharply defined. The rash often extends downwards to the thighs and calves, and upwards to the umbilicus, while beyond this area there are smaller patches and many pin-head, red, scaly papules. Other parts that are attacked frequently are the bends of the knees, the flexures of the elbows, the axillæ, the side of the neck, the naso-labial fissure, and behind the ear. On the scalp will always be found a red, squamous or crusty eruption. The diagnosis rests upon the distribution and the sharply defined margins, with the patches and crusted papules. It is assisted by the readiness with which the eruption yields to mild local parasiticidal applications. In cases of congenital syphilis which mimic this condition, the presence of the concomitant specific signs, as enumerated above, will prevent confusion between that disease and 'seborrhœic eczema.'

In adults the same region, known as the bathing-drawers area, is liable to attack in a number of affections. In *eczema margination* (tinea marginata, as I prefer to term it), *dhobie's itch*, and *erythrasma* the eruption occurs exclusively, or almost exclusively, in this region; of these affections the differential diagnosis has been given under RINGWORM (p. 247). In *pediculosis pubis* the pubes may alone be affected, or the parasite may wander to the abdomen, the thorax, the axillæ, and may even reach the beard, whiskers, and eye-lashes. The diagnosis of this condition can present no difficulty. In *scabies* the lesions may be very slight on the hands and wrists, and the brunt of the attack may be borne by the penis and scrotum, the lower part of the abdomen, and the thighs. In *psoriasis* the eruption is sometimes very severe in the bathing-drawers area, of which the surface is an almost uniform deep red, and is the seat of profuse desquamation, while on the special sites of election—the knees and elbows—the lesions may be quite insignificant. In *eczema intertrigo* and *erythema intertrigo* the folds in the area under consideration are only liable to attack in common with folds in other parts. The diagnosis of these affections has been given elsewhere; but it may here be mentioned that in diabetes, eczema may begin on the penis or the vulva, and may spread thence to other regions. Other conditions which may affect this area specially are: acute traumatic erysipelas, pruritus ani, small-pox in the prodromal stages, and the various forms of syphilis. In syphilis the commonest site for the moist papule is around the anus and genitalia (see PAPULES, p. 490).

Malcolm Morris.

NEURITIS, OPTIC.—(See OPHTHALMOSCOPIC APPEARANCES, NOTES ON, p. 415.)

NIGHTMARES may occur at any age, but they are particularly common in children between four and eight, when they may be so bad and persistent as to merit the term night-terrors. The commonest cause for a nightmare in an adult is some indiscretion in diet, the last meal having been taken too late in the evening, or else having contained some injudicious article. The symptom is not otherwise of diagnostic import, though some individuals, particularly those of nervous inheritance, are more liable to nightmare than others, and the tendency is certainly increased by such excitement as the reading of thrilling novels or participating in unusual events. Children are particularly prone to night-terrors during term-time, when they are working at high pressure; during the holidays the symptom often disappears. Those who are keenest upon their school work are apt to suffer most, and similar evidence of excitability of the nervous system is exhibited particularly by those who have a tendency to acute rheumatism in the form of chorea. Night-terrors may occur in these patients without any other cause than over-pressure, particularly if they lie upon the back rather than upon one side during sleep; but the tendency is much increased by errors of diet, such as the eating of unripe fruit and so forth, by the presence of intestinal worms, and by the existence of adenoids, with or without enlarged tonsils. *Herbert French.*

NIPPLE, DISCHARGE FROM.—(See DISCHARGE FROM THE NIPPLE, p. 181.)

NODULES in ordinary dermatological usage are solid elevations larger than a papule and smaller than a tumour; the definition, however, makes no pretence to scientific exactitude. Nodules differ from papules not only in size but also in their greater tendency to downward growth; the substantial difference between a nodule and one of the larger papules is that the one is a solid lesion extending upwards, while the other is a solid lesion projecting both upwards and downwards. They may be neoplastic, or hypertrophic and inflammatory.

Little need be said here of the nodules met with in some malignant diseases, for the differential diagnosis of carcinoma and of sarcoma will be found under TUMOURS OF THE SKIN (p. 730). Fibroma, myoma, and cysts are also dealt with under this heading, and glanders in the articles on PUSTULES (p. 559).

The nodules of *lupus vulgaris*, arising in either the superficial or the deep part of the corium, are soft, brownish-red, and translucent, resembling apple jelly. At first buried in the skin, they presently appear as discrete papules the size of a pin's head, arranged in groups or in irregular circles, dull red at the outset, but afterwards pale. Gradually the papules develop into nodules, the intervening skin meanwhile becoming thickened by cellular infiltration, reddened by inflammatory stasis, and raised into a patch which is covered with fine branny scales. Around the edge of the patch new nodules spring up, and thus a large area of skin may be invaded. The disease usually starts from a single focus, but others may arise and, spreading separately, may involve large areas of cutaneous surface. The patch may undergo slow involution and be followed by scarring; but much more often ulceration occurs, the sore being covered with a greenish-black crust, around the ragged edges of which will be seen apple-jelly nodules in various stages of development. In parts like the nose there may be necrosis of cartilage, but there is never erosion of bone. The apple-jelly nodule is the chief diagnostic feature of lupus vulgaris. In typical cases the patch described above, with its infiltrated, raised surface, its well-defined edge studded with the nodules, and its covering of fine scales, can hardly admit of misinterpretation. Less typical cases may require to be differentiated from lupus erythematosus, rodent ulcer, epithelioma, scrofulodermia, and syphilis. *Lupus erythematosus* begins as minute red points, not as dull-red papules, and the lesions never develop into apple-jelly nodules, nor do they ever ulcerate or extend to the deeper parts and erode cartilage. They are symmetrical in distribution, as lupus vulgaris scarcely ever is, and the affection seldom appears before puberty, as lupus vulgaris almost invariably does. It is only when the lesions peculiar to lupus vulgaris are masked by œdematous swelling that the two affections can be confused; but if the skin at the spreading edge be stretched, small amber-coloured nodules can usually be seen.

In *rodent ulcer* there is usually but one lesion, which runs a much more sluggish course than the nodules of lupus vulgaris; the ulcer has an indurated border and a firm base, and penetrates deeply into the tissues ; and the disease is essentially one of later life (see Ulceration of the Face (p. 735). *Epithelioma*, again, is a disease of later life. The hard, everted edge of the growth, the foul base, frequently roughened with warty formations or sprouting with cauliflower-like excrescences, the implication of neighbouring glands (which very occasionally, however, occurs in lupus), and the secondary deposits, form quite a different clinical picture from that of lupus vulgaris.

In one form of *scrofulodermia* nodules develop under the skin, and an ulcer is formed which is bordered by dark bluish, thin, undermined skin that has too little vitality to allow of repair ; there is no infiltration as in lupus vulgaris, the nodules do not present the apple-jelly aspect, and other evidences of the disease will be found on the neck or elsewhere, in the form of enlarged glands or scars. As, however, the two conditions frequently co-exist, and the treatment is virtually the same, diagnosis between the two is of little practical importance.

In the diagnosis from '*lupoid*' *tertiary syphilis*, again, the apple-jelly nodule of lupus vulgaris plays the chief part. The syphilitic process, further, is much more rapid, nor is acquired syphilis generally a disease of early life. The nodules and ulcers of late syphilis—neoplasms that grow by infiltration of the surrounding parts and often break down into ulcers which are prone to become serpiginous, and show little or no tendency to spontaneous cure—have in turn to be differentiated from other conditions. They may be mistaken for abscess, but if opened they give issue not to pus but to a gummy liquid. If the ulcer into which the gumma breaks down be on the leg, it may resemble callous ulcer, but its obduracy to ordinary treatment and its response to the iodides will reveal its true nature. Wassermann's serum test will probably be positive. From syphilitic ulcer rodent ulcer differs in its hard edge, and red, shining, dry floor, as well as in its favourite situations ; from epithelioma, in that a process of new growth has preceded the ulceration ; from scrofulodermia, in the undermined border of the ulcers and the slow rate of the process.

In *yaws*, as in syphilis, the nodule is the most characteristic lesion of the tertiary stage. It arises in the subcutaneous tissue, and generally leads to the formation of superficial ulcers which spread serpiginously, like the ulcers of tertiary syphilis. New nodules frequently appear in the neighbourhood of the older ones, and masses resembling syphilitic gummata may form and break down into ulcers. These late ulcers mostly appear on the lower part of the leg, especially around the ankle, but they are not uncommon about the lips, and indeed may occur in any part of the body. The clavicle, sternum, ulna, tibia, and the metacarpal and metatarsal bones, are often the sites of nodules which may occasion permanent thickening, or break down and cause ulcers. Between yaws and syphilis there are obvious resemblances in the tertiary stage, but there are marked differences in the primary and secondary stages. In yaws the inoculation lesion is not indurated, there is seldom distinct glandular enlargement, the mucous membrane lesions of syphilis are absent, and the most characteristic lesion, which appears in the secondary period, is the frambœsial granulomatous excrescence known as the yaw (see Scabs, p. 601). In yaws, the exanthem, the alopecia, the iritis, the affection of the permanent teeth, the bone lesions, the polymorphism, the nerve lesions, and the gummata of syphilis are wanting. Yaws is never hereditary nor congenital ; yaws and syphilis confer no immunity against each other, and yaws may die out in a community while syphilis remains, or it may be universal in a community where syphilis is unknown. The minute histology of the lesions of the two diseases also furnishes important differences.

From *tuberculosis* yaws differs (apart from the tubercle bacillus) in the absence of the characteristic tuberculous architecture with its giant-cells and daughter plasma-cells, more marked disintegration of the fibrous stroma, and complete disappearance of the blood-vessels.

In *leprosy* the nodule (*Fig.* 173) marks one of the three types of that affection, the others being nerve or anæsthetic leprosy, and mixed or complete leprosy. In nodular (or tubercular) leprosy the macules, which are always the primary lesion, are transformed into nodules by sudden increase of inflammatory infiltration. When fully developed they vary in size from a small shot to a filbert, or larger, are round or oval, but raised considerably above the level of the skin. They may mimic lupous nodules, syphilitic papules, rosacea, erythema nodosum, or sycosis. Sometimes telangiectases may be observed on their surface. They are elastic to the touch, are at first sometimes hyperæsthetic, but later very frequently become temporarily or permanently anæsthetic. Nodules on the mucous membranes are red or grey, and may resemble syphilitic lesions. Both on skin and on mucous membrane they tend to break down, but in exceptional cases they either undergo cicatricial shrinking or reach the ulceration stage by way of suppuration. The differential diagnosis of leprosy in the macular stage is given under Macules (p. 383). In the later stages the identification of the disease seldom presents difficulty. The nodules of leprosy may resemble those of lupus vulgaris and the tubercular syphilide, but the lupous and syphilitic eruptions are both of limited extent, and there is no anæsthesia. The syphilide also is serpiginous, or occurs in crescentic groups. Wassermann's serum test may be positive in leprosy without syphilis, and therefore cannot be relied on in differentiating the two. In the early stages of nodular leprosy the lesions may strongly resemble those of *erythema nodosum*, and, as in that affection, there may be pains about the joints ; but if the case be one of erythema nodosum the nodules will disappear within a fortnight, though successive crops may arise for three or four weeks longer. Preceded and accompanied by pains about the joints, by pyrexia and other symptoms of constitutional disturbance, oval nodules, ranging in size from a walnut to a hen's egg, appear on the legs and feet and, less frequently, elsewhere. They are most common between the knees and ankles, next between the wrists and elbows. In colour they are at first bright red, but soon become bluish in the centre and purple at the periphery, exhibiting as they subside the changes of tint presented by a bruise. Erythema nodosum is an affection of adolescence, and girls are attacked by it twice as often as boys. There is nearly always considerable pyrexia, and acute osteomyelitis of the tibia may be suspected if it is not remembered that erythema nodosum is nearly always bilateral, which osteomyelitis seldom is. There is never ulceration, and this, with the pains and swellings about the joints, distinguishes it from syphilitic nodules. The same features distinguish it also from an *erythema of the legs*, the result apparently of excessive standing, to which young girls are sometimes subject, and from the node-like swellings which sometimes occur in the legs of women suffering from *varicose veins*.

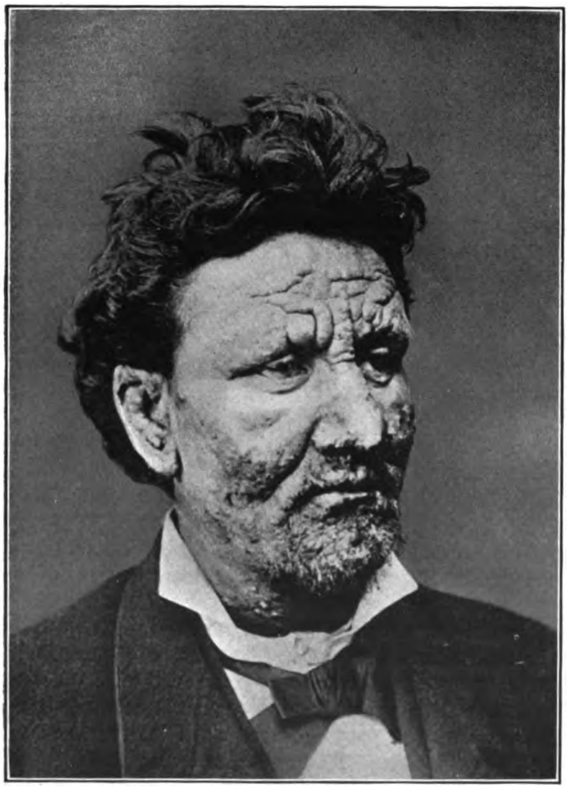


Fig. 173.—A typical case of nodular leprosy in a Norwegian. *(From a photograph by Dr. Armauer Hansen.)*

The absence of ulceration and the presence of joint-pains are points which differentiate erythema nodosum from *erythema induratum scrofulosorum* (Bazin's disease). Here the nodules, which occur chiefly on the legs, are at first subcutaneous, and can only be felt, not seen. They are generally discrete, but may become fused together into a solid, infiltrated mass, and are apt to break down into irregular ulcers. They differ from the nodules of erythema nodosum not only in the features already noted, but also in colour, being violet instead of bright red, and not undergoing successive bruise-like changes of tint. From gummata they differ in being less painful and in running a less rapid course, as well as in being more numerous, and in attacking both legs. The only effect of antisyphilitic treatment is to aggravate the condition. The nodules of *erythema keratodes* differ from those both of erythema nodosum and of erythema induratum scrofulosorum in that they appear only on the back of the finger-joints, while on the palms and soles there is overgrowth of the horny tissue, accompanied by œdema and tenderness.

The condition which Boeck designated *multiple benign sarcoid*, or *miliary benign lupoid*, presents some resemblance to lupus and sarcoma. The nodules, at first rose-coloured, afterwards become livid, then brownish. In size they vary from a millet seed to a large bean. The favourite sites of the eruption, which is always symmetrical, are the face, shoulders, wrists, and the extensor surfaces of the upper limbs; but exceptionally the scalp, the back, and the lower limbs are attacked. Occasionally the lymphatic glands are enlarged. The nodules never break down, but after a period, it may be of several years, shrink and disappear, leaving a slight atrophic scar. The affection, which often accompanies visceral tuberculosis, is distinguishable both from sarcoma and from lupus vulgaris by histological examination, as well as by the course it runs. From the latter affection it is distinguished also by the negative reaction in inoculation experiments.

The subcutaneous nodules of *acute rheumatism* generally occur over the sheaths of tendons and the fascia covering bony prominences, around joints, and on the scalp. They may be as small as a pin's head or as large as a bean. Within limits they can be made to glide on the underlying tendon-sheath or fascia. They are sometimes met with in adults whose hearts have not been damaged by the toxæmia, but much more frequently in children with distinct valvular lesions, and according to some authorities they are analogous to, if not identical with, the nodules that have been found post mortem on the borders of the mitral curtain and in the myocardium. The coincidence of nodules such as these with rheumatic fever can leave no doubt as to their true nature. *Heberden's nodes* (*Fig.* 155, p. 343), the little knobs on the finger-joints which are caused by osteophytic outgrowths from the bases of the distal phalanges in certain elderly persons, are unmistakeable, and the same applies to the nodular pads which sometimes develop on the backs of the knuckles (*Fig.* 160, p. 347). Multiple subcutaneous *cysticerci* are a rarity the diagnosis of which may be suggested by the eosinophilia, but can only be clinched by excision and microscopical examination of one of the nodules.

Malcolm Morris.

NOISES IN THE EARS.—(See TINNITUS, p. 722.)

NOISES IN THE HEAD are complained of by two entirely different classes of patient; namely, (1) *The insane*, and (2) *The sane*.

1. **The Insane.**—In these cases the noises may be of indistinct or indeterminate nature, but more often, in addition to mere noises, buzzings, singings, roarings, hootings, there are more definite subjective auditory sensations, which as a rule take the form of voices. They then constitute a variety of either hallucination or delusion—the former if there is some organic mischief at the bottom of sounds which are misinterpreted, the latter if the voices are pure fancy. In either case the hearing of the voices needs to be persistent to constitute evidence of insanity, for most normal persons have transitory subjective sensations of having been spoken to when they are quite alone. If, however, the patient persistently hears voices when there are none, other evidence of insanity should be looked for, though it often takes an expert psychologist to detect the nature of the mental malady. The voices may appear to the patient to be definitely within his own head; on the other hand, they often appear to be external voices, sometimes strange to the patient, sometimes familiar; attributed perhaps to a non-living person, a dead wife, or God, or Christ; perhaps to a living person who is far away—a straying daughter, an old friend, a loving mother. The voices may say different things at different times, or they may constantly reiterate the same sentence; in the worst cases they urge the patient to this or that particular action, especially suicide or homicide. When they have reached this degree there can be little doubt as to their indicating insanity. They do not, however, belong to any one type of insanity in particular; they may occur in melancholia, accusing the patient of having committed the unpardonable sin and urging him to suicide; or in mania, urging to homicide for some supposed wrong; or in general paralysis of the insane; or in either the melancholic or maniacal phase of *folie circulaire*. The chief difficulty in diagnosis arises in the early stages, or when a patient hears diffused sounds of subjective origin, due perhaps to organic causes, but feared by the patient

himself to be a sign that he is going mad. In such cases much discretion may be required in deciding the psychological import of the noises complained of.

2. **The Sane.**—Perfectly sane persons, however, may be bothered tremendously by subjective noises in the head—sensations resembling the blowing off of steam by railway engines; crackings and groanings; hissing or buzzing noises; rhythmical pulsating noises; clatterings and dins like hundreds of drums beating at one time; roarings; hammerings, and so on. As a rule it is possible, on careful inquiry from the patients, to differentiate these into two main types—namely, (1) Those in which the noises rapidly wax and wane, though they are never absent, the variations having a more or less rhythmical character related to the pulse rate; and (2) Those in which there is no such rhythm in the subjective noises heard. The former group comprises cases in which the fault lies either in the blood itself or in the cerebral vessels through which it flows; in the latter group the cause usually lies in the ear itself—external, middle, or internal—or in the hearing centre in the temporo-sphenoidal lobe of the brain. In either case the patient may live for years, and get used to the troublesome noises; general hearing may be quite good in spite of them; and it is often very difficult to be quite certain of their cause because there is so little post-mortem evidence to base their pathology on. The patients do not die in hospitals, and in general practice post-mortem research into the causation of the noises is rare.

The hæmic and vascular conditions to think of in connection with the first group are the following:—

Arteriosclerosis: Granular Kidney.—Evidenced by high blood-pressure readings with the sphygmomanometer and by albuminuria.
Aortic Regurgitation.—Evidenced by the aortic diastolic bruit and the highly pulsatile arteries generally.
Atheroma of the Cerebral Arteries.—Guessed at on account of the age of the patient and the condition of the thickened radials and the tortuous temporal arteries; there need be no increased systemic blood-pressure.
Severe Anæmia (see ANÆMIA, p. 20).—Any condition of severe anæmia may cause noises in the head, but it is most marked in cases of pernicious anæmia (p. 24), severe chlorosis, and anæmia due to any sudden blood loss,—due for instance to post-partum hæmorrhage, bleeding of a duodenal ulcer, hæmatemesis, or hæmoptysis.
Graves's Disease (p. 229).—In which all the arteries may be pulsating very violently.
Purely Functional Conditions, with or without Hysteria.—Especially when there is also undue pulsation of the abdominal aorta (p. 543) and great exaggeration of the knee-jerks, often visceroptosis, and mobility of the right kidney.
Toxic Conditions.—Especially intestinal toxæmia from any form of chronic constipation; or from alcoholism, acute and chronic; or from the effects of certain drugs, especially salicylates, aspirin, quinine, arsenic, iodide of potassium, plumbism.

When these causes can be excluded, and there is reason to suppose that there is or has been trouble in the ears themselves, the various conditions that need to be considered are:—

Wax in the ear.
Inflammation of the external auditory meatus.
Blows upon the ear.
The effects of work carried on amid exceptional circumstances of noise, e.g., boiler-makers, rivetters; or under exceptional conditions of external pressure, e.g., under high pressures: divers, caisson workers, workers in very deep pits; or under low pressures: mountaineers, those who live at high altitudes, balloonists, airmen who ascend to great heights.
The effects of chronic otitis media (otosclerosis), generally preceded by OTORRHŒA (p. 421).
Chronic thickening of the bones containing the ears; for instance, as part of osteoporosis, osteitis deformans (p. 155), acromegaly (p. 237), chronic syphilis of the bones, leontiasis ossea.
Chronic thickening of the meninges in relation to the petrous bone; secondary to otitis media; due to injury, age, or syphilitic pachymeningitis.
Tumour, abscess, or inflammatory changes affecting one temporo-sphenoidal lobe.

When the noises in the head are due to ear trouble, they are likely to be much more marked upon one side of the head than the other; when due to vascular or hæmic states, they are more likely to be symmetrical; this general rule, however, is liable to exceptions either way, and in all cases a full examination of the ears is necessary, especially by means of the aural speculum and by the tests for hearing described under DEAFNESS (p. 164).

Any of the conditions mentioned in either of the main groups above may be associated with vertigo, so that the latter is not so useful a symptom in the differential diagnosis as might be supposed. Upon the whole, however, it is true that vertigo is to be expected more with either local ear conditions or with arteriosclerosis than with any of the others, so that in a case in which vertigo is prominent but the blood-pressure is not raised, the probability of some local affection of the outer, middle, or internal ear, especially perhaps of the semi-auricular canals, will be considerable.

The Wassermann reaction should be tested in all cases in which there is any hesitation in the diagnosis ; if it is positive a syphilitic cause will be likely, and it is surprising how many nerve symptoms of obscure origin are really syphilitic. Incidentally, in regard to this, though it has no connection with the present subject, it is noteworthy that quite a considerable proportion of cases of apparently simple sciatica have been shown to be due to syphilis in this way.

The great majority of patients who complain of noises in the head are adults, and most of them are past middle age. The symptom is quite uncommon in children.

Herbert French.

NOSE, BLEEDING FROM.—(See EPISTAXIS, p. 220.)

NOSE, DISCHARGE FROM.—(See DISCHARGE, NASAL, p. 178.)

NOSE, REGURGITATION OF FOOD THROUGH.—(See REGURGITATION OF FOOD THROUGH THE NOSE, p. 588.)

NUMBNESS OF THE FINGERS.—(See SENSATION, ABNORMALITIES OF, p. 604.)

NYCTALOPIA.—(See VISION, DEFECTS OF, p. 763.)

NYSTAGMUS.—Several varieties of associated tremor of the two eyes are comprised under the term nystagmus. These are : (1) Searching movements ; (2) Pseudo-nystagmus ; and (3) Nystagmus proper.

1. Wide purposeful and slow movements of the eyes in all directions are usually seen in people who are born blind or have lost the power of fixation as the result of some obstruction of the retina or choroid at the yellow spot. The eyes appear to be seeking for something but never rest on any definite object.

2. Pseudo-nystagmus, which is commonly confused with true nystagmus, is the term applied to rapid jerking movements of the eyes when they are carried to the extremity of an excursion in any direction. The eyes, instead of remaining fixed on the object, rapidly recede from their position and return to it at the rate of four or five oscillations a second. This condition is a characteristic symptom in *Friedreich's* or *hereditary ataxy*, and is also met with in 40 or 50 per cent of cases of *disseminated sclerosis* and in many cases of *cerebellar tumour*. For the differential diagnosis of these conditions see PARAPLEGIA, p. 510.

3. Nystagmus proper is the term applied to the condition in which the eyes make rapid regular oscillations about a fixed point, not only at the extremity of an excursion, but when the eyes are otherwise at rest, and looking directly forward. The oscillations may be in the vertical or the horizontal meridian, or may in some cases exhibit a rotatory form. The condition is usually bilateral, though it is occasionally met with affecting one eye only, and in some rare cases the character of the nystagmus may differ in the two eyes.

True nystagmus is caused by :—

(*a*). Conditions causing defective vision in the early months of life. As a result of such affections, the macular region is not differentiated from the surrounding portions of the retina as is the usual course in the early months of infant life, and power of fixation is never acquired. Conditions which may thus cause nystagmus are ophthalmia of the new-born, congenital cataract, colour blindness, albinism, and certain cases in which there is an unusual distribution of the retinal pigment. The diagnosis of these various conditions depends on an accurate examination of the eye.

(*b*). Conditions developing in later life due to constant strain from peculiar occupations, as for example *miners' nystagmus*, in which it is probably caused by continued work in a dim light, where the central vision necessary for steady fixation is comparatively ineffective, and in which it is associated with other symptoms of failure of the central nervous system. As a rule it improves on the cessation of the occupation which causes it.

(*c*). *Aural irritation*, in which it is usually associated with vertigo.

(*d*). Nystagmus may also occur in about 12 per cent of all cases of *disseminated sclerosis*.

(*e*). In certain cases of *cerebellar tumours* it is a marked symptom; and it may occur,

(*f*). In various rare conditions, after traumatism or poisoning, and possibly syringomyelia.

H. L. Eason.

OBESITY implies an excessive accumulation of fatty tissue in the body. It is not necessarily pathological, but even in otherwise healthy persons obesity ultimately incommodes them, and is very liable to lead to cardiac symptoms due to fatty changes in and around the heart. The following are some of the chief causes :—

Heredity
Continued over-eating
Continued drinking of malt liquors
Too little exercise
A pre-glycosuric state
Chronic parenchymatous nephritis
Testicular atrophy or excision

Ovarian insufficiency
Hypothyroidism
Hypopituitarism
Cerebellar tumour
Hypernephroma
Adiposis dolorosa or } Dercum's disease.
Diffuse lipomatosis }

The majority of the above need little discussion. Families in which all the members tend to run to fat are familiar enough ; the individuals may weigh anything from 16 to 30 stone, without necessarily being ill. Over-eating, over-drinking, and under-exercising are generally obvious if the patient's mode of living is known. The *pre-glycosuric state* is particularly important from the point of view of life insurance : when a young man or woman under thirty-five begins to run to fat without apparent cause, it is clear that there is an error in his metabolism ; there may be no glycosuria at this time, but in quite a number of these cases the error of metabolism develops as time goes on, until presently there is glycosuria, and finally typical diabetes mellitus.

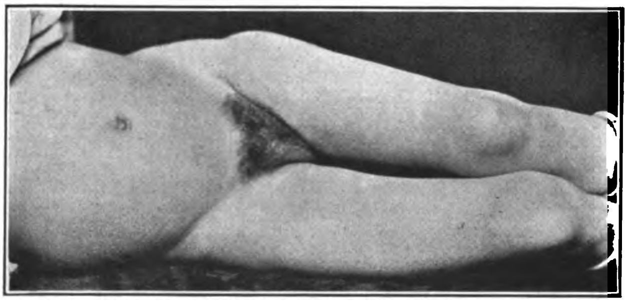


Fig. 174.—A girl, aged 6, suffering from hypernephroma, which proved fatal from secondary deposits in the lungs: the photograph shows the premature development of pubic hair, which in this case appeared at the age of eighteen months.

Chronic parenchymatous nephritis sometimes gives rise to a large, pale person, who looks, and is, fat and flabby. Part of the apparent fatness may be due to excess of fluid in the tissues, but there need be no obvious œdema with pitting on pressure. There may or may not be a history of previous acute nephritis—some of these cases arise insidiously : the diagnosis is not difficult, however, where renal tube casts and an abundance of albumin are found in the urine, particularly if there is a big heart, a prolonged first sound at the impulse, a ringing aortic second sound, a high blood-pressure. and perhaps albuminuric retinitis.

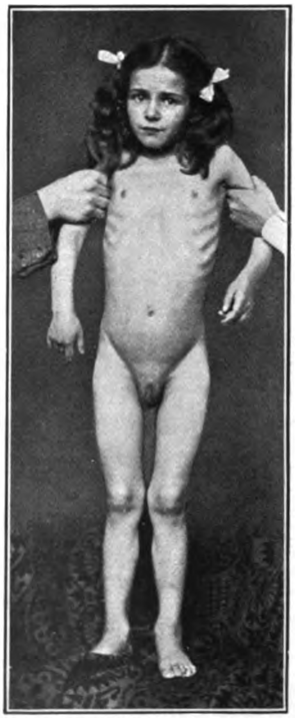


Fig. 175.—The same case as *Fig* 174 after removal of the pubic hair : showing the hypertrophy of the external genitalia, without development of the breasts.

Testicular atrophy or excision as a cause for undue fatness is best exemplified by eunuchs ; similar fat accumulation sometimes occurs in less degree as the result of atrophy after bilateral gonococcal orchitis or epididymitis ; it does not follow tuberculous destruction, for the patient then wastes instead. Palpation of the scrotum may indicate the diagnosis.

Ovarian insufficiency is probably a potent cause for undue stoutness in certain women, but it is difficult to prove this, because many of the patients suffer from hypothyroidism at the same time : there is a close inter-relationship between the thyroid gland and the ovaries. Only a small proportion of those cases in which both ovaries have been excised become obese ; but when the normal ovarian activities are beginning to abate, especially at and immediately after the menopause, it is common for women to become very stout. They develop at the same time peculiar nervous symptoms, and it is remarkable how easily both the latter and the obesity may be relieved by relatively small doses of thyroid extract ; such cases may be termed sufferers from hypothyroidism, even though they may not have the typical signs of complete myxœdema—increasing stoutness, loss of strength, broad features, increasing slowness of the intellect, broadening and thickening of the fingers and hands, malar flush, and falling out of hair and eyebrows. The best test of the diagnosis is the effect of administering carefully graduated doses of thyroid extract.

There are certain boys and girls—especially boys—who tend to become enormously fat long before they reach the age of puberty. The papers were full of a typical example of this malady a while ago—the Fat Boy of Peckham. This abnormal development of fat and size is in some cases associated with an affection of a suprarenal capsule or kidney—*hypernephroma*. The latter does not always cause this overgrowth, however, for in another type of patient the tumour leads merely to premature development of the pubic hair and external genitalia. *Figs.* 174 and 175 are from a girl, aged seven, who had had thick pubic hair since she was eighteen months old. The clitoris was enlarged, but there had been no menstruation. The diagnosis was confirmed post mortem, the congenital suprarenal tumour having produced secondary deposits in the lungs after seven years.

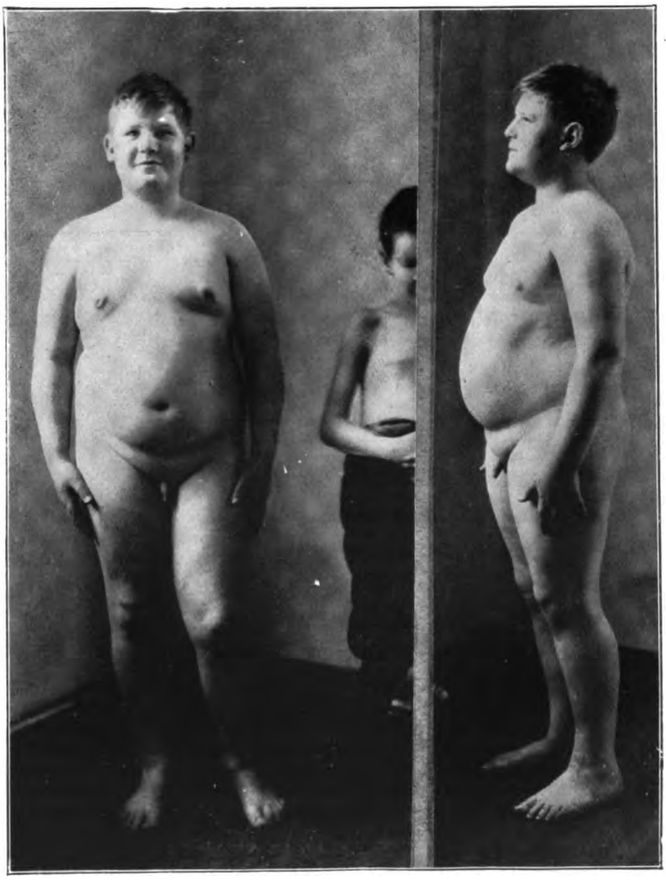


Fig. 176.—Case of dystrophia adiposogenitalis.

Recent observations seems to show that obesity may be due to *affections of the pituitary body* as well as to those of the suprarenal. We are told that when there is over-activity of the anterior lobe of the pituitary, acromegaly results (p. 237) ; and that when there is deficiency in either the anterior or the posterior lobe, the resultant error of metabolism affects not so much the bones as the soft parts ; *Fig.* 176 indicates the kind of patient that results. The diagnosis is suggested when, along with the rapidly increasing and clearly pathological fatness, there is persistence of the infantile type of intellect, voice, and genital organs, a voracious appetite, abundant stools, and a proneness to unusually long hours of sleep. It is not necessary that the intracranial lesion should be primarily in the pituitary body itself; a tumour at a distance from the latter can sometimes so interfere with it indirectly that the same kind of symptoms may result as if the pituitary gland was the chief focus of disease; this is probably the explanation of the fatness that is so often observed in cases of *cerebellar* or *cerebral tumour.*

Adiposis dolorosa, diffuse lipomatosis, and *Dercum's disease* all seem to be closely related. There are two types—the alcoholic and the congenital syphilitic; the former is the commoner, and occurs in older patients than does the other. Extreme fatness develops, but not quite universally; the abdominal wall, especially on either side of the umbilicus, the neck, shoulders, arms, forearms, thighs, and legs may become enormous, but the hands, feet, scalp, ears, nose, and forehead escape. The patient's muscular power, as tested by the dynamometer, is very small, sometimes not a tenth of the normal; and when any of the fat parts are taken hold of firmly, without any pinching or other procedure that would be unpleasant to an ordinary patient, some of these cases experience acute pain—the name adiposis dolorosa describing the two main symptoms of the malady. There are often mental symptoms at the same time; a patient of thirty may periodically imagine she is only eight, and behave and speak as though for the time being she were a child again; and so on in other cases, the types of mental symptoms being protean. Superficially these cases may simulate myxœdema, but a moment's observation will show that there is no affection of the hands and feet, which are just the parts to be first broadened and thickened by myxœdema, besides which thyroid treatment does not bring about material improvement. *Herbert French.*

OBSTIPATION.—(See CONSTIPATION, p. 121.)

OBSTRUCTON, INTESTINAL.—(See VOMITING, p. 767; and CONSTIPATION, p. 121, and consult Index at end.)

ŒDEMA, ASYMMETRICAL.—Œdema of one leg or arm, or of some local part of the head, face, neck, or trunk, may be due to any of the following causes:—

Congenital:—
- Constriction by amniotic bands
- Communications between arteries and veins

Acquired:—
- Blockage of veins:
  1. From within—non-infective thrombus, varicose veins, infective thrombus, e.g., white leg
  2. By pressure from without—by glands, tumours, aneurysms, etc.
- Blockage of lymphatics, e.g., in cellulitis, or other local inflammation; filariasis
- Artificial, by ligature
- Drug œdemas, such as those caused by—
  - Aspirin
  - Antitoxic sera
  - Iodides
  - Bromides
  - Mercurials; and any which cause urticarial lesions (p. 771)
- Angioneurotic œdema.

Congenital Causes are rare, but as a rule they are identified easily.

Acquired Causes.—The diagnosis may be obvious: For instance, there may be toothache with unilateral œdema of the face, or a scalp wound with boggy swelling all round it, or a well-marked *cellulitis,* with red streaks extending up the limb showing the course of acutely inflamed lymphatics. It is only rarely that a cellulitis presents any difficulty in recognition; namely, when the inflammation is not very acute, and when there is no obvious source of infection, such as an abraded toe or a suppurating wound of a finger. Cellulitis may then be confounded with gout; but the history, the presence of leucocytosis, and the absence of other gouty manifestations will indicate the real complaint. There may be considerable pyrexia in acute gout (*Fig.* 151, p. 339), so that the temperature chart does not serve to distinguish it from cellulitis.

Varicose veins are a frequent cause of asymmetrical œdema, especially in the leg, and if there is thrombosis as well, very marked swelling results. The thrombus, however, does not always lie in a superficial vein, and if it is in a deep one such as the popliteal, femoral, or iliac, the case may not be so clear. The thrombus in these cases is often due to septic infection, and the common source is sepsis in connection with the uterus following parturition—white leg; or it may arise in the course of a prolonged febrile illness such as typhoid fever, or in a case of cachexia resulting from malignant disease or other prolonged and debilitating malady, or from one of the blood diseases such as leukæmia.

When none of these causes is present it is necessary to examine carefully to ascertain whether there is any swelling pressing on and obstructing the veins, such as an aneurysm in the popliteal space or a mass of malignant glands; and not only must the whole limb be examined, but also the rectum, vagina, and lower part of the abdomen, and the neck and upper thorax in the case of the leg and arm respectively. For instance, there may be a tumour springing from some structure in the pelvis causing pressure on the iliac veins; and swelling of the arm might be caused by an aneurysm, subclavian or thoracic, or by a mediastinal new growth, in which case x-ray examination may be of material assistance in verifying the cause. The œdema due to venous obstruction pits readily on pressure, but where the lymphatics are blocked the œdema is much more solid; this may be an important diagnostic point.

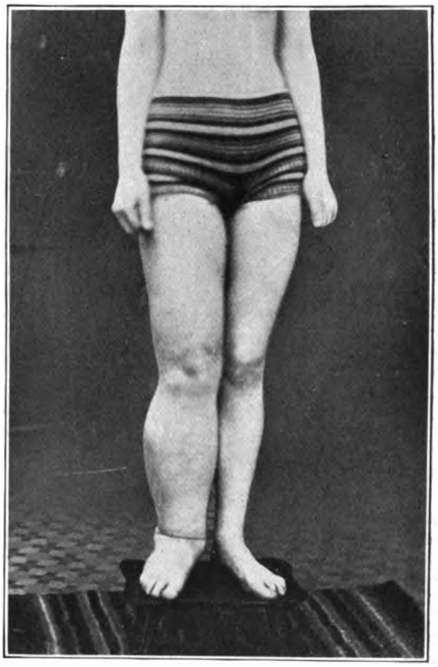


Fig. 177.—Milroy's or Meige's disease: A case of unilateral hereditary trophœdema of the leg. The condition had developed spontaneously in a girl who had never been out of England, and who suffered little inconvenience from the affection. She was 21 when the photograph was taken, and had had the swelling for years. Note that the enlargement of her right leg ceases abruptly at the groin.

Elephantiasis due to blockage of lymphatics by the parasite *filaria sanguinis hominis* is not common in England, though a pseudo-elephantiasis, due to long-standing lymphatic obstruction with resulting roughening, thickening, and fibrotic changes in the skin and underlying tissues, is not uncommon, and may result from long continuance of a tumour, or be associated with a badly-united fracture, or follow some operation in which the lymphatics have been removed, e.g. after amputation of the breast and axillary contents for carcinoma. Probably the most difficult group of all cases to diagnose is that in which there is a thrombus of one of the deep veins of the leg without any obvious disease, and in this event the diagnosis can only be arrived at by a process of exclusion. *Milroy's Disease* (*Fig.* 177) is diagnosed from the family history (see p. 414).

Ligature.—It sometimes happens that a patient, generally a female, presents herself with an œdema of a limb for which no explanation can be offered. It has to be borne in mind that there are some neurotic individuals who will tie a ligature round their limbs in order to simulate disease or to excite sympathy, and who have even gone so far as to suffer amputation. It is often extremely difficult to detect the fraud; but if the possibility be suspected, the nurse in charge must be instructed to keep watch, and at unexpected times to search the patient, when a handkerchief or a piece of string may be found constricting the limb. The fact that the upper limit of the œdema is sharply defined should awaken suspicion. It may be difficult to differentiate this from *angioneurotic œdema* (*Quincke's disease*), but the latter condition is, as a rule, transitory, and affects different parts of the body, e.g. the tongue, lips, eyelid, hands, etc., at different times (*Fig.* 178, p. 412); the fact that the patient has had previous attacks generally points to the diagnosis, and the malady often occurs spontaneously in several members of the same family.

George E. Gask.

ŒDEMA, SYMMETRICAL.—Owing to accidents of posture—such, for instance, as the patient sitting with one leg to the ground and the other supported upon a chair, or lying in bed turned well over to one side, and remaining in this asymmetrical position for a long time—it is possible for œdema which would really be symmetrical to appear asymmetrical. Allowing for this source of fallacy, however, the causes of symmetrical œdema are different from those of asymmetrical œdema (see above). One may subdivide cases into three main groups, namely : (1) *Those in which the œdema is universal ;* (2) *Those cases of œdema in which the swelling involves the face, neck, and arms, but not the legs or the lower half of the trunk ;* (3) *Those in which the œdema affects the legs, or the legs and lower half of the trunk, but not the arms, neck, or face.*

Œdema of the legs is by far the commonest type, and by far the most important point in the diagnosis is to decide as soon as possible whether this œdema is due to Bright's disease, heart failure, or to some other cause. The broad distinction into these groups is seldom difficult. The urine should be tested at once ; if albumin be present, microscopic examination for renal tube-casts is essential, their presence indicating renal mischief, their absence probably excluding it, unless the renal lesion is very acute, in which case there will be renal epithelial cells even if there are no tube-casts ; if there be no albumin in the urine, renal inflammation as a primary cause of œdema of the legs is unlikely.

It will be easy as a rule to decide whether there is failure of cardiac compensation or not ; if there is, the differentiation between the four main groups of causes of heart failure, namely, primary valvular, primary muscular, primary lung affections, and primary arterial or renal conditions, will be made upon the lines indicated upon p. 14.

Other causes for œdema of the legs will be suggested by other symptoms in the case or by the history, but they cannot be diagnosed with certainty until both renal inflammation and heart failure have been excluded. It seems worth while, however, to discuss in rather greater detail each of the main groups indicated above.

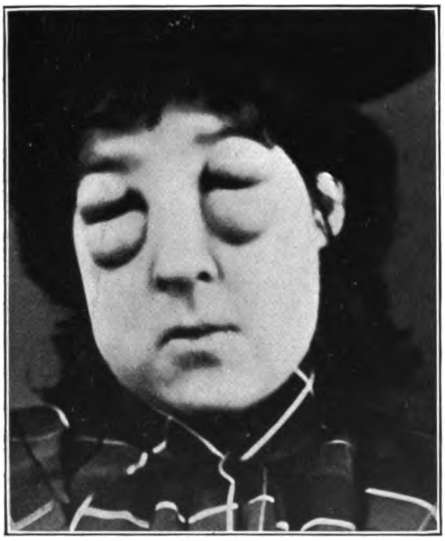


Fig. 178.—Angioneurotic œdema of the eyelids simulating acute nephritis.

Cases in which the Œdema is Universal.—When a patient has a tendency to universal symmetrical œdema, the great probability is that he is suffering from either *primary acute nephritis* or *acute nephritis superposed upon chronic nephritis* ; the diagnosis is indicated by the occurrence of albumin with tube-casts. The degree of œdema exhibited in different regions varies partly by reason of the looseness of the subcutaneous tissues in different places, and partly by means of the effects of gravity. Other things being equal, the œdema shows most in the legs, lumbar region (lumbar cushion), penis, scrotum, labia, eyelids, and face, though careful examination may show that there is some degree of œdema in every tissue from scalp to toes ; it is due to the influence of gravity that when the patient is up and about the œdema is most marked in the legs ; is very marked in the lumbar cushion and the genital organs when the patient sits propped up in bed ; and is most prominent in the eyelids when the patient has been lying horizontally, as during sleep.

Other causes for universal œdema are rare, but it may sometimes be due to a universal condition of *angioneurotic œdema* (*Fig.* 178), though this is much more often asymmetrical ; or to overloading of the tissues with fluid—for instance, as the result of excessive transfusion or infusion, or in patients who have been swilling beer day after day until their bodies have become sodden. Such cases present an appearance highly suggestive of acute nephritis, but the absence of albumin from the urine, the history of excessive drinking over long periods, and the complete recovery when the drinking is stopped, point to the diagnosis. Generalized œdema, usually not of extreme degree, is apt to be complained of by some anæmic girls, generally between 15 and 20 years of age, often in association with gastric symptoms, especially persistent vomiting after food, and with constipation. These cases have no name; they are chiefly of importance in that they are very apt to be diagnosed as nephritis, although there is no albuminuria as a rule, and they get well by themselves. The œdema is doubtless toxic, but whether the causal toxin is intestinal, or derived from some other source, no one knows. The same applies to certain cases of acute universal œdema in infancy or childhood, simulating acute nephritis, but differing from the latter in that there is no albuminuria and spontaneous recovery occurs. Both types differ from true angioneurotic œdema in that the condition is not familial like the latter (p. 411). Similar universal œdema, perhaps toxic in origin, but unaccompanied by any evidence of nephritis, follows severe *gastro-enteritis* in children, even when no saline infusion has been resorted to.

Certain poisons may produce universal œdema, though rarely; *iodide of potassium* has been known to do so to a mild degree; one of the effects of *snake-bite* also is to produce universal œdema with or without albuminuria, though as a rule the part originally bitten is very much more swollen than are the other portions of the body.

Aspirin affects certain individuals in a curious way, producing urticarial wheals and universal swelling, transient as a rule, or lasting little more than twenty-four hours; though sometimes so severe that the whole face is swelled up and bloated to such an extent that the patient is for the time being unrecognizable. The symptoms appear to depend here upon personal idiosyncrasy to the drug.

Only in very rare cases does *heart failure* produce œdema of the hands and arms as well as of the legs, and when it does so the patient usually has been ill some time, the diagnosis has already been made, and the end is not far off.

Œdema of the Face, Neck, and Arms, but not of the Legs or Lower Half of the Trunk, is nearly always due to obstruction to the superior vena cava or to the main branches which go to form this, and the commonest cause of this obstruction are *thoracic aneurysm, mediastinal new growths*, or *gumma, chronic mediastinal fibrosis*, and *thrombosis* spreading to the main trunk from, for instance, an axillary vein infected from a whitlow or from other sources of phlebitis. When the swelling comes on acutely, as it may in any of the above conditions, acute Bright's disease may be simulated on account of the extreme puffiness of the eyes; but further examination will show a remarkable limitation of the œdema to the head and upper limbs, whilst the urine will probably not contain albumin. If the obstruction to the superior vena cava persist, there will be evidence of collateral circulation in the form of varicose veins upon the chest wall (see VEINS, VARICOSE THORACIC, p. 750).

It only remains to add that, instead of being asymmetrical, inflammatory lesions may sometimes produce almost symmetrical œdema of the face or neck, in which connection one may mention *erysipelas, cellulitis, anthrax, angini Ludovici*, the differential diagnosis of which is based upon the history, the constitutional symptoms, the local appearances of the inflammation, and the results of bacteriological examination.

Similar symmetrical swelling may be produced in the hands or arms either by *angioneurotic œdema* (*Fig.* 178), or by allied vasomotor neuroses, such as *Raynaud's disease*. Swelling of the eyes and face suggestive of œdema may sometimes be due to bouts of *crying*, prolonged attacks of *coughing*, as for instance in whooping-cough, or as the result of catarrh due to a *common cold, measles*, or to the effect of such remedies as *potassium iodide* or *arsenic*.

Œdema of the Legs and Lower Part of the Trunk, without any of the Neck or Face, is suggestive of heart failure or of nephritis, and the main points that arise in the differential diagnosis have been discussed above. If both these main groups of causes can be excluded, however, it is important to remember how often the legs may swell as the result of poverty of the blood in any condition of anæmia. This is perhaps seen best of all in cases of *chlorosis*, for patients suffering from the severer types of anæmia, such as *pernicious anœmia, lymphatic* or *splenomedullary leukœmia, Hodgkin's disease, splenic anœmia, pseudo-leukœmia infantum*, are less continuously up and about than are many cases of chlorosis. The same applies to the severe *anœmia which follows loss of blood* from hæmoptysis, hæmatemesis, post-partum and other hæmorrhages; or to the less acute anæmias that result from *parasitic infections* such as *Bothriocephalus latus* or *Ankylostomum duodenale*, or the effects of certain drugs; or to *cachectic conditions* such as result from carcinoma, sarcoma, syphilis, tuberculosis, starvation, malaria and various other tropical infections. The differential diagnosis of these conditions will seldom depend upon the presence of œdema alone, and each of the maladies will be found discussed under the heading of some other symptom.

Obstruction to the inferior vena cava may lead to extreme œdema of the legs; if due to phlebitis, the clotting of the inferior vena cava itself is nearly always preceded by that of the veins of one leg, so that even when the final result is symmetrical, the history nearly always points to it having begun asymmetrically. When the inferior vena cava is obstructed by new growth or by the pressure of ascitic fluid, the diagnosis will depend upon the discovery of some abnormal mass, or upon the interpretation of the cause of the ASCITES (p. 43). Much difficulty sometimes arises, as in a case mentioned on p. 7.

The influence of the vasomotor nerves in controlling the balance of lymph production and lymph absorption in the legs is sometimes interfered with. One sees a good example of this in the œdema which develops in the lower extremities in *convalescent patients* when, having been long in the horizontal position from any cause, they first begin to walk about; it is probable that a perfectly normal person kept at rest in bed for three months would suffer from œdema of the legs in varying degree for some days or weeks after first beginning to use his limbs, and the tendency is still more marked in those who have been laid up by gastric or duodenal ulcers, typhoid fever, fractured femur, and so on. It may at first arouse a suspicion of some kidney lesion, though the absence of albumin, and the way in which the œdema disappears spontaneously in time, especially under the influence of massage, indicate the diagnosis when the history of long confinement to a horizontal position is known. Diseased conditions of the vasomotor system may produce even more marked œdema, as seen in *elderly people*, in some cases of *Raynaud's disease*; in *angioneurotic œdema*; in association with *peripheral neuritis*, especially in the tropical variety called *beri-beri*, an epidemic febrile illness generally seen in this country only in seaport towns as the result of an outbreak amongst seamen on board ships in which the diet has consisted largely of decorticated rice.

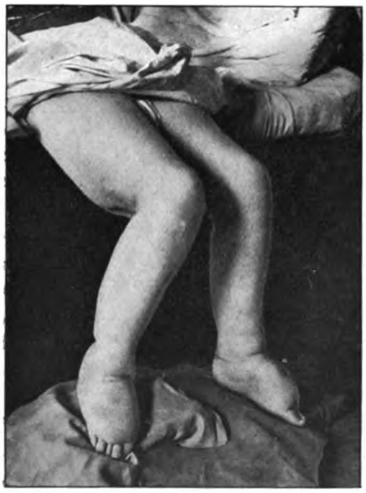


Fig. 179.—Bilateral hereditary trophœdema of the legs in a girl of 21, twelve other members of the family being affected by the complaint also. She had never been out of England. There was no abnormality above Poupart's ligament. Milroy's or Meige's disease. (For a full account see Hope and French, *Quart. Jour. of Med.*, vol. i. No. 3, p. 312.)

There is a peculiar hereditary disease in which œdema of the lower extremities, occurring in many members of a family (*Fig.* 179), may be a prominent feature; in the early stages this œdema is asymmetrical, affecting one leg before the other, but sooner or later both legs may become involved, until, if the family and personal history were not known, the œdema of Bright's disease might be suspected. The affection is known as *Milroy's disease*, *Meige's disease*, or *hereditary trophœdema*. The sudden demarcation between the swollen and the non-swollen parts at the level of a joint—ankle, knee, or hip—is characteristic. There is sometimes a history of periodic acute attacks of pyrexia and of gastric disorder associated with an increase in the swelling, not altogether unlike those occurring in angioneurotic œdema. The swelling may cease at the ankles in the early stages; when a subsequent spread occurs, it may reach almost suddenly up to the knees, ceasing there for a variable number of years, until ultimately it spreads to the groins, above which it seldom extends. The diagnosis is easy when the family history is obtainable.

Myxœdema is a condition in which the swelling of the legs may simulate actual œdema very closely, and indeed in not a few cases the subcutaneous tissues of the feet and legs do pit to a certain extent on pressure. When there is actual œdema as well as myxœdema, considerable doubt as to whether there may not be a cardiac or other factor, as well as thyroid insufficiency, will arise. The urine often contains albumin, moreover, though generally without tube casts; the patient is nearly always a woman of middle age (*Fig.* 104, p. 234), who has recently begun to get much stouter, and at the same time less active both mentally and physically. The diagnosis of myxœdema will be confirmed if the untoward symptoms and the abnormal state of the subcutaneous tissues disappear under the influence of thyroid medication.

It is not easy to include all the possible causes of œdema in a classified list, but the following include those which have been discussed above :—

1. **Universal Œdema.**

- Primary acute nephritis
- Acute nephritis as an exacerbation of chronic nephritis
- Angioneurosis
- Excessive transfusion or infusion
- Soddening from beer drinking
- Toxins (? intestinal) in girls and children
- Iodide of potassium
- Snake-bite
- Heart failure.

2. **Œdema of Face, Neck, and Arms, but not of Legs.**

- Obstruction to the superior vena cava by :
  - Thoracic aneurysm
  - Mediastinal new growth
  - Mediastinal gumma
  - Mediastinal fibrosis
  - Thrombosis
- Erysipelas
- Cellulitis
- Anthrax
- Angina Ludovici
- Raynaud's disease
- Angioneurosis
- Crying
- Coughing
- Measles
- Common cold
- Ptomaine poisoning, shell-fish variety
- Arsenic
- Aspirin.

3. **Œdema of the Legs, without any of the Neck or Face.**

- Heart failure secondary to :
  - Valvular disease
  - Myocardial affections
  - Chronic lung affections
  - Renal or arterial affections
- Bright's disease
- Chlorosis
- Pernicious anæmia
- Lymphatic leukæmia
- Splenomedullary leukæmia
- Hodgkin's disease
- Splenic anæmia
- Pseudo-leukæmia infantum
- Anæmia following excessive blood loss
- Parasitic affections, especially
  - Bothriocephalus latus
  - Ankylostomum duodenale
- Cachectic states due to :
  - Carcinoma
  - Sarcoma
  - Syphilis
  - Tuberculosis
  - Starvation
  - Malaria
  - Tropical affections
- Inferior vena cava obstruction by :
  - Thrombosis
  - New growths
  - Ascites
- Convalescence
- Old age
- Raynaud's disease
- Angioneurosis
- Beri-beri
- Milroy's disease (hereditary trophœdema)
- Myxœdema.

Herbert French.

OLIGOCYTHÆMIA.—(See ANÆMIA, p. 20.)

OLIGURIA.—(See ANURIA, p. 39.)

OPHTHALMOPLEGIA.—(See STRABISMUS, p. 649 ; PUPIL, ABNORMALITIES OF THE, p. 551 ; and DIPLOPIA, p. 174.)

OPHTHALMOSCOPIC APPEARANCES, NOTES ON.—(*Plates XIX* and *XX.*)

Fig. a.—**A Physiological Cup** may vary in size, but usually occupies the centre of the disc. The retinal vessels dip over the edge, which is usually steeper on the nasal side, the temporal slope being more gradual. At the bottom of the cup is seen the lamina cribrosa, which is mottled by the openings through which the retinal nerve fibres pass.

A physiological cup is distinguished from that caused by glaucoma (*Fig. v*) by the fact that it occupies only the centre and not the whole of the disc.

Fig. b.—**Congenital Crescents** are common, and usually situated at the lower part of the disc, in contrast to myopic crescents (*Figs. h* and *i*), which are seen on the outer side. They are probably due to an uneven distribution of connective tissue in the lamina cribrosa, and are often associated with hypermetropia.

Fig. c.—**Pigmented Crescent in Disc Margin.**—The disc margin is always more or less pigmented, the amount varying from a small crescent to a complete ring. The pigment has no pathological significance.

Fig. d.—**Coloboma of the Choroid** is a congenital deficiency, and it may be recognized by its situation below the disc, the small amount of pigment at the edge of the white area, and the presence of healthy retinal vessels on its surface. It may be associated with other congenital abnormalities, such as coloboma of the iris, optic disc, or lens.

Figs. e, f.—**Opaque Nerve Fibres** exist normally in the retinæ of some mammals, e.g., the rabbit. The condition is due to the persistence of the medullary nerve sheath of the retinal fibres, the sheath being lost usually at the passage of the nerve fibre through the lamina cribrosa. The condition may be recognized by the brilliant white colour of the nerve fibres, the striated appearance of the white patch, and the fact that the retinal vessels are more or less embedded among the nerve fibres.

Fig. g.—**Advanced Syphilitic Choroiditis.**—In advanced choroiditis the inflammatory process has ended in the total destruction of the choroid in patches, which in some places have joined to disclose large bare areas of sclerotic. There are large masses of pigment, usually surrounding the white areas, the pigment being chiefly derived from the choroid. The retinal pigment is also increased in the great majority of cases, and vision is rendered extremely defective. Macular choroiditis is degenerative in origin, and does not usually appear till middle life. It probably commences in the form of macular hæmorrhages, which lead to destruction and fibrosis of the retina.

Figs. h, i.—**The Myopic Crescent** is usually found on the outer side of the disc, and may vary in size and extent from a thin crescent to a large atrophic area surrounding the whole disc (posterior staphyloma). Usually, the size of the crescent varies with the amount of the myopia and increases with age.

Figs. k, l.—**Recent Optic Neuritis** is characterized by the swelling of the disc and the blurring of its outline by retinal œdema. The retina is greyish and striated in appearance, owing to œdema between the retinal nerve fibres, and the veins are extremely dilated and tortuous. Flame-shaped hæmorrhages are also seen on the disc and in the surrounding retina, and numerous small retinal vessels on the disc, usually invisible, become dilated and apparent. In the later stages of the neuritis the hæmorrhages may disappear, and the whole disc become greyer and paler, the condition ultimately terminating in post-neuritic atrophy (*Fig. l*). The outline of the disc is entirely lost, and in severe cases the disc may be so swollen as to resemble a small mushroom in shape. Radiating lines of white patches may also be seen in the macular region, resembling albuminuric retinitis (*Figs. p, q, r.*).

Figs. m, n.—**Primary Optic Atrophy** (*Fig. n*) is characterized by the pallor of the disc, white or bluish-white, sharply defined lamina cribrosa, well-marked edge, and retinal vessels of normal size. In *post-neuritic atrophy* (*Fig. m*) the disc is covered with fibrous tissue, which fills up the physiological cup ; the colour is greyish-white, the retinal vessels are thin and tortuous, and the edge of the disc is irregular. In some cases of old post-neuritic atrophy or fibrosis, following slight optic neuritis, it may be impossible to distinguish the condition from primary atrophy.

Fig. o.—**Thrombosis of the Central Retinal Vein.**—In thrombosis of the central retinal vein the disc is extremely swollen and œdematous, the edge being indistinct and blurred. All the retinal veins are enormously dilated and tortuous, and the fundus is covered with flame-shaped and petechial hæmorrhages. The œdema of the retina from the obstruction of the venous circulation may be so great that the vein may occasionally be hidden entirely.

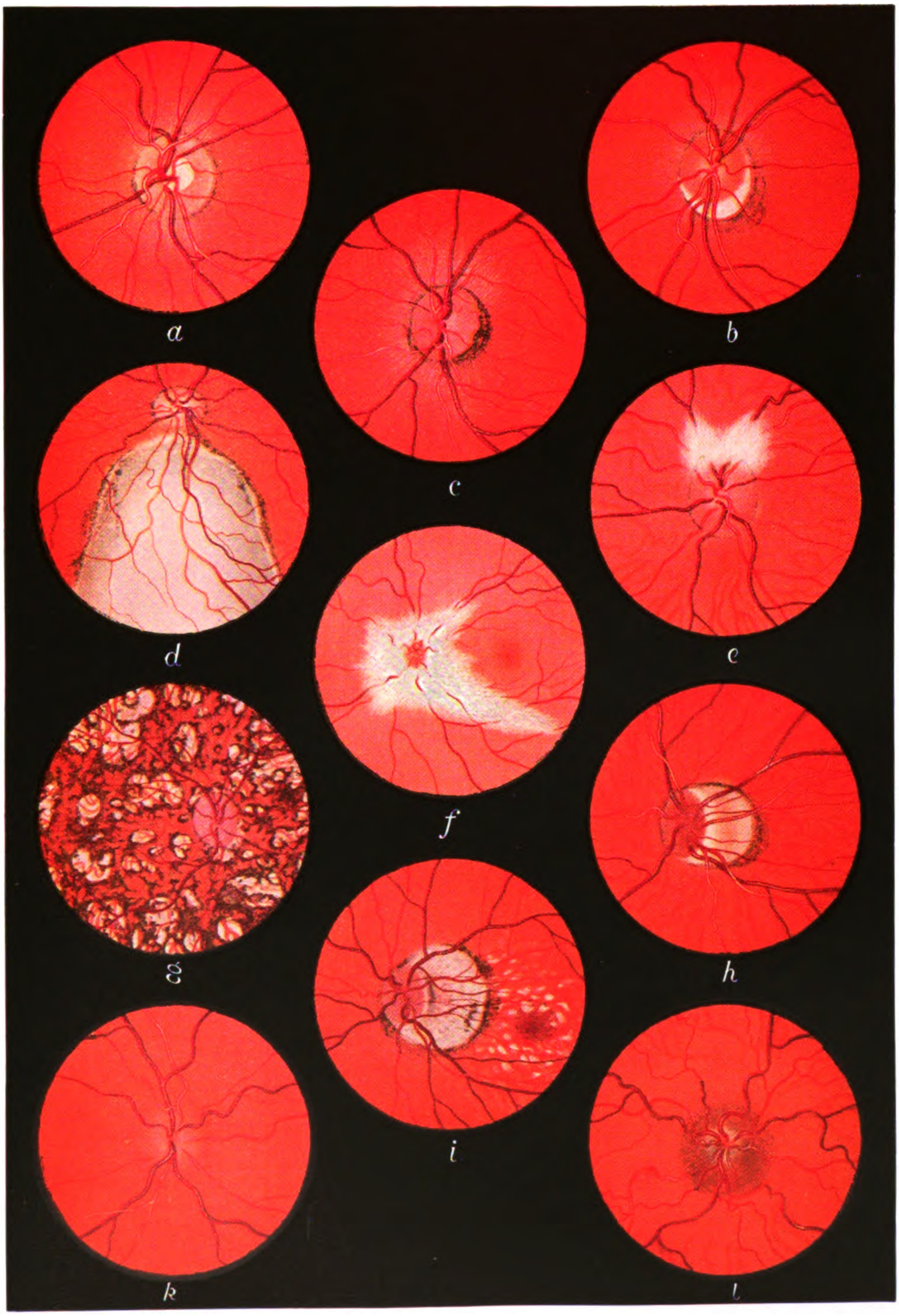
Figs. p, q, r.—**Albuminuric Retinitis** is characterized by the presence of flame-shaped hæmorrhages in the nerve-fibre layer of the retina, and white patches. The white patches are of two kinds. Those seen in the early stages of the disease are ill defined and woolly, scattered about the macular region in an irregular manner. These are due to exudate in the nerve-fibre layer of the retina. In the later stages, smaller white patches may be seen usually arranged in radiating lines from the macula, which are well defined, and glistening or chalky white.

Fig. s.—**Embolism of the Central Retinal Artery.**—In embolism of the central retinal artery the retina is generally pale grey or white, owing to the anæmia consequent on the obstruction of the artery. The macula itself being adherent to the choroid does not share in the general pallor, and appears as a bright cherry-red spot in contrast. The retinal arteries are extremely small, being only fine white threads in places, and the veins may be nearly empty. The optic disc is white, blurred, and indistinct.

Fig. t.—**Detachment of the Retina.**—The detached portion of the retina is silvery-grey in colour, and raised above the surrounding fundus. In cases due to serous exudate, the detached part of the retina is transparent, arranged in billowy folds, and may float

PLATE XIX

OPHTHALMOSCOPIC APPEARANCES



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A. W Head, del.

about on movement of the eye. When the detachment is due to growth, the retina is usually smooth and opaque. The retinal vessels are small, very tortuous, and dark in colour.

Fig. v.—**Glaucomatous Discs.**—The excavation of the optic disc may be distinguished from the physiological cup by the fact that it affects the whole of the disc, the edge often being surrounded by an atrophic ring. The retinal vessels bend sharply over the edge, and may disappear from view behind the overhanging margin of the disc, reappearing on the bottom of the cup. The lamina cribrosa is well marked, and the disc is white and atrophic.

Fig. w.—**Tubercles in the Choroid** are seen as ill-defined circular masses varying in size from a pin's head to masses nearly the size of the optic disc. They are usually associated with miliary tuberculosis, grow rapidly, but rarely attain any great size owing to the death of the patient. It is stated that they occur most commonly in the neighbourhood of the disc, but this is due to the fact that only the posterior portion of the fundus is visible with the ophthalmoscope. Post mortem they are found all over the choroid.

Fig. x.—**Hypermetropic Astigmatism.**—In hypermetropic astigmatism the disc is often oval and ill defined. The physiological cup is absent, the disc red, and the margin ill defined. The vessels may be tortuous though not dilated, and unless the error of refraction is observed, the condition may be mistaken for optic neuritis.

Herbert L. Eason.

OPISTHOTONOS is a rare but characteristic condition, in which the muscles of the neck, back, and legs are contracted rigidly in such a way that the body is over-extended in the form of an arch, supported by the occiput above and by the heels below. This position may be maintained ; more often it is assumed periodically, with partial or complete relaxations between the tetanic seizures. Its chief cause is *tetanus*, but it may also be due to *strychnine poisoning*, *spinal meningitis*, *uræmia*, and *hysteria*.

Tetanus.—The history will often point to the correct diagnosis. If there has been a punctured wound recently, and if stiffness of the neck muscles and of the lower jaw (lockjaw or trismus) (p. 729) has set in, to be followed within a day or so by generalized rigidity, with severe paroxysmal exacerbations, the opisthotonos is almost certainly due to tetanus. The fixed smile—risus sardonicus—is common to tetanus and to strychnine poisoning. An attempt will be made to obtain the drumstick bacilli (*Plate XXVIII, Fig. T*, p. 614) from the suspected wound, very often without success. In some cases there will be no obvious wound or contusion, but although the source of contagion will then be obscure, the early lockjaw and the course of the disease will point to tetanus.

Strychnine poisoning does not give rise to lockjaw, and the paroxysms of opisthotonos are separated by intervals of more complete relaxation than is the case in tetanus ; there may be evidence of the source of the poisoning, either accidental, suicidal, or homicidal, in the form of a bottle, a hypodermic syringe and needle, a packet of vermin-destroyer, or something of that kind. In some cases the diagnosis can only be arrived at by analysis, either of the gastro-intestinal contents, or of the viscera post mortem.

Spinal meningitis seldom causes difficulty in the diagnosis, for it is generally part of acute cerebrospinal meningitis, of which the general symptoms and pyrexia will have existed some days, if not a week or more, before opisthotonos is likely to occur. Optic neuritis may be found, and in some cases the bacteriological and cytological results of lumbar puncture, especially the discovery of the meningococcus, may clinch the diagnosis.

Uræmic convulsions are associated with complete coma, whereas in tetanus and in strychnine poisoning consciousness is retained perfectly ; the convulsions are epileptiform rather than tetanic ; there is no persistent lockjaw ; and the urine will nearly always contain albumin and renal tube-casts.

Hysteria sometimes takes a form that may for the moment be difficult to distinguish from tetanus or from strychnine poisoning. Unlike malingering, hysterical contractions that are even violent enough to cause opisthotonos do not always make the patient perspire, nor do they lead to fatigue in the way that similar voluntary efforts certainly would. The diagnosis of hysteria is generally arrived at by watching the case. Persistent lockjaw may be present, as in tetanus ; but whereas in strychnine poisoning and in tetanus there is a great similarity between one exacerbation and the next, hysterical convulsions are apt to be polymorphous ; the more the writhing and the change of attitude and position, the less likely is the attack to be organic. The mind remains perfectly clear in tetanus and strychnine poisoning, though its outward expression may be prevented by the muscular paroxysms; in hysteria, the mental attitude is in one way or another abnormal for the time being. In arriving at a diagnosis it may be of great assistance to know full details of the patient's previous history, for there may have been similar hysterical outbursts on former occasions.

Herbert French.

OPTIC ATROPHY.—(See Ophthalmoscopic Appearances, Notes on, p. 415.)

OPTIC NEURITIS.—(See Ophthalmoscopic Appearances, Notes on, p. 415.)

ORTHOPNŒA, or the inability to breathe unless propped above the horizontal position, may be due to many causes, which may be grouped as follows:—

Failure of the Right Side of the Heart:—

Secondary to valvular disease of the left side of the heart:

Mitral stenosis
Mitral regurgitation
Mitral stenosis and regurgitation
Aortic disease with secondary mitral regurgitation.

Secondary to affections of the heart muscle:

Fatty heart
Fibroid heart
Adherent pericardium
Some cases of pericarditis
Primary alcoholic heart.

Secondary to high systemic blood-pressure:

Arteriosclerosis
Granular kidney.

Secondary to lung affections:

Emphysema
Chronic bronchitis
Fibroid lung
Pneumothorax.

Secondary to the effects of chronic poisoning:
Especially from tobacco smoking.

Obstruction to the Larynx or Trachea:—

Acute œdema of the larynx
Acute abductor paralysis
Post-pharyngeal abscess
Laryngeal diphtheria
Laryngismus stridulus
Catarrhal laryngitis, especially at the onset of certain cases of measles
Acute pneumococcal or streptococcal laryngitis
Œdema due to potassium iodide
Laryngeal crises of tabes dorsalis
Foreign body
Enlarged thyroid gland
Enlarged thymus gland
Aortic aneurysm
Mediastinal new growth
Malignant glands in the neck
Lymphadenomatous glands in neck
Irruption of a caseous gland into the trachea
Œsophageal tumour.

Acute Obstruction of the Bronchi and Bronchioles:—

Acute bronchitis
Acute capillary bronchitis
Some cases of acute bronchopneumonia
Asthma
Whooping-cough.

Mediastinal Masses:—

Aneurysm
Huge heart
New growth
Hydrothorax.

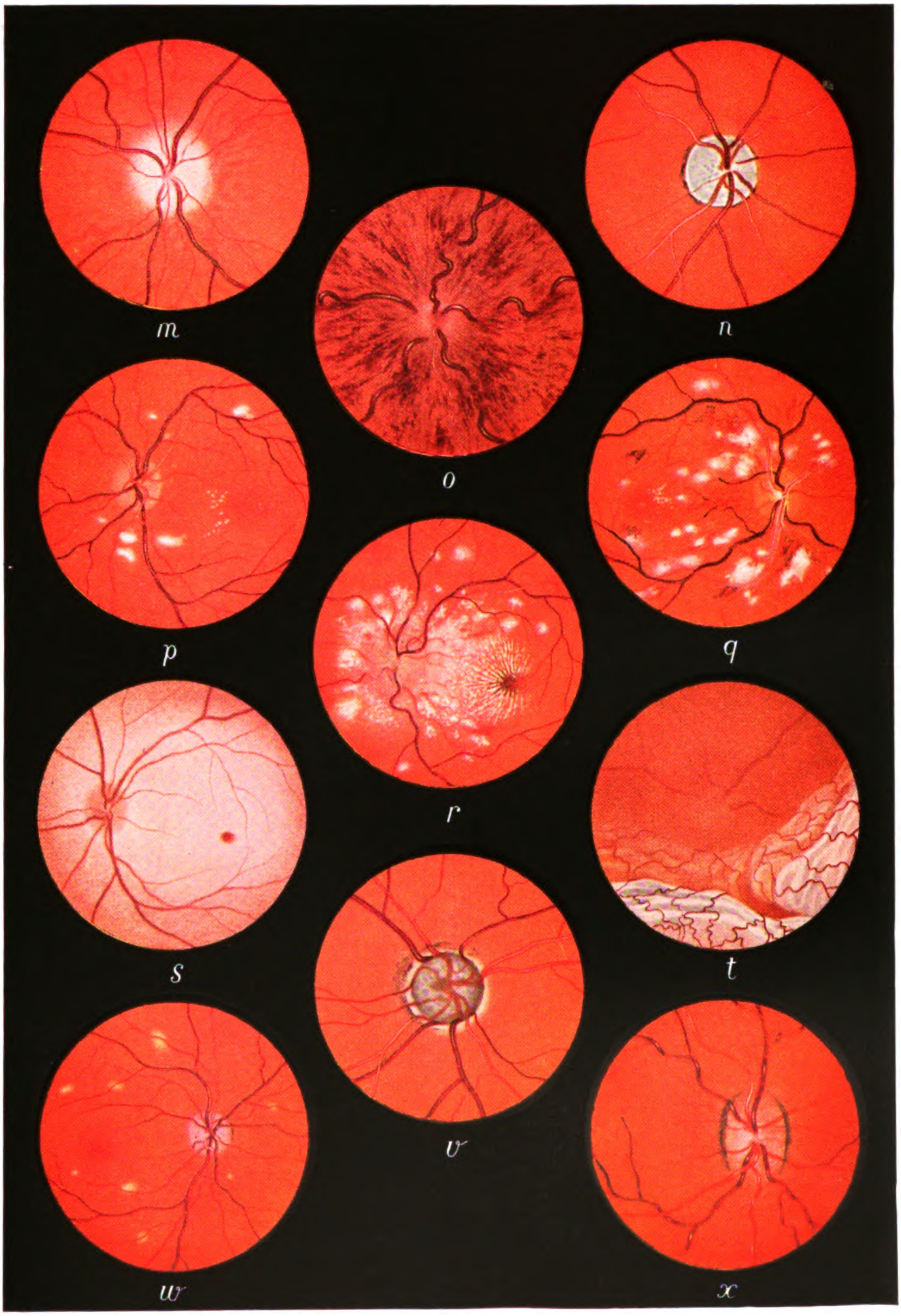
Enormous Distention of the Abdomen by—

Ascites
Tympanites
Neurosis or hysteria
Solid or cystic tumours.

By far the commonest cause of orthopnœa is *heart failure* of some kind or another, and the differential diagnosis of the variety of heart failure, and whether it is due to valvular disease, to myocardial degeneration, to arteriosclerosis, to granular kidney, or to the effect of difficulties in the pulmonary circulation from bronchitis and emphysema or fibroid lung, has to be decided upon the various grounds that are discussed on p. 14.

PLATE XX

OPHTHALMOSCOPIC APPEARANCES



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When orthopnœa is due to *obstruction to the larynx* or *trachea*, the fact is generally obvious on account of other symptoms, such as stridor, up-and-down movements of the larynx itself, sucking in above and below the clavicles and of the lower intercostal spaces, the main difficulty in some of these patients being to decide whether the obstruction is sufficiently near the larynx to be relieved by tracheotomy, or whether it is due to mischief lower down in the trachea, bronchi, or bronchioles. The nearer the obstruction is to the larynx the greater will be the spasmodic up-and-down movements of the thyroid cartilage, and the stridor. If the evidence is that the obstruction is in the larynx itself, and if the orthopnœa and difficulty with respiration are extreme, the probability is that tracheotomy will be resorted to as an urgency measure, the precise diagnosis being determined later. The history, or a local examination, would serve to diagnose or exclude acute *abductor paralysis*, *post-pharyngeal abscess*, *foreign body*, *enlarged thyroid gland*, *malignant glands* in the neck, *lymphadenomatous* glands in the neck. *Enlargement of the thymus gland* can seldom be more than conjectured, or diagnosed by a process of exclusion, unless there is definite dullness behind the upper part of the sternum in a child under ten years of age, together perhaps with an *x*-ray shadow of the gland (***Fig.*** 180). Aortic *aneurysm* or *mediastinal new growth* obstructing the trachea will generally have given rise to other characteristic symptoms at the same time; particularly in the case of mediastinal new growth to obstruction of the innominate veins or the superior vena cava, with varicose distention in the superficial thoracic veins by way of collateral circulation (***Fig.*** 99, p. 208) The *x*-rays may be useful in confirming the diagnosis (***Fig.*** 100, p. 209).

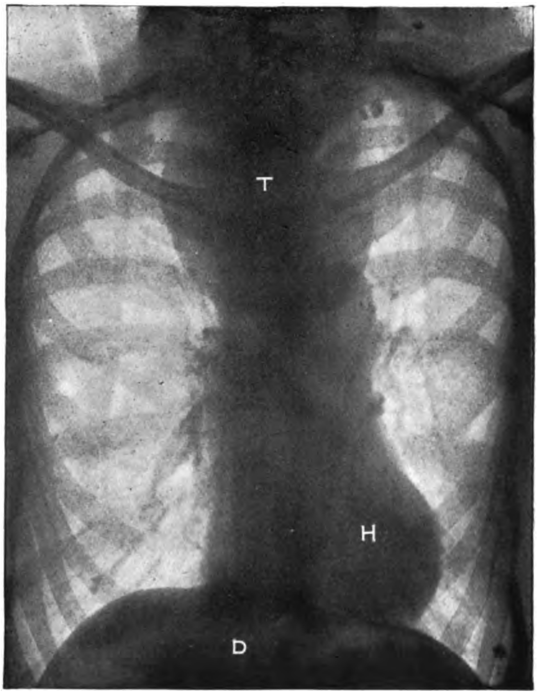


Fig. 180.—Skiagram showing an enlarged thymus gland. No thoracic symptoms. The large thymus was discovered in the course of a routine *x*-ray examination on account of dyspepsia in a female, aged 48.

T, Shadow of the large thymus. H, Shadow of the heart. D, Shadow of the diaphragm.

(*Skiagram by Dr. C. Thurstan Holland.*)

In a great many cases, particularly in children, none of the above will be the least likely, and if foreign body and post-pharyngeal abscess have been excluded by digital examination, the first suspicion will be that the patient is suffering from *laryngeal diphtheria*. This may be confirmed by the presence of a small quantity of membrane on the pharynx, the uvula, or elsewhere, though quite commonly when laryngeal diphtheria is extensive there is no obvious exudate upon any of the visible parts at the back of the mouth. The existence of cases of diphtheria in the same house or in the neighbourhood may point to the diagnosis; but in every case swabbings should be obtained from as far back in the throat as possible, and examined for Klebs-Loeffler bacilli, both in direct films and by culture. Until laryngeal diphtheria can be excluded by the absence of Klebs-Loeffler bacilli—and a single negative result does not necessarily exclude the disease—the nature of the case will probably remain in doubt. *Acute œdema of the larynx* is nearly always due to some microbial infection, and therefore in a sense it includes acute pneumococcal or streptococcal laryngitis, the diagnosis of which depends upon bacteriological cultivations from swabbings from the throat. Œdema may also be due to similar infection of ulcerated places in the throat developing in the course of *tuberculous*, *syphilitic*, *malignant*, *lupoid*, *traumatic*, or *post-typhoidal laryngeal ulceration*. The previous history, with the results of examination of the lungs, larynx, and sputum, will indicate the diagnosis of these various conditions.

The *laryngeal crises of tabes dorsalis* are exceedingly rare; they might be suggested if the patient were known to have no knee-jerks and Argyll Robertson pupils, but even then there might be doubt as to whether they were really crises and not the result of syphilitic ulceration, or due to the administration of potassium iodide in these cases. Acute œdema of the larynx is sometimes spoken of as one of the complications of *acute Bright's disease*, but it is very rare in this malady, rarer than acute œdema of the lungs; it is usually a terminal factor, the diagnosis of nephritis having been made previously on account of general œdema and albuminuria with tube-casts. *Laryngismus stridulus* is a dangerous diagnosis to make, for many cases thought to be this are really examples of diphtheria: if laryngismus stridulus does occur, it is to be expected in rickety children who show a tendency to spasmodic muscular contractions in other parts besides the larynx, such for instance as convulsions from slight causes, or the carpo-pedal contractions of infantile tetany ('spasmophilia'). It is supposed that similar spasmodic contractions of the muscles of the larynx produce paroxysms of laryngeal obstruction with acute dyspnœa, orthopnœa, and cyanosis; but no such cases should be diagnosed as simply neuromuscular until every precaution has been taken to exclude all other causes of laryngeal obstruction, especially diphtheria. Now and then one meets with a case in which an apparently healthy child is seized suddenly with acute dyspnœa, cyanosis, orthopnœa, and general respiratory distress, without any signs of laryngeal obstruction, the result of *irruption of a caseous bronchial* or *mediastinal gland* into the lower part of the trachea or a main bronchus. The symptoms are precisely such as one would expect if the patient had suddenly inhaled a foreign body of some size, and if one can be quite sure that no such foreign body has been inhaled, the correct diagnosis may sometimes be guessed at. It would be confirmed if, as occasionally happens, a sudden effort of coughing leads to the caseous or cretaceous mass being expectorated.

The difficulty of being certain whether, in a given case of severe respiratory distress with evidence of obstruction, the mischief lies in the larynx or in the lungs, is sometimes considerable; in either case there may be marked cyanosis, orthopnœa, dyspnœa, sucking in above and below the clavicles and of the lower intercostal spaces: the most important point to note is whether the larynx itself remains stationary as when the mischief is in the lungs, or whether it moves up and down with the respiratory movements as it does when the trouble is in the larynx. Very often both the lungs and the larynx are involved, and it may then be very difficult to decide which is the more so, and consequently whether tracheotomy is indicated or not. The chief point on which to lay stress, besides the movements of the larynx, is the result of a physical examination of the chest for evidence of *acute bronchitis* or of *bronchopneumonia*.

True *asthma* is a spasmodic variety of dyspnœa, the diagnosis of which, and the difficulty of distinguishing between asthma complicated by bronchitis and bronchitis simulating asthma, are discussed elsewhere (p. 535).

Whooping-cough is seldom difficult to diagnose, for even when the patient himself does not exhibit the typical whoop, he will be affected by a severe paroxysmal cough, possibly leading to vomiting, and at the same time relatives or friends may be affected by cough which gives rise to the typical whoop.

Mediastinal masses, such as aneurysm, a huge heart, new growth, hydrothorax with marked displacement of the heart, and *enormous distention of the abdomen* by ascites, tympanites, or by large tumours, will generally have been diagnosed before they reach the stage of producing orthopnœa. The chief reason why a very large heart or a thoracic aneurysm may produce orthopnœa, even when there are no signs of failure of the cardiac compensation, is that when a patient sits up there is a greater distance between the sternum and the vertebræ than when he lies back. The cause for the orthopnœa is thus mechanical, the patient sitting up to allow a bigger space for the accommodation of the abnormal mass; hence in some of these cases he may be able to walk about and see to his business without distress during the daytime, and yet be unable to lie down at night. The cause of the orthopnœa associated in this way with a huge heart is quite different from that in which there is failure of the right side, the former being a mechanical means of giving a big mass more room, whilst the latter is due to the need of maximum assistance from the respiratory blood-pump. *Herbert French.*

OTORRHŒA.—Discharge from the ear may result from a variety of causes, some trivial, others serious. The skin of the normal external auditory meatus contains numerous ceruminous and sebaceous glands, the secretion of which, known as cerumen or wax, may lead to trouble by forming a hard solid plug which gives rise to deafness, tinnitus, and vertigo. Though not usually included under the heading of 'discharge,' impacted cerumen may be the cause of a purulent discharge from the ear.

When a patient complains of an aural discharge, the colour, amount, and character should be ascertained. Any offensive odour should also be noted. The discharge may be purulent, muco-purulent, or serous, but occasionally it consists of blood, either alone or mixed with one of the above-mentioned varieties.

A discharge of blood from the external auditory meatus (*otorrhagia*) may be the result of an *injury.* The following lesions may cause this symptom :—

Fracture of the base of the skull. When the line of fracture crosses the middle fossa it traverses the petrous portion of the temporal bone and opens the tympanic cavity, tearing the tympanic membrane.

Injury to the external auditory meatus, usually at the junction of the cartilaginous and osseous portions, the result of a blow on the chin, the force being transmitted backwards along the inferior maxilla.

Rupture of the tympanic membrane, which may occur as the result of the introduction of a foreign body through the external auditory meatus, or after a fall or blow on the head without any injury to the base of the skull.

Bleeding from the ear is by no means diagnostic of fracture of the middle fossa ; in most cases the skull is not injured. Care must be taken to make sure that the blood has not trickled into the meatus from some small wound of the scalp or external ear. If the external auditory meatus has been injured, the source of the hæmorrhage may be discovered by mopping the blood away with plugs of cotton-wool and examining with a speculum. A tear in the tympanic membrane may be visible if the hæmorrhage comes from the tympanum. If the skull is fractured, the hæmorrhage is usually profuse, while if the membrane alone is injured it is comparatively slight. The hearing should be tested, for if the base of the skull is fractured, injury of the auditory nerve is an occasional complication.

A variety of acute inflammation of the middle ear known as *acute hæmorrhagic otitis* is characterized by a discharge of blood. This trouble is usually associated with influenza, but may also occur in hæmophilic patients. The hæmorrhage is preceded by pain, and the membrane is markedly hyperæmic, or may show petechial spots. A hæmorrhage or blood-stained purulent discharge occurring in the course of chronic suppurative otitis media will probably be due to the presence of *polypi* or of *masses of granulations.* A similar blood-stained discharge may be present in *malignant disease* of the external or middle ear.

Very rarely *erosion of the internal carotid* artery may lead to a severe fatal hæmorrhage from the external auditory meatus. The artery in its course through the carotid canal is separated from the anterior part of the tympanic cavity by only a thin plate of bone, which may be deficient. Owing to chronic suppuration, this portion of bone may be destroyed and the walls of the artery weakened, so that it may give way suddenly, leading to a quickly fatal bleeding from the ear, nose, and mouth. A similar severe or fatal hæmorrhage may result from *erosion of the lateral sinus* from a similar cause.

Cerebrospinal fluid may escape from the external auditory meatus after a *fracture of the middle fossa,* and its presence may be regarded as diagnostic of this injury. The flow is usually copious, and may last for some days. Liquor Cotunnii may escape when the labyrinth is injured. In appearance it resembles cerebrospinal fluid, but the amount is very small. In chronic suppurative otitis media the discharge may be serous, and bear some resemblance to cerebrospinal fluid, from which it may have to be distinguished in a patient unconscious as the result of a head injury. Apart from examination of the membrane through a speculum, this can be effected by collecting some of the fluid and testing for albumin, which is present in considerable quantity in the fluid from the labyrinth. A serous septic discharge also contains much albumin, while cerebrospinal fluid has only the merest trace.

Purulent Discharges.—In by far the greater number of cases the discharge is obviously purulent, muco-purulent, or sero-purulent. Such a discharge may arise from some lesion of the external auditory meatus, from disease of the middle ear, or from suppuration in some adjacent structure, the pus making its way into the external or middle ear, and so draining from the external auditory meatus.

The following lesions of the external auditory meatus gives rise to such a discharge :—

Eczema.—The discharge in this case may be serous. The trouble may be caused by the irritation of a plug of impacted cerumen, or it may be associated with eczema of the auricle or of some other region of the body. It must be remembered that eczema of the external auditory meatus and of the external ear may be caused by a discharge of pus from the middle ear, and hence, when the external auditory meatus is eczematous, every care must be taken to make sure that chronic middle-ear suppuration is not also present.

The presence of a *foreign body,* such as a plug of cotton-wool which has been inserted and forgotten, or of such foreign bodies as children occasionally insert, may produce a dermatitis of the meatus leading to a discharge of pus. This condition is recognized easily on examination with the speculum.

Furunculosis.—This not uncommon trouble gives rise to very acute pain and swelling of the meatus, followed by a discharge of thick pus. A furuncle may usually be seen on otoscopic examination, and the trouble may be diagnosed from the extreme tenderness and swelling of the meatus, and the presence of an opening from which the pus escapes.

Secondary Syphilis.—In this disease condylomata may occur in the external meatus. The discharge is usually serous, and may have a very foul odour. The diagnosis will depend upon the presence of other secondary troubles or the history of the primary sore. Tertiary syphilitic ulceration may also occur at the orifice of the meatus. Wassermann's reaction should be tested.

Suppurating Sebaceous Cyst.—The cutaneous lining of the external auditory meatus is well supplied with sebaceous glands, which may give rise to cysts ; these may inflame and suppurate. The signs, symptoms, and appearances closely resemble those of furunculosis.

Diphtheritic Inflammation of the ear is rare, but the nature of the abundant swelling and muco-purulent discharge to which it gives rise may be overlooked entirely unless the Klebs-Löffler bacillus (*Plate XXVIII, Fig. L,* p. 614) is looked for by cultural methods.

Epithelioma may occlude the external auditory meatus. The appearance is often so characteristic that the diagnosis presents no difficulty ; but it may be simulated by *chronic inflammation* with granulation tissue, by *lupus* of the ear, or by *rodent ulcer* ; the duration of the disease may assist in the diagnosis, but histological examination of a portion of the affected tissue should be resorted to in cases of doubt.

Caries or *necrosis* of the bony external auditory meatus will give rise to a profuse purulent discharge, associated with the presence of polypi or of masses of granulations.

The diagnosis of the above causes of an aural discharge may be easy, but on the other hand it may be a difficult matter to make sure that there is no disease of the middle ear at the same time, for there is likely to be so much swelling, and probably tenderness of the meatus, that it is impossible to get a view of the membrane. It must also be borne in mind that when there is a discharge of pus from the middle ear the lining membrane of the canal is frequently swollen, inflamed, and eczematous.

The most frequent cause of a purulent discharge from the ear is *suppurative otitis media,* either acute or chronic. In the former case, the discharge is preceded by acute pain, usually paroxysmal, with pyrexia and more or less severe constitutional symptoms. The discharge usually consists of thick pus, and there may be much swelling of the meatus preventing a satisfactory view of the membrane. When this can be seen it has a characteristic hyperæmic and swollen appearance, and the perforation through which the pus escapes can be made out.

A bacteriological examination of the discharge will often throw considerable light on the cause and also help considerably in prognosis. The most serious forms are associated with diplococci and the streptococcus pyogenes. The former include the meningococcus (Weichselbaum), the gonococcus, micrococcus catarrhalis, and the Gram-positive pneumococcus. Staphylococcus pyogenes albus and aureus are much less virulent than the others, and are found in connection with furunculi as well as with middle-ear disease. The tubercle bacillus is often found to be the cause of otitis media and otorrhœa in children. Deafness and tinnitus are also present, but there is usually no pyrexia, and pain is remarkably absent. Examination shows a swollen dull red or pink membrane, while a perforation, often in the anterior region may be made out after cleansing the meatus. In this disease, rapid destruction of the bone may also occur without pain.

In chronic suppurative middle-ear disease the character and quantity of the discharge vary enormously, from only a slight serous discharge to a profuse flow of foul pus, Frequently the amount and character vary from time to time, and occasionally the flow is intermittent. The presence of other symptoms, such as deafness, tinnitus, pain, and vertigo, must always be inquired for. The membrane must be examined after syringing out and drying the meatus. In practically every case a perforation will be found, though occasionally the pus may make its way along some bony track which opens into the external auditory meatus. The position of the perforation should be ascertained, whether it is in the membrana propria or in Shrapnell's membrane. In the latter case the suppuration occurs chiefly in the attic, and the ossicles are likely to be carious. Generally speaking, perforations in the anterior portion of the membrana propria are of less serious nature than those in the posterior portion. The presence of polypi or granulations must be noted, and if possible the site from which they spring determined.

In addition to the above causes of an aural discharge, abscesses in adjacent structures may occasionally burst into, and lead to a copious discharge of pus through, the external auditory meatus. An acute *mastoid abscess* may discharge in this way through a sinus which opens on the posterior aspect of the meatus. In chronic mastoid suppuration pus may make its way from the mastoid air-cells in a similar manner. Rarely a *cerebral abscess* in the temporo-sphenoidal lobe may burst into the tympanum and discharge through the external auditory meatus. Occasionally, an *abscess in the parotid gland* may extend backwards and discharge through the anterior wall of the meatus. A similar result may happen with an abscess which originates in *disease of the temporo-mandibular joint*, or even in the neighbouring lymphatic glands. *Philip Turner.*

OXALURIA.—This term is generally used to include any condition under which crystals of calcium oxalate are to be found on microscopical examination of the urine. They occur in two forms, of which by far the most characteristic and common is familiarly described as the 'envelope' crystal—really a regular octahedron; when crystallization has occurred imperfectly, a spheroidal form with a central constriction like that of a 'dumb-bell' may be seen occasionally (*Fig.* 181). Either form is transparent, highly refractile, and usually quite colourless. If the precipitate is sufficiently abundant to be visible to the naked eye, it is generally pure white; it often comes down after more or less mucus has already gone to the bottom of the specimen glass, so that a dense white layer is seen lying on the top of a less white flocculent mass; this appearance has been described as that of the 'powdered wig.'

The crystals are soluble in any mineral acid, but they are insoluble in water or ordinary acetic acid solution. They may be found in any urine, whether acid or alkaline, but are commonest in acid urines. They may be in the urine when it is voided; more often they form as the urine stands in the specimen glass, and it is important to remember that if a slide is prepared from the centrifugalized deposit of a urine and allowed to stand for a while before it is examined under the microscope, numbers of very minute calcium oxalate crystals may appear even when none are to be found in a similar specimen examined at once.

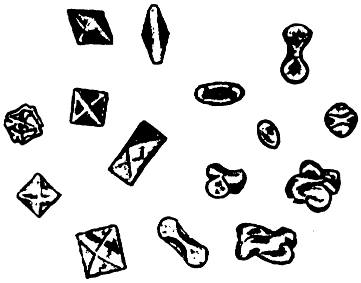


Fig. 181.—Varieties of crystals of calcium oxalate (high power).

The deposition of calcium oxalate is by no means necessarily pathological; indeed, upwards of 20 milligrams are excreted in the urine daily, even 35 milligrams not being beyond the normal limit. So long as the oxalic acid is combined to form soluble salts such as those of sodium or potassium, no envelope crystals appear, but it is common for the proportion of oxalic to other acids on the one hand, and of calcium to other bases upon the other, to be such that the insoluble oxalate of lime is formed and precipitated, either in the urinary passages or after the specimen has been voided.

Certain patients presenting symptoms of gastric or intestinal indigestion have a curious tendency to suffer from alternating oxaluria and phosphaturia ; in their dietetic efforts to obtain comfort they cause marked variations in the reaction of their urine ; at one time it is markedly acid, and oxalates are precipitated, at another it is alkaline and an abundant deposit of phosphates forms. No special significance attaches to this alternation.

Circumstances which cause an absolute increase in the amount of oxalic acid excreted will naturally increase the tendency to visible oxaluria. The best-known exogenous sources of oxalic acid are certain vegetable products, of which the following in particular may be mentioned: tea, cocoa, rhubarb, spinach, gooseberries, figs, coffee, chicory, chocolate, peas, beans, beetroot, artichokes, tomatoes, and beer. It is probable, however, that there is also an endogenous source for oxalate of lime, for even a starving person still excretes oxalates in his urine. The source of these is still obscure, but it is held by many that uric acid, creatinin, and possibly other allied substances, may be a source of oxalate. The fact that uric acid crystals and those of oxalate of lime are to be seen not infrequently, either together, or alternating with one another on different days, would seem to favour this view, and gouty persons are perhaps more liable than others to oxaluria. Calcium oxalate crystals may be found in the urine at any age, however, from infancy onwards.

In great part, oxaluria is physiological and dietetic ; nevertheless there is a decided tendency now-a-days to revert to the older view that when a patient's metabolism is such as to cause a constant deposition of calcium oxalate crystals in the urine, it is also apt to lead to a group of symptoms of which nervous dyspepsia, neurasthenia, and even hypochondriasis are the chief. The discovery of calcium oxalate crystals in the urine of such patients therefore might be of assistance in determining the nature of the dietetic and other treatment that should be adopted for the cure of the other general symptoms.

There are at least three other ways in which the knowledge of the existence of oxaluria may be important :—

1. Concerns patients who present symptoms that may be due to renal or vesical calculus. Microscopical examination of the centrifugalized urinary deposits in such a case may serve to detect not only pus cells and red blood-discs, but also calcium oxalate crystals that, instead of being all separate from one another, as is the rule in a dietetic or neurasthenic case, may be obviously agglomerated into minute calculi ; if there are clinical symptoms of stone, the discovery of microscopic aggregations of crystals is highly suggestive of there being a larger calculus present somewhere in the urinary system.

2. If oxalate crystals are deposited in numbers in the urine whilst it is still in the bladder, irritability of the latter is apt to follow, with a tendency to undue frequency of micturition ; such oxaluria is not altogether infrequent as the cause for persistent nocturnal enuresis in girls and boys.

3. In some ways this is one of the most important features of oxaluria. The same irritation by oxaluria that may produce the nocturnal enuresis in girls and boys may lead to the urine of adults containing a considerable excess of nucleo-proteid, and, in the male, numbers of spermatozoa. There may be no symptoms whatever, and in that case the only importance of the condition lies in the fact that the nucleo-proteid may be mistaken for albumin ; for if the boiling test is applied to a specimen containing nucleo-proteid in excess, a cloud of phosphates may come down, and then when acetic acid is added to dissolve up this cloud, a residual haze may remain behind because the acetic acid, at the same time that it dissolves the phosphates, precipitates some of the nucleo-proteid. This source of fallacy may be obviated in either of two ways : the haze of nucleo-proteid will clear up on addition of a drop of nitric acid, whereas a similar haze, due to albumin, will remain ; or, to make quite certain, three test-tubes may be used : into the first, put plain urine without any boiling ; into the second, urine plus acetic acid without boiling ; and into the third, urine plus acetic acid, the mixture being thoroughly boiled. If the haze is due to a nucleo-proteid only, it will be equally marked in the second and third tubes, whereas if there is albumin as well, the haze in tube three will be denser than that in tube two.

Just as oxaluria may lead to nocturnal enuresis in adolescents, so in a few adult males it has been regarded as a factor in the causation of excessive nocturnal emissions or spermatorrhœa.

Herbert French.

PAIN, ABDOMINAL (General).—Most abdominal pain is local, e.g., that due to a renal or biliary stone or to appendicular colic. The most serious cause of general abdominal pain is *acute general peritonitis*. If this be perforative, at first the pain is local at the seat of perforation, and the abdomen is retracted ; but soon, whether the peritonitis is or is not perforative, the abdomen becomes distended from paralysis of the bowel, and the pain becomes general. Increase in the rate of the pulse, rigidity, and immobility of the abdominal wall on breathing are most important signs for diagnosis. Quickly the pulse becomes more rapid and wiry, the patient looks ill, the temperature is raised a little, the bowels are constipated, and there is some nausea, perhaps vomiting. In cases of doubt it is a good plan to count the pulse-rate at intervals of ten minutes ; a progressive rise in the successive pulse-rates often points to the need for urgent laparotomy. There may be a rapidly progressive leucocytosis. It is often said that the drawing up of the knees on to the abdomen is of importance ; sometimes it is very striking, but in many patients with acute general peritonitis the legs are not drawn up, and they may be drawn up in other conditions. The early diagnosis of acute general peritonitis is of the utmost importance. It has been estimated that in many cases each hour's delay in opening the abdomen means that the chances of death are increased 5 per cent. Morphia should never be given when it is thought there is even a remote possibility that any illness is acute general peritonitis, for it makes the subsequent diagnosis so difficult. The onset of *pneumonia* is sometimes announced by an abdominal pain so acute that the patient is thought to have acute general peritonitis ; the relatively rapid respiration rate may point to the lesion being in the chest, but in some of these cases it is only by anxious watching that one can decide whether the disease is primarily thoracic or abdominal. Sometimes it is both—for instance, in pneumococcal septicæmia.

Chronic General Peritonitis.—This usually causes a dull feeling of heaviness rather than a general acute pain. The chief points to be observed in arriving at a diagnosis are the chronicity of the trouble, the presence of fluid in the peritoneal cavity, and the fact that masses of thickened peritoneum can often be felt. The most usual is the puckered, thickened omentum, forming a tumour lying transversely at the middle of the abdomen ; sometimes other lumps can also be felt. It must not be forgotten that an infiltration of the stomach with new growth will give rise to a tumour lying transversely across the abdomen, and so may a diseased colon. The presence of these peritoneal thickenings often gives the abdomen a dough-like feel. The commonest cause of chronic peritonitis is tubercle. Often there is no discoverable tubercle elsewhere to help us to a diagnosis, but the hectic, irregular temperature may be a guide. As the fluid increases, the umbilicus becomes flattened out (see ASCITES, p. 43), and in tuberculous peritonitis sometimes red.

Intestinal Colic.—This is due to many causes which lead to painful contraction of the intestinal muscles. The pain is always paroxysmal and usually recurrent, so that a severe attack consists of frequently recurring paroxysms. There are all degrees, from quite a slight pain to one that causes the patient to shriek and break out into a cold sweat. The temperature is usually normal, but is occasionally slightly raised. The pulse is usually of normal rate unless the temperature be raised. The abdomen is generally distended, and in a bad case peristaltic movements of the coils of intestine may be seen. Often the abdominal muscles are reflexly contracted and rigid. The pain may come on without warning, or may be accompanied by nausea, eructations, and borborygmi. It is usually felt at the umbilicus, from which region in a severe case it spreads over the whole abdomen. The patient tosses about in the severity of it, and finally selects a position in which he can bring pressure to bear on the abdominal wall ; in peritonitis this, so far from relieving the abdominal pain, increases it. Intestinal colic is usually brought on by eating some indigestible article of food, so the history will help us ; but it may be due to obstruction. In children, intestinal colic is recognized by their cries, restlessness, and the drawing up of the legs.

Acute or *subacute Intestinal Obstruction* is a common cause of general abdominal pain, and requires most careful diagnosis (see VOMITING, p. 767).

Lead Colic.—This is diagnosed by the symptoms of colic, as given above, by the history and occupation, and by the presence of other signs of lead poisoning (p. 34), of which the most characteristic is a blue line on the gums.

Gastric Crises may cause general abdominal pain, but they will be recognized by the absence of knee-jerks and other signs of tabes dorsalis.

Abdominal Neuralgias.—This phrase is applied to severe abdominal pains unassociated with any organic disease. The greatest caution must be exercised, and a diagnosis of abdominal neuralgia must be looked upon with great suspicion, for there is no doubt that such a diagnosis is often wrong, the patient really having organic disease. The pain may be local, e.g., those neuralgias of the kidney which resemble renal calculus, or it may be general. Disease of the spine must be excluded carefully. Often these patients have neuralgia elsewhere. The cases last a long while; they are commoner in women than men. In a few, opening the abdomen has shown that the small intestine or colon is spasmodically contracted (*enterospasm*), and indeed, it may be felt through the abdominal wall as a swelling like a thick cord. These patients are often given morphia, but this should not be done. It is not infrequent to find that severe abdominal pain is apparently due to the administration of morphia, for the pain ceases when the drug is withheld. I have seen two such cases recently.

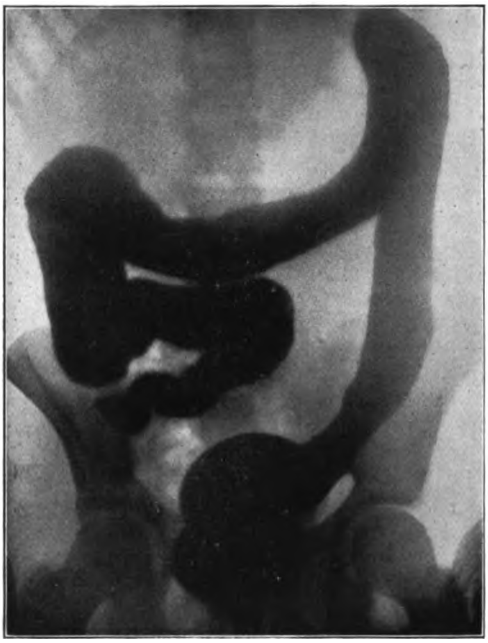


Fig. 182.—Skiagram to show the normal appearance of the rectum, colon, and cæcum after injection of the bowel with a bismuth enema. Note that food has passed the ileocæcal valve and entered the small bowel. Male, age 14.
(*Skiagram by Dr. C. Thurstan Holland.*)

General Visceroptosis often causes a general dull, dragging abdominal pain. It can easily be diagnosed by feeling the displaced liver or kidney, by looking at the abdominal outline seen from the side when the patient stands up (see *Fig.* 56, p. 127; see also CONSTIPATION, p. 121), and by observing the displaced stomach or intestines by the *x*-rays after the administration of bismuth. (Compare *Fig.* 182 with *Fig.* 183.) *W. Hale White.*

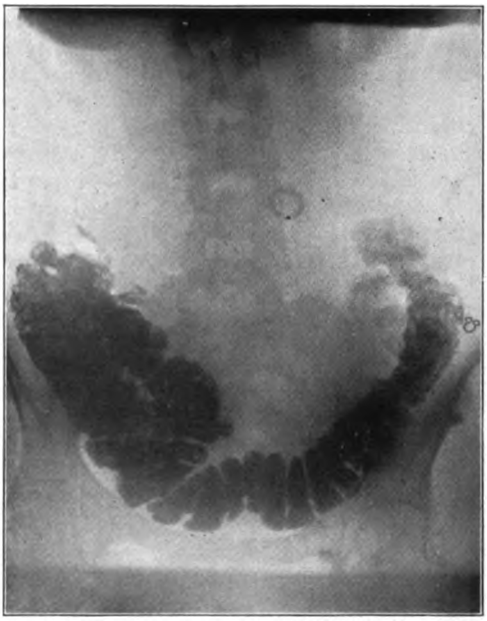


Fig. 183.—Skiagram after a bismuth meal, showing ptosis of the ascending and transverse colon. Twenty-five hours after the meal. Female, age 19. Erect posture.
(*Skiagram by Dr. C. Thurstan Holland.*)

PAIN, BEARING-DOWN.—This form of pain is very frequent in diseases of women, and is an associate of many pelvic conditions. It is impossible in many instances to dissociate it from chronic aching pain; but it is not every chronic pain which has the bearing-down character. It is usually the result of impaction of some pelvic structure, and owes its character more particularly to pressure on the rectum, and sometimes on the bladder. Displacement of pelvic organs, or even simple congestion of them, will sometimes produce it. Its source is not always strictly gynæcological, as it may be the result of rectal disease, such as cancer, ulcer, or hæmorrhoids. It is thus closely associated with rectal tenesmus. The commonest cause is, perhaps, backward displacement of the uterus, and it is most marked in retroversion of the pregnant uterus, especially if impaction of the organ occurs. Impaction of a pelvic tumour may produce it, uterine fibroids, ovarian tumours, and pelvic hæmatocele being the chief swellings which give rise to it. These produce pain of a different character in addition, due to pressure on nerves ; but the bearing-down character is more particularly referred to the rectum, hence it is commonly believed to have some relation to pressure on the rectum. A pelvic abscess of peritoneal origin is an unusual impacted swelling, which gives rise to very severe bearing-down pain : impacted, because it is bound down by peritoneal adhesions, and exercising pressure because of the tension in it. The bearing-down character becomes most marked if the abscess involves the rectal wall, as it so frequently does, causing a flow of mucus and much irritation of the rectum.

The differential diagnosis of the causes of this type of pain can be made only after a complete pelvic examination by abdominal palpation, and bimanual examination by the vaginal and by the rectal touch. Further, it may be necessary to examine the bladder by the cystoscope, or the rectum by the finger or sigmoidoscope. The differential diagnosis of the pelvic disorders mentioned is discussed under SWELLING, PELVIC (p. 688).

T. G. Stevens.

PAIN IN THE ANKLE.—(See JOINTS, AFFECTIONS OF THE, p. 337.)

PAIN IN THE ARM.—(See PAIN IN THE EXTREMITY, UPPER, p. 442 .)

PAIN IN THE BACK.—From occiput to anus, a pain referred to the spinal axis is a frequent complaint, and the diagnosis of its cause is very often a most troublesome problem. We start with the broad generalization that a pain in any area must be due to irritation, either of the trunk or the terminals of the sensory nerves supplying the spot, or, it may be, of a nerve which is in immediate anastomosis with that to the painful area. Pain referred to any one spot and due to central (cerebral) irritation is so rare as not to require mention here (except that arising from gross cerebral trouble, which will be referred to by the patient as HEADACHE, p. 293) ; and applying this principle to the spinal axis, we find that the sensory divisions of the spinal nerves, from the first cervical to the coccygeal, all divide into branches for (*a*) the skin, (*b*) the bones and meninges of the spinal canal, (*c*) the muscles lying on the vertebral column, and (*d*) the viscera contained in the cranium, neck, thorax, abdomen, and pelvis. Consequently, to interpret rightly the meaning of a pain in the back, we must look not only to general conditions affecting the blood (fevers of all sorts are often associated with a general backache as a prominent feature), but to the condition of the organs contained in that spinal segment (or the one immediately above or below it) in which the pain is complained of.

Another very useful generalization is this. We may draw a distinction between a pain complained of spontaneously in a spot not associated with tenderness on firm pressure, and one in which such tenderness is present. In the latter case, the tender spot is located in all probability at or near the seat of the trouble ; in the former case it is probable that the pain is one referred by the brain to the spot, but not really arising there—a ‘ referred pain,’ as it is termed ; and this is the more likely if we find that the skin over the area is very sensitive to light stimulus, but not more sensitive—perhaps even less so—to a stimulus which is rather rougher, a pressure rather firmer than a light touch. Carrying this to its extreme, we have the paradoxical phenomenon of severe pain being complained of in an area the skin over which is absolutely anæsthetic ; this indicates a complete lesion of the trunk of the nerve concerned.

Coming now to the practical diagnosis of a pain in the back, we can pretty easily and accurately eliminate those cases owning a pyrexial origin by observing that the patient not only complains of a pain in the back but looks acutely ill ; if he does so, take his temperature, and if this be found to be raised above 100° F., we may be sure that we have to deal with a zymotic disease at its onset, or perhaps it may be with a meningitis or a myelitis or even acute rheumatism, and in all of these the pain in the back is only an obtrusive symptom, to which will very soon be added some of the signs distinctive of the disease.

Often, however, we have to deal with cases in which the patient, except for the pain in the back, is comparatively well, and he is concerned to know what it means. Two or three questions immediately arise in such a case, the answers to which will throw light on the nature of the trouble. The first thing is to ask him to locate the pain; the next to enquire how did it arise, i.e., did it come suddenly after a blow? after some unusual exertion? after some unintentional movement, say of the head and neck, or a slip off a pavement? And then again, how long has he had it, and has he ever had a similar pain before? Again we proceed to ask, is it constant or intermittent? If the latter, what action on the patient's part will cause it to return, or what position will ease it when it is present?

It is but seldom that we have not by these questions arrived at a provisional diagnosis in our own minds, but we must never omit to make a careful physical examination for points which will corroborate or correct this diagnosis.

Inspection may reveal skin conditions, such as a patch of herpes, which may be either the real cause or an outward manifestation of a cord or bone lesion; swellings or redness may be apparent, or undue prominence of a spinal process; bruises or purpura may be seen, or a pulsating tumour proving aneurysm; glands may be visible in the posterior triangle of the neck. It will also reveal any trace of lateral curvature, a frequent source of backache in young people.

Palpation may reveal great tenderness on pressure, either of muscles or bone; it may show fluctuation (remember that this fluctuation must be vertical, not lateral, to be reliable); it may prove the absence of tenderness, and may also show hyperæsthesia of the skin, suggestive of pain referred from a viscus. A very useful hint is frequently derived from the observation of the results of palpation; sometimes these can better be seen when a special stimulus such as an electric current or persistent rubbing is applied to the skin; thus it may be found that over one small area a blush is raised more easily, or is more persistent, than elsewhere; this is strong evidence pointing to visceral disease as the cause of the pain; it is due to, and proves disorganization of, the sympathetic nerve distributed to the viscus. This method will also reveal hyperæsthesia or anæsthesia if testing be conducted with a light touch and a pin.

The next step is to apply tests for disease of the bony walls of the canal; tap each spinal process in turn with a percussion hammer, and note whether pain is elicited at any spot; jar the heels alternately with the leg held rigid from the hip; letting the patient come down on the heels himself is more risky and less satisfactory; also test for pain on resisted movements of the limbs or trunk.

We may then find if pain is aroused by movements of any kind—flexion, extension, and rotation.

Lastly, with the patient lying on his back, a careful examination must be made from the front in the ordinary way for evidence of any visceral disease, or of growth of any kind.

If the cause of the trouble should still remain obscure, or perhaps in any case for future reference, two or three *x*-ray photographs of the painful area will be taken.

We may now consider the reverse order of procedure, and ask what are the local diseases associated with pain in the back, and what are their distinguishing points. We may enumerate these according to the structures involved, thus:

Skin.—Ulcers, herpes, etc.; obvious on inspection.
Muscles.—Abscesses, trauma, acute inflammations; so-called rheumatism, stiff neck, lumbago, etc.; simple debility; overwork.
Joints.—Rheumatism; implication in caries or in rheumatoid arthritis, etc.
Bones.—Caries, aneurysm, growths eroding; trauma.
Meninges.—Inflammations; growths.
Cord itself.—Tumours; inflammation; trauma.
Viscera in front.—Aneurysm; gastric or duodenal ulcer; dyspepsia; gall-stones; uterine or ovarian trouble; appendix; rectum; bladder and vesiculæ seminales; kidneys.

It would be impossible within the limits of this article to give a complete differential diagnosis of all the above, but the procedures of investigation which we have already noticed will almost certainly enable us to come to some conclusion, and it remains here only to indicate a few of the more special points in differential indications, and a few of the commoner mistakes.

Lumbago v. Tumours.—If a patient complains of 'lumbago' of some standing, it is essential to test the nervous system, the knee-jerks and other leg reflexes, and to contrast them on the two sides; to look for wasting of muscles, especially on one side, to investigate the power of the muscles in walking and in simpler movements; to examine the pelvic organs and the abdomen for growths of any kind. Only when all these points yield negative results can we permit ourselves to think that it is simple lumbago. Lumbago is almost always on both sides; a tumour most frequently gives one-sided symptoms first, though they may spread to the other side later. Rectal examination should never be omitted, and in suitable cases vaginal examination should be made also.

Aneurysm in Thorax v. Indigestion, etc.—It cannot be said that this is a common mistake, but it is a very serious one. The difficulty is that an aneurysm is extremely hard to recognize when it arises from the descending arch; bruits are usually absent, and it is perhaps only when a pulsating tumour in the back appears that the diagnosis is made. The severity, the dull, aching character, and the persistence of the pain are the main features that may help to suggest such a serious cause. The patient should be investigated by the x-rays.

Occipital Headaches v. Caries of Spine.—Owing to the frequency with which delicate patients, particularly women and young subjects, complain of pain in the neck, it is well to draw special attention to this locality, though diagnosis is fairly easy. The occipital headache due to a tumour is unmistakable by its severity, and the almost invariable association of vomiting and optic neuritis. The dull, constant, wearing pain of caries, worse on any slight jar, and the fixed position in which the patient holds the head, are sufficient to arouse suspicion; the x-rays will almost certainly clear up the diagnosis. A simple stiff neck is acute in its onset, and generally preceded by a definite history of sitting in a draught. A 'crick in the neck,' possibly the equivalent for the rupture of a few ligamentous or muscular fibres, can be recognized by its sudden occurrence in the midst of health with no history of previous pain.

Debility in Youth, or *Lateral Curvature v. Caries.*—The shapes of the curves are usually sufficient; but care must be taken to examine the integrity of the bones by the tests given above, and one must not be satisfied until all the bone tests have been tried and found negative. Local rigidity over the painful area, best tested by making the patient stoop and rise again, whilst the physician feels the spine with the flat of the hand, is strong evidence, if persistent, of caries.

Pelvic Organ Trouble v. Lumbago.—This mistake is of course more frequent in women than in men. The only rule to be laid down is always to think of these organs when a woman complains of 'lumbago' or 'backache,' and to inquire carefully into the history of confinements and menstruation, and to make a thorough examination. More mistakes are made in the diagnosis of a pain in the back from want of thought and from carelessness in examination, than from any inherent difficulties in the diagnosis, at any rate in so far as the more serious causes are concerned. *Fred J. Smith.*

PAIN IN THE BREAST.—When pain in one breast is the chief symptom that a patient complains of, the first and most important step in arriving at a diagnosis is to make a most thorough examination of both breasts by inspection and palpation, with a view to detecting any abnormality at all which might suggest an early carcinoma. The methods of such examinations are described on page 685. Unfortunately pain is not by any means an early sign, however, in cases of carcinoma of the breast, and generally by the time it is pronounced there is an obvious stony hard tumour already infiltrating the skin.

Other causes of pain in the breast that will generally be obvious upon inspection or palpation are:—

Cracked nipple
Inflammation of the nipple
Cyst of the breast
Submammary abscess
Mastitis, acute, subacute, or chronic
Galactocele
Epithelioma of the nipple
Tuberculous disease of the breast.

The diagnosis between these various conditions is discussed under the heading of SWELLING, MAMMARY, p. 685.

Pains in the breast due to intra-uterine or to ectopic *pregnancy* will generally be bilateral and associated with the other signs of pregnancy; in unexpected cases suspicion may be aroused by the dark brown colour of nipples which should be pink, and by the broad secondary areola and swollen Montgomery's glands developing around it.

The pains in the breast that are associated sometimes with *menstruation* are also bilateral, and their cause may be indicated either by their development synchronously with the first menstruation or by their periodic recurrence at each menstrual period in an older person.

The chief difficulty in the diagnosis occurs in patients whose breasts may be irritated perhaps by a hard upper border to their stays, or who may have had a breast pain caused by some forgotten injury, but who, perhaps from the occurrence of other cases in their own family or amongst their friends, become terrified by the thought that the sensation which they have is an indication of incipient cancer of the breast. Once this fear has started the pain may assume larger and larger proportions without any existing cause at all; whether to label such a pain in the breast hysterical or a neuralgia or something else is difficult to decide, but the function of the physician or surgeon will be to examine the breast with extreme care, not only once but at intervals, in order to convince himself, and subsequently the patient, that no tumour at all is forming there. He may very likely be in some doubt himself for a while, but in the absence of any trace of even a minute nodule he will be justified in waiting for a re-examination at short intervals of say a week; should the slightest nodule become palpable he will generally be justified in advising its removal for microscopical examination, even though he has little doubt that it is non-cancerous, but if week by week nothing whatever can be found abnormal in the breast, the diagnosis of functional breast pain will be established; and when the patient's mind is set at ease by the absence of any further developments, the pain, previously to her mind severe, will generally disappear.

Herbert French.

PAIN IN THE CHEST is common in all sorts of disorders. Except in the case of highly intelligent persons, or of patients who have had large experience of chest-pains due to various causes, no great help in diagnosing the cause of such pains can be obtained by inquiring into their individual characters. More assistance is furnished by investigating the circumstances in which the pain is chiefly felt, and the conditions that ease or aggravate it. Thus chest-pains due to disease of the heart will be increased by anything that makes the heart beat more rapidly; those due to lung-disease by anything causing the patient to breathe faster or cough; those caused by disorders of the stomach will generally be aggravated by or soon after taking food. For pains in the back wall of the chest, see PAIN IN THE BACK (p. 427) and PAIN, INTERSCAPULAR (p. 461). For clinical purposes, pains in the chest are best classified according to their pathology, and the nature and situation of the disorders to which they are due.

1. **Pains due to Diseases of the Tissues composing the Thoracic Walls:** the pain is in most cases a direct pain:—

Inflammation of the skin and subcutaneous tissues; mastitis
Adiposis dolorosa; neurofibromatosis
Myalgia: pleurodynia: stitch
Neuralgia: mastodynia
Herpes zoster
Pressure on nerves
Disease of the bones of the chest.

2. **Diseases of the Thoracic or Abdominal Viscera;** the pain is in most cases a referred pain:—

Pleurisy: empyema
Pneumothorax
Pneumonia
Pulmonary embolism
Heart disease—Valvular disease, aortitis; angina pectoris, true and false
Pericarditis
Aneurysm: dissecting aneurysm
Mediastinal new growths
Mediastinitis
Œsophageal obstruction
Diseases of the spinal cord.

Diseases of the Thoracic Wall.—Pain in the chest due to *inflammation of its superficial tissues* should not be hard to diagnose. The pain will be confined to the inflamed parts and their immediate neighbourhood, and the other three cardinal signs of inflammation—heat, redness, swelling—will not be absent. In most cases a superficial wound or abrasion will be found; in others, the inflammation will have spread to the surface from some deep-seated lesion, caries of a rib, for example, or an empyema or hepatic abscess, or a metastatic abscess arising in the course of pyæmia. The diagnosis must be made on general lines in these unusual cases. *Mastitis* or mammary *carcinoma* will be diagnosed by palpation. The inflammatory phenomena of *herpes zoster* are considered below.

The very rare condition known as *adiposis dolorosa*, or Dercum's disease, is characterized by symmetrical and painful deposits of fat about the body and limbs. It occurs mainly in middle-aged women of full habit, though males are not exempt; chronic alcoholism is its usual precursor. *Neurofibromatosis* is characterized by the growth of multiple benign false neuromata on the nerves, which give rise to pain; but they are not tender on pressure, and so contrast with the single false neuromas, which equally give rise to pain over the area or distribution of the nerves on which they are situated.

When pain is felt in the intercostal or other muscles about the chest, and can be referred to nothing more definite than 'muscular rheumatism,' the condition is referred to as one of *myalgia* or *pleurodynia*. Tenderness of the affected muscles is the only physical sign present, and it is important that graver mischief, such as pleurisy or pneumonia, should be excluded before the diagnosis of pleurodynia is made. The sudden pain in the side familiar to untrained athletes as *stitch* comes on after sudden exertion, and is in all probability due to overstrain of the fibres of part of an intercostal muscle. All these muscular pains are relieved by rest or pressure, and aggravated by exertion.

Pains in the chest may be due to *neuralgia*, a term which is theoretically applied to pain felt in a nerve that shows no evidence of active or old disease. Practically, however, neuralgia is the name also given to nerve-pains that follow organic disease both in the nerve itself (herpes, neuritis, etc.) and in other parts of the body (gout, tabes, etc.). In *intercostal neuralgia* the pain is felt along the course and distribution of one or more of the intercostal nerves. There is marked tenderness on pressure in the affected intercostal space, with three points of maximum tenderness corresponding to the posterior primary, lateral cutaneous, and anterior cutaneous branches of the nerve, given off near the vertebral spines, the mid-axillary region, and the costosternal articulations. The pain is increased by movement or breathing. Unilateral intercostal neuralgia often follows herpes, and must be distinguished carefully from pains that may be felt in organic disease, such as tabes, aneurysm or mediastinal tumour, and vertebral caries, in which the intercostal nerve is directly or indirectly involved. In *phrenic* or *diaphragmatic neuralgia*, a rare condition, the pain is felt in the lower part of the thorax along the line of insertion of the diaphragm, which may be tender on pressure; coughing and breathing are acutely painful, but there will be no physical signs of disease except the tenderness on pressure. The diagnosis must be made from diaphragmatic pleurisy or peritonitis, acute hepatic or splenic disorders, and spinal caries, on general lines. *Mastodynia*, mammary neuralgia, or the 'irritable breast' of Astley Cooper, occurs in women during pregnancy or lactation, or in connection with pelvic disease. The pain is constant, with paroxysmal exacerbations, and its severity may lead to the fear of cancer. Local changes —redness, swelling, tenderness—may be found about the breast and nipple.

Pains in the chest are habitually felt in *herpes zoster* of the intercostal nerves, sometimes before, always during, and often after the attack: the third, fourth, and fifth intercostals are those most often involved. Groups of vesicles arise over the area of distribution of the affected nerve, filled with serum and implanted on an inflamed base; they are most marked about the exits of the posterior primary, lateral cutaneous, and anterior cutaneous branches. The axillary glands become enlarged if the herpes is above the seventh dorsal nerve, the inguinal if it is below it. In about a week the eruption scabs over; in all but the mild cases, small whitish scars remain as permanent evidence of the attack. The diagnosis is obvious in cases presenting the eruption or its scars, but may be difficult until the herpetic vesicles have appeared. It is especially in older patients that severe neuralgic pains are likely to remain for months or years as a legacy from herpes, and they may be very intractable.

Pains in the chest will be felt whenever there is *pressure on an intercostal nerve*; in many cases such pressure is bilateral, when the patient will complain of girdle-pains. Injury or fracture of the spinal column may involve the posterior nerve-roots or the intercostal nerves, either at once by the pressure of fractured bone or of effused blood, or later by the pressure of callus; abscesses, aneurysms, or primary or secondary new growths, may press on the nerves and give rise to severe pain in their areas of distribution. In the great majority of cases there will be other physical signs or symptoms to point to the diagnosis; but where there are none, and the pain is due, perhaps, to a minute carcinoma in the spinal canal, or to a small thoracic aneurysm that strikes backwards and presses on an intercostal nerve, there is no little danger lest the patient be treated for functional disorder or malingering. The pains are very severe, and persist for months in spite of treatment, while the patient is likely to lose health, weight, and strength. It is true that these phenomena may also be observed in functional cases; but the diagnosis of functional disease or neuralgia should not be made until the most careful physical examination, including the use of the x-rays, has excluded organic disease of all sorts.

Chest-pains are, of course, common in *inflammation* or *injuries of the bones* of the chest—coccal infections, tuberculosis, hydatid disease, etc.—or of the *joints* connected with these bones. In few such cases will the diagnosis of inflammation present difficulty.

Diseases of the Viscera.—Pain in the chest is extremely common in the various diseases of the thoracic viscera, inflammatory or otherwise. In *acute pleurisy* the onset is often insidious, and the pain felt most acutely in the mammary or axillary region, being made worse on breathing deeply or coughing. The pain is stitch-like, lancinating, described as resembling 'a knife,' 'stabbing,' 'tearing'; it is relieved by anything that assists in immobilizing the affected side. The intercostal spaces are tender to pressure in pleurisy, just as they are in intercostal myalgia. The diagnosis turns on the discovery of other physical signs of pleurisy, whether with or without effusion, particularly of pleural friction-sounds. In *diaphragmatic pleurisy*, the pain is felt in two chief sites: one near the costal margin, corresponding to the attachment of the diaphragm; the other about the crest of the shoulder, corresponding to the cutaneous distribution of the fourth cervical nerve; this is a referred pain, due to afferent stimuli coming up the phrenic nerve to the spinal centre of the fourth cervical nerve. The pleuritic friction sounds often fail to make themselves heard in diaphragmatic pleurisy, and the diagnosis of intra-abdominal disease (cholecystitis, appendicitis, peritonitis) has often been made and acted upon in such instances. The pain in *empyema* is much like that of pleurisy; it should be noted that the appearance of a pleural effusion, whether serous or purulent, often coincides with a diminution in the amount of pain felt, because the two inflamed pleural surfaces become separated by the fluid and cease to be rubbed together by the respiratory movements. *Chronic pleurisy* and old pleural adhesions give rise to much of the chronic pain in the chest and shoulders and root of the neck that occurs from time to time in patients with pulmonary tuberculosis. Pain and tightness in the chest are common in *bronchitis*, with or without *emphysema*; here the diagnosis will not be difficult if pleurisy can be excluded, and much of the pain is probably due to overstrain of the intercostal muscles.

In *pneumothorax*, about half the cases show an acute onset, with the sense of something tearing or giving way in the chest as the patient coughs, and sudden very severe pain in the side, aggravated by breathing. In addition the patient exhibits dyspnœa, prostration, cyanosis, and rapid and feeble action of the heart. The onset in more than half the cases is insidious, and the condition subacute or chronic, with comparatively little complaint of pain. The diagnosis, if not made from the history, should be manifest on consideration of the physical signs. The affected side of the chest moves very little on respiration, and is increased in measurement; vocal fremitus is absent; the note on percussion is usually tympanitic, in rare cases dull; and the voice- and breath-sounds are absent on auscultation. If the pneumothorax is at all extensive, the heart will be displaced considerably towards the sound side. Examination with the x-rays will show that the diaphragm is immobile on the affected side, and the air-containing pleural cavity extremely translucent (*Fig.* 226, p. 531); the lung forms a shrunken and opaque mass near the middle line and against the spinal column. After a few days, more or less evidence of pleural effusion at the base of the pleural cavity will usually be found.

In *pneumonia*, chest-pain is extremely common, and is due to pleurisy. If the physical signs characteristic of pneumonia delay their appearance, as is sometimes the case, and if the pleural friction escapes detection, the diagnosis of some relatively harmless condition such as pleurodynia or intercostal neuralgia may incautiously be made. This mistake should never occur; nor is it likely to do so if due attention be paid to the patient's temperature, aspect, pulse, and pulse-respiration ratio.

Pain in the chest is common in *acute pericarditis*, and is referred to the precordia generally, or to the lower part of the sternum. In many cases no complaint of pain is made; in a few instances the pain is exceedingly severe, resembling that of angina pectoris. The diagnosis will turn on the discovery of other signs or symptoms of pericarditis, particularly of pericardial to-and-fro friction-sounds; the patient is often pale and anxious-looking, and very short of breath. It should be remembered that the friction-sounds often remain unchanged when a dry pericarditis has been converted into a wet one by the effusion of fluid. Pericardial friction-sound is characteristically a superficial grating, rubbing, or creaking, usually double or to-and-fro; in rhythm it is often not synchronous with either systole or diastole, beginning in one and being carried on into the other. It can often be altered by pressure with the stethoscope or by changing the patient's position; often it varies from day to day; and it is not conducted well in any direction beyond the precordia, being heard within an area that often does not correspond with the areas of audition of valvular murmurs. These characters should suffice to distinguish pericardial friction-sounds from the murmurs of valvular disease; but it may be very difficult in certain cases to distinguish pericardial from *pleuropericardial friction-sounds*—that is to say, friction-sounds generated in pleurisy by the heart's movements. If there is pleurisy of the thin anterior edge of the left lung that comes between the parietal and pericardial pleuræ, the beating of the heart will readily give rise to friction-sounds that have a cardiac and not a respiratory rhythm, but are due to pleurisy and not to pericarditis. Pain in the chest will be felt in either case; the two may generally be discriminated by the influence of deep inspiration and expiration on the friction-sound. In pericarditis, expiration will strengthen and inspiration will weaken (but not abolish) the friction-sounds. Pleuropericardial friction will in all probability be altered profoundly by respiration, being much increased in one phase (whether inspiration or expiration), much diminished, or lost, in another.

Pain in the chest is common in cases of *heart disease*, taking in general two forms: (1) Precordial pain; and (2) PALPITATION (p. 484). There is nothing characteristic about the precordial pain felt in heart disease, except the fact that it is brought on most often by exertion or excitement. Very similar pain may be experienced by patients with sound hearts who are suffering from *flatulent dyspepsia*; here the pain is usually felt after meals, but may be brought on by exertion if the latter is made soon after food has been taken. The diagnosis must be based on the general signs and symptoms exhibited by the cardiac patient. In *aortic incompetence*, the precordial pain is sometimes exceptionally severe, taking the character of angina pectoris, and radiating down the left arm or through to the back. The sensory nerves of the heart are connected with the spinal cord from the first to the eighth dorsal nerve roots; the first and also the most painful impressions are usually received at the second dorsal roots, which are described as being most central to the paths of pain from the heart. The painful impressions received from the heart at these root-centres are referred to the corresponding areas of cutaneous nerve distribution. Those from the ventricle ascend from the second to the fifth; from the auricle, the fifth to the eighth; from the ascending aorta, the third and fourth cervical, and the first to the third dorsal root-centres. These anatomical connections explain the distribution of the pains in the chest and elsewhere felt in diseases of the heart and aorta. Severe pain in the chest, often of anginal character, is felt in acute or chronic *aortitis* occurring in young syphilitic or rheumatic patients, with or without valvular disease; the pain is most marked when the base of the aorta and the coronary orifices are involved.

Pains in the chest, together with mental anguish, are the outstanding features of *true angina pectoris*, and are in most cases brought on by exertion. The pain is in the region of the heart, and suggests that the heart has been caught in a vice, so excruciating is it. A sense of impending dissolution, or of a pause in the operations of nature, has been described as added to the physical torture. Radiations of the pains through to the shoulder, down the left arm's inner side to the little and ring fingers (less often down the right arm), up the neck, into the supra-orbital region, are very common. The patient becomes faint and collapsed, pale, and clammy; the pulse changes; flatulence and the passage of abundant pale urine follow the attack, which may last for a few seconds or minutes, or may continue, with varying intensity, for hours. The diagnosis will rest on the extreme severity of the pain, its association with valvular disease or arteriosclerosis, or both, and the fact that the attacks are almost always brought on by exertion or severe emotion. The true must be distinguished from *false angina pectoris*, also called pseudo-angina or vasomotor angina pectoris. This commonly has a neurotic, less often a toxic (tobacco, tea, coffee) basis ; it is less severe, and is never fatal. It is far commoner in women than in men ; often comes on when the patient is at rest, or at night ; may occur at any age, and is not associated with cardiac or vascular disease. Attacks of false angina last for an hour or two. True angina is perhaps ten times commoner in men than in women, and occurs between the ages of forty and sixty as a rule ; false angina is perhaps five times as common in women as in men, and occurs in younger patients. Typical cases of the two conditions will be distinguished readily from one another, but every gradation is met with, and it may be impossible to refer intermediate cases—for example, patients with severe heart-attacks and valvular or myocardial lesions—with precision to either one class or the other.

Chronic or recurrent pain in the chest is a very variable symptom of *aortic aneurysm*. In some patients, a large aneurysm may erode rib-cartilages and intercostal spaces, and present itself at the surface of the body without having made itself felt. In others, agonizing pain (true angina pectoris) may be occasioned before an aneurysm at the root of the aorta has grown large enough to produce any physical signs at all ; in these the pain is no doubt due to aortitis or mesaortitis for the most part, or to obstruction at the coronary orifices. Speaking generally, the pain of aortic aneurysm may arise in two ways : (1) From changes in the aortic wall, already considered ; and (2) From pressure on neighbouring structures, particularly the walls of the chest. Pressure-erosion of the sternum or costal cartilages may be comparatively painless in fortunate cases. Erosion of the vertebral bodies commonly gives rise to intense and continuous boring pains in the chest that wear the patient out and make life insupportable ; girdle-pain may result from pressure on the intercostal nerves (direct pain), and referred pains up the neck or down the inner side of either arm may also be felt. Pressure on the œsophagus may give rise to dysphagia and pain, the pain being increased by swallowing. Compression of the lung may lead to pulmonary collapse and inflammation, when pain from pleurisy will probably be felt. It appears that no particular complaint of pain follows compression of the trachea, bronchi, phrenic or vagus nerves, or heart. An acute and severe pain, on the other hand, may arise should the aortic aneurysm perforate and allow blood to escape into the adjoining parts. Such perforation may take place into the air-passages, œsophagus, large intrathoracic pulmonary or systemic veins, pericardium, heart, pleura, peritoneum, or spinal canal. The appearance of the appropriate physical signs will suggest the diagnosis of such a perforation. If the effused blood is limited in amount, the patient will appear more or less blanched and collapsed ; if a great quantity escapes, rapid or sudden death may occur. Particular mention may here be made of the pain due to the formation of a *dissecting aneurysm*. The arteries are acutely sensitive to pain, as may be seen when an artery is ligatured in a conscious patient ; the establishment of a dissecting aneurysm is a terribly painful experience, and is equivalent to an attack of true angina pectoris. If the escape of blood is limited by the walls of the aorta, recovery is likely to occur. The diagnosis of such an incident could only be made on grounds of probability.

Pain in the chest is usually an early symptom of *mediastinal new growth*, and varies in its nature and distribution with the cause and site of its origin. If the growth is in the anterior mediastinum, the pain will be behind the sternum : if in the posterior mediastinum, pressure on or erosion of the vertebræ will set up the severe continuous boring pain referred to above as occurring in aortic aneurysm ; if one side of the chest is involved, the pain will be felt in the side, and down the arm if the brachial plexus is pressed upon. It is often of a darting and lancinating character, shooting up into the neck and head, or down into the abdomen. It may be constant, intermittent, or paroxysmal ; in some cases it is a discomfort rather than a pain, the complaint being of fullness or tightness in the chest. Other symptoms of mediastinal tumour are, first and foremost, continuous or paroxysmal dyspnœa ; evidences of pressure on the air-passages, œsophagus, or nerves ; cough, expectoration, hæmoptysis, alterations in the voice or cough ; disturbances in the action of the heart, and evidence of venous obstruction. Anæmia or even cancerous cachexia are not rare. The general diagnosis of mediastinal tumour (including aneurysm) is seldom difficult once pressure-symptoms of any sort have appeared, for these are very rarely caused by other lung-conditions associated with pain in the chest, such as bronchitis, bronchiectasis, or pulmonary tuberculosis. But it may be very difficult to decide between aortic aneurysm and mediastinal new growth in certain cases. Aneurysm is commoner in men than in women, and rare in persons who have not had syphilis; the patients commonly give a positive Wassermann reaction; anginal pains and the very severe pain of bone-erosion are commoner in aneurysm than in mediastinal new growth; anæmia, cachexia, and irregular pyrexia are in favour of new growth; and so is the discovery of new growth in other parts of the body and of secondarily-infected lymphatic glands. Examination under the *x*-rays will prove of the greatest help (*Fig.* 100, p. 209, and *Fig.* 184), the rays being passed through the patient's thorax in a number of horizontal directions successively; if this be done, the presence of an aneurysm and its connection with the aorta can almost always be established when one is present, to the exclusion of mediastinal new growth.

In *acute mediastinitis* and *mediastinal abscess*—both of them rare disorders and due to syphilitic, tuberculous, or other infection of the mediastinum—pain behind the sternum is commonly the chief complaint, with marked superficial tenderness and a tendency to radiation through into the back or shoulder. Local signs of fullness and inflammation may develop, particularly about the intercostal spaces in front and the episternal notch; and mediastinal crepitations resembling pleural friction have been heard about the sternum.

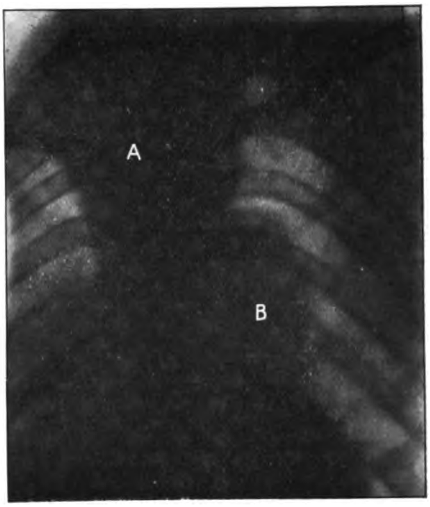


Fig. 184.—Skiagram of an aneurysm of the innominate artery (A), with dilated aortic arch (B).
(*By Dr. Alfred C. Jordan.*)

In *chronic mediastinitis* or *mediastino-pericarditis*, another rare condition due to inflammation arising in the mediastinum, or spreading to it from the pericardium, and seen in youth or early adult life, chronic pain behind the sternum and a sense of tightness and dragging in the chest may be present. The main symptoms will be cardio-vascular, valvular disease of the heart and adherent pericardium leading to cardiac troubles, and the mediastinitis causing venous obstruction. Shortness of breath, with sudden and severe attacks of dyspnœa, lividity, anasarca, ascites, and progressive distention of the thoracic veins; new growth being excluded by the duration of the case.

Deep-seated pain within the chest and at the bottom of the sternum may be felt in diseases of the *œsophagus*, being evoked by the act of swallowing. In cicatricial stricture or carcinoma of the tube, pain is less prominent than Dysphagia (p. 194), and progressive emaciation is the rule. In younger and neurotic patients, on the other hand, spasmodic stricture of the œsophagus may give rise to difficulty in swallowing, with much complaint of pain and constriction in the throat and chest. This condition, termed *œsophagismus*, or *cardiospasm* (*Fig.* 98, p. 198), occurs in hysterical young persons and in hypochondriacal old ones; it is improved or cured by the passage of a bougie; is associated with other evidences of the neurotic temperament; and must be diagnosed from organic œsophageal stenosis.

Pain in the chest is met with frequently in diseases of the abdominal viscera, particularly of the stomach. 'Pains round the heart,' often accompanied by or productive of Palpitation (p. 484), are the common basis upon which patients build when they come complaining of 'heart disease' or 'weak heart,' while as a matter of fact they are suffering from the less serious condition of *gastritis*, or *flatulent dyspepsia*. The pain is felt at the bottom of the sternum and in the epigastrium; it is often of a dull boring character, and radiates out towards the left breast and through to the back between the blade-bones. It is definitely connected with the taking of food, and relieved by vomiting or the eructation of wind; and these are the characters by which it must be diagnosed. In other instances, the complaint is of 'heart-burn,' a burning pain felt over the lower part of the sternum, and probably due to the regurgitation into the œsophagus of the gastric contents during digestion. It is a referred pain felt in the area of distribution of the fifth dorsal nerve, and is often associated with pyrosis or water-brash, the regurgitation of acrid waterv gastric contents into the mouth. For the most part, however, pain due to gastric disorders (such as ulcer, new growth) is referred to the upper part of the abdomen rather than the chest.

Pains in the chest are not rare in various diseases of the *spinal cord.* Girdle pains or girdle sensations are common in *tabes dorsalis,* the patient feeling as if constricted by a hot or painful girdle. They occur early in the disease, and so are often set down vaguely to gout or rheumatism, when a more careful examination would yield early evidences of tabes. In *transverse myelitis,* or *fracture of the dorsal column* with injury to the cord, girdle pains round the chest may be felt at the level of the cord lesion, with loss or abolition of sensation below it.

A. J. Jex-Blake.

PAIN IN THE EAR.—(See Earache, p. 202.)

PAIN IN THE EPIGASTRIUM.

A. **Sudden, severe epigastric pain** may result from the rupture of a gastric or duodenal ulcer, of a gangrenous appendix, or from acute pancreatitis. The pain in such a case is attended by severe shock and signs of collapse, and it may be difficult to say to which of the above causes it is due. The past history of the patient and a careful study of the other signs present, may guide one to a correct conclusion; but as all the conditions mentioned require immediate surgical treatment, the differential diagnosis is made by laparotomy.

When the diagnosis of an abdominal emergency has to be considered, if the history, symptoms, and signs do not exactly fit acute intestinal obstruction, or stomach or duodenal perforation, perforating appendix, or acute cholecystitis, and yet have some resemblance to each of them, pancreatitis is the most probable cause.

The pain of *acute intestinal obstruction* may be referred chiefly to the epigastrium. Vomiting is usually a prominent symptom in such a case. (See Constipation, p. 121; Meteorism, p. 388; Vomiting, p. 767.)

During an attack of *biliary colic* the pain may be chiefly epigastric. The restlessness of the patient in such a case is often of diagnostic value.

B. **Chronic or recurrent pain in the Epigastrium** may be due to a variety of causes:—

(1). It should be remembered, in the first place, that epigastric pain may be due to *extra-abdominal causes.* Amongst these are *spinal caries* (especially to be thought of in children), *pleurisy,* and *intercostal neuralgia.* The first two of these can be distinguished by the usual signs; intercostal neuralgia is to be diagnosed by the presence of tender points along the course of the nerve, and by the absence of all signs and symptoms of organic disease. An *x*-ray examination of the chest should be resorted to to exclude gross intrathoracic changes, such as aneurysm, before intercostal neuralgia is diagnosed finally.

A dilated right ventricle may also be the cause of severe epigastric pain, which may even simulate the pain of gastric ulcer or gall-stones. In cases of emphysema or heart failure this should be borne in mind. In such cases the pain is aggravated by exertion.

Small *epigastric herniæ* may cause recurring attacks of severe epigastric pain. They can be detected by careful palpation, usually in the linea alba.

Affections of the abdominal muscles, e.g., strain from coughing, or rheumatism (in children), may also cause pain in the epigastrium.

(2). Assuming these to be excluded, the cause of the pain may be looked for in the following organs:—

(*a*). *Stomach.*—The chief causes of gastric pain are carcinoma, ulcer, hyperchlorhydria, and gastralgia. (See Indigestion, p. 315.)

The pain in *carcinoma* is usually more or less continuous, although apt to be aggravated temporarily by food. A tumour may be felt. Vomiting is usually present, and the gastric contents show absence of free HCl in most cases.

In cases of *ulcer* the pain is sharper and more definitely related to the taking of food, and often passes through to the back. Vomiting is usually a feature, with or without hæmatemesis. There is localized deep tenderness on pressure, often over quite a small and well-defined area. The gastric contents usually show the presence of an excess of acid.

In cases of *hyperchlorhydria* the pain is less severe than in either of the above conditions ; it occurs in the late period of digestion, and is relieved temporarily by the taking of food. There is an absence of other signs and symptoms, and of local tenderness ; and a test-meal shows the presence of an excess of acid.

Gastralgia should only be diagnosed when all other possible causes of gastric pain have been excluded. The patient is usually a young woman ; the pain may occur even when the stomach is empty, but is aggravated by food, even by liquids. Vomiting is usually absent, and on physical examination there is diffuse deep tenderness over the whole of the gastric area.

Epigastric pain may also be felt to a greater or less degree in all conditions of the stomach associated with flatulence, and in that case it is relieved by the bringing up of wind. (See FLATULENCE, p. 240.)

The *gastric crises* of tabes may be attended by severe epigastric pain, and as these may occur in the pre-ataxic stage of the disease, before other signs are present, the diagnosis may be in doubt. The characteristic features are the sudden onset of the pain, and the fact that it is usually attended by urgent vomiting. There is no rise of temperature, but during the attack the blood-pressure is raised ; whereas in all other forms of acute abdominal pain (except lead colic) it is lowered. Absence of the knee-jerk, and the characteristic pupil signs of tabes, are not necessarily present.

Perigastric adhesions are a possible cause of epigastric pain, but are difficult to diagnose with any certainty. If the pain is much influenced by muscular movements, or change of posture, it is in favour of adhesions being the cause ; but except for this, it has no other characteristic features.

(*b*). *Duodenum.*—The characteristic ‘ hunger-pain ’ of duodenal ulcer (p. 271) may be referred to the epigastrium.

(*c*). *Liver* and *Gall-bladder.*—Epigastric pain may be produced by *congestion of the liver*, either active (hepatitis), or passive, as in mitral disease. It is also produced by such conditions as hepatic abscess and carcinoma (see LIVER, ENLARGEMENT OF THE, p. 366).

Stone in the gall-bladder may sometimes be the cause of epigastric pain, which may even be definitely related to meals, or to the taking of a particular article of food. Pressure over the gall-bladder will often elicit tenderness ; and if the patient is made to take a deep breath whilst the pressure is applied, there will be a painful catch in the breath as the diaphragm descends. In a doubtful case, in which the diagnosis lies between gall-stones and gastric ulcer, the following points are in favour of gall-stones ; (i) The occurrence of the pain at rather long intervals, with comparative freedom from symptoms between ; (ii) Long duration of the attack of pain ; (iii) Continuance of the pain in spite of vomiting ; (iv) The occurrence of slight shivering and rise of temperature with the attacks ; (v) A comparatively low degree of acidity of the gastric contents. Gall-stones are relatively commoner in women ; ulcer in men.

(*d*). *Pancreas.*—Pancreatic calculi, chronic pancreatitis, or new growth, may all be the cause of epigastric pain. An accurate diagnosis of these conditions is difficult, and often impossible ; but other signs of disturbed function of the pancreas may be present, such as fatty diarrhœa, or a ‘ pancreatic reaction ’ (p. 100) in the urine. A tumour also may be felt. Glycosuria may be present, but is not invariable. In cases of chronic pancreatitis there is usually a history of gall-stones.

(*e*). *Abdominal Aorta.*—An *abdominal aneurysm* may cause pain in the epigastrium, but the pain is more marked in the back. The patient is usually a young man with a syphilitic history, and a pulsating expansile tumour can be felt on deep palpation. The *x*-rays will confirm the diagnosis.

Abdominal angina, which is associated with arterial atheroma and a high blood-pressure, is an occasional cause of severe epigastric pain, which comes on in paroxysms, especially upon exertion. The pain in such a case tends to radiate like that of true angina, and is often attended by flatulence, tenesmus, and other abdominal symptoms.

There are usually signs of atheroma in the peripheral vessels; and the diagnosis may be confirmed by the yielding of the pain to vascular depressants, and especially to diuretin.

(*f*). *Colon.*—Spasmodic contraction of the transverse colon (enterospasm) may be a cause of epigastric pain, which may simulate gastric pain by being induced by the taking of food. Such pain, however, tends to be relieved by pressure, and by the passage of gas per anum. Obstinate constipation is usually a feature of the case, and there are often mucus and shreds of membrane in the motions (muco-membranous colitis). A similar pain may be due to *plumbism*, for the diagnosis of which see p. 34. *Robert Hutchison.*

PAIN IN THE EXTREMITY (LOWER).—The causes of pain in the lower limbs are so numerous that much space would be required if any attempt were made to discuss them in full. Fortunately the majority are detected easily when attention is paid to the site, nature, and history of the pain, and the painful spot is examined. An attack of gout in the big toe, an ingrowing toenail, a flat-foot, a synovitis of the knee or ankle, phlebitis of a varicose vein, a tumour of one of the long bones, and many other localized pathological processes require only an elementary medical knowledge and the enlightened use of eyes and fingers for a correct diagnosis to be made. On the other hand, there are many conditions of which pain of a more or less diffused type is a prominent symptom, and which require very careful investigation if mistakes are to be avoided and diagnostic traps escaped. The fact that the nerves of the leg spring from the lower part of the spinal cord in the dorso-lumbar part of the vertebral column, and that they have a somewhat lengthy course within the lumbo-sacral vertebral canal and the pelvic cavity, where they are comparatively inaccessible, before they reach the limb, explains why the origin of some pains referred to the lower extremity is rendered obscure. Moreover, some of the painful conditions met with are connected only indirectly with the nervous paths, and are more directly associated with morbid conditions of other structures, such as joints, blood-vessels, etc.

The classification of the various painful conditions in the lower extremity which need our attention from the point of view of diagnosis is no easy matter. One may consider first those which are primarily nervous in origin, and use them as a basis for comparison with those due to disease of other tissues.

1. Pains of Neuralgic or Neuritic Origin.

Sciatica.—This name is applied commonly to a condition of the sciatic nerve which may sometimes be described as a neuralgia and sometimes as a neuritis, according to the severity of the attack and the amount of alteration in nervous function to which it gives rise. It is characterized by pain of a neuralgic type referred to a part or the whole of the course of the sciatic nerve and its branches, from the sciatic notch to the sole of the foot. Usually the pain is most severe along the back of the thigh and along the outer side of the leg. Tenderness is found on pressure over the gluteal region, over the sciatic notch, and generally all along the nerve. Exacerbation of pain is produced by stretching the nerve, for instance by forcibly flexing the thigh on the trunk with the knee fully extended. The pain is intensified by muscular exertion, and is often severe at night, especially when the patient lies on his back. Sciatica is often associated with lumbago, pain and tenderness in the muscles of the lumbar region. In long-standing cases the nutrition of the affected leg suffers and the muscles appear generally smaller than those of the other limb, but localized atrophy picking out individual muscles suggests that there is something more than a simple neuralgia or neuritis at work. Numbness, and even slight cutaneous anæsthesia, may be found on the dorsum of the foot, in the distribution of the musculo-cutaneous branch, in cases of simple sciatic neuritis. The knee-jerk is never affected in sciatica, but the ankle-jerk is often diminished or lost, and may remain absent for a long period after the pain has disappeared. The plantar reflex is of the flexor type.

Before making a diagnosis of sciatica or sciatic neuritis in a patient who complains of pain in the course of that nerve, the physician must satisfy himself that there is *no gross disease in the hip-joint, pelvis, or spinal column* which could give rise to the symptoms. The mobility of the hip-joint must be investigated carefully, and, if doubt exists with regard to its integrity, the joint should be skiagraphed. The pelvis should be examined externally and internally per rectum or per vaginam. The writer has seen a case of *sarcoma of the innominate bone* mistaken for sciatica, when a glance at the pelvis as a whole was sufficient to demonstrate the swelling on the affected side. In the same way the mistaken diagnosis of sciatica has frequently been made when a rectal or vaginal examination would detect a *pelvic inflammatory* or *malignant mass pressing on the nerve.* Even a *retroverted uterus* may sometimes cause pain in the sciatic distribution. *Tuberculous, gummatous,* or *malignant disease of the lumbo-sacral vertebræ, tumour,* or *meningitis involving the lower part of the spinal cord and cauda equina,* are also capable of producing pain which resembles that of sciatica. In such cases, inquiry into the action of the sphincters of the bladder and rectum may suggest the position of the lesion, and should always be made in patients complaining of sciatica. Skiagraphy of the lower part of the vertebral column may help to disclose disease of that structure. Lumbar puncture may be necessary for diagnosing syphilitic meningitis. In most cases of this kind, however, a careful scrutiny of the lower limb will show that the pain is not limited to the distribution of the sciatic nerve, that the latter is not acutely tender on pressure, that there are atrophy and paresis of certain groups of muscles, some of which are supplied by other branches of the lumbo-sacral plexus, that there are patches of anæsthesia corresponding to root rather than nerve areas, or that the knee-jerk is lost and perhaps the plantar reflex altered in character. It should also be remembered that sciatica is nearly always unilateral, whereas growths or inflammation within the vertebral canal tend to produce signs and symptoms in both legs at a comparatively early stage.

The frequency with which sciatica is diagnosed when some much more serious disease is really present, is sufficient excuse for laying emphasis on the above points, and every practitioner would do well to make it his invariable rule, when faced with a case apparently conforming to the picture of sciatica, to inquire into the action of the sphincters, to inspect carefully and palpate the pelvis and spine, to make a rectal examination, and finally to keep a sharp look-out for signs of present or past malignant disease in other parts of the body. In all cases of neuralgic or neuritic pain the urine should be examined for the presence of sugar.

Anterior Crural Neuralgia and Anterior Cruritis.—Neuralgia in the distribution of the anterior crural nerve is much less common than sciatica, and precautions similar to those just mentioned must be adopted before the diagnosis is made. In this condition, the pain and tenderness involve the front of the thigh as far as the knee, and the knee-jerk is sometimes diminished. In some cases the pain extends along the saphenous branch to the ankle, inner aspect of foot, and big toe. There is often considerable wasting of the quadriceps extensor muscle, which may also exhibit reaction of degeneration. Occasionally the affection is associated with sciatica.

Obturator Neuralgia.—Pain in the distribution of the obturator nerve is rarely of simple neuralgic origin. *Disease of the hip-joint* and *obturator hernia* are two of the conditions which may give rise to it.

Meralgia Paræsthetica is the name given to a variety of pain which is referred to the course of the external cutaneous nerve of the thigh. The relations of this nerve to the psoas muscle and the fascia lata render it liable to stretching or pressure in standing or walking, with the result that the neuralgia is intensified by the maintenance of the erect posture. In certain instances of great obesity, prolonged sitting has been supposed to play a part in producing this type of neuralgic discomfort. There is sometimes a painful pressure-spot just below the anterior iliac spine. A flat-foot is met with not uncommonly in these cases.

Metatarsal Neuralgia, or *Morton's Affection of the Foot.*—This neuralgia is of the paroxysmal type, and is described as dull throbbing pain in the base of the fourth—sometimes of the second—toe, and spreading up the leg. There is tenderness on pressure over the metatarsus. In a certain number of cases the pain is probably related to the wearing of tight or ill-fitting boots, or to the presence of flat-foot.

Calcanodynia is another form of pain liable to occur in neuralgic subjects who are doing much walking. The pain is often bilateral, worse in the evening and at night, and tends to spread from the heel to the base of the toes. There are no objective signs of disease. One patient who consulted me for this condition returned a year or two later with a typical brachial neuralgia. In all obscure cases of painful feet the possibility of a gonorrhœal bursitis or fibrositis should be kept in mind; and also of calcification in the posterior end of the long plantar ligament producing a spine-like projection beneath the os calcis, visible to the x-rays (*Fig.* 185).

Multiple Neuritis.—The lower extremities are often the site of multiple neuritis giving rise to great pain, but the diagnosis is rarely difficult owing to the association of atrophic palsy of the muscles, the electrical reaction of degeneration, dulling of cutaneous sensibility below the knees, and loss of the knee- and ankle-jerks. Perhaps the most characteristic and constant phenomenon in such cases is the presence of marked tenderness of the muscles on pressure. The nerve trunks are sometimes hypersensitive also, but not so constantly as are the muscles below the knees. The pain in multiple neuritis is often acute, worse at night, and aggravated by movement and the pressure of bed-clothes.

Tabes Dorsalis.—The pains of tabes are more often complained of in the legs than in any other part of the body. Unlike the neuralgias, they are usually bilateral and not referred to the distribution of any particular peripheral nerve. The 'lightning' pains are so characteristic that they can hardly be compared with pains of any other origin. Whether trivial and 'niggling,' or so intense as to draw sweat and cries from the most heroic of sufferers, they are always short and lightning-like in duration, often rapidly repeated in the paroxysms, irregularly periodic in their attacks, and fleeting or hovering in their localization. It is a practical point of importance to remember that many patients, when asked if they suffer from pains, emphatically deny it, but readily admit to 'rheumatics,' and then describe in a graphic manner the lightning pains of tabes. The idea of rheumatics is evolved from the fact that these pains are often provoked by changes in the weather. In addition to lightning pains, sufferers from tabes often complain of dull aching or boring pains, which are more continuous and less intermittent than those just described. Tabetic pains may precede all other signs and symptoms of the disease, in which case their diagnosis may be difficult. The following points should be investigated carefully when pains answering to the description given above are complained of: (1) A history of syphilis, congenital or acquired. The writer has known a woman, probably the subject of congenital syphilis, to suffer from lightning pains from early childhood up to forty years of age, when she presented other signs of tabes; (2) The presence or absence of a positive Wassermann serum reaction, though this test may be negative without tabes dorsalis being excluded; (3) The presence or absence of a lymphocytosis in the cerebrospinal fluid; (4) The reaction of the pupils to light; (5) The condition of the knee- and ankle-jerks; (6) The presence of deep and superficial analgesia over the legs; (7) A history of gastric crises; and (8) The condition of the sphincter vesicæ. Particular attention is drawn to a valuable sign of tabes which is not referred to so commonly as are Argyll Robertson pupils and absent knee-jerks, i.e., the impaired pain-sensibility in the calf and other muscles when they are squeezed.

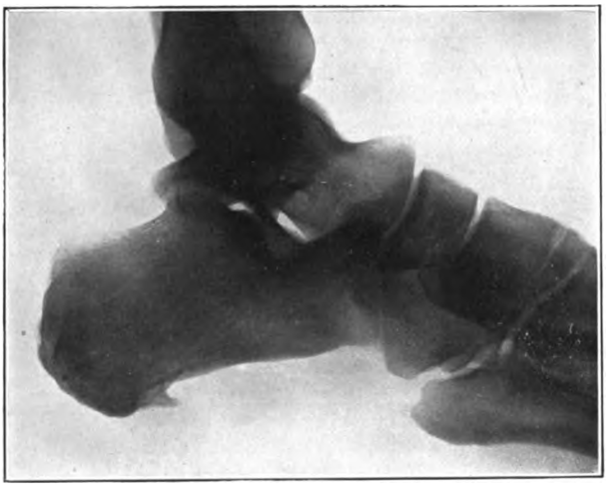


Fig. 185.—Skiagram of a 'spine' on the under surface of the os calcis, causing painful heel. (*By Dr. Lindsay Locke.*)

Acroparæsthesia.—(See p. 444.)

2. Pain in connection with Disturbances of the Circulation.

Intermittent Claudication.—This term is applied to a condition the pathology of which is still obscure, but which certainly depends on an insufficient blood-supply to the muscles of the lower extremities when they are called into activity during locomotion. It may lead eventually to gangrene. The malady occurs chiefly in men over forty years of age, and particularly in those who have indulged freely in tobacco, who have contracted syphilis, or who have thrown strain upon their legs over a long period of time. The patient complains of pain in one or both legs, generally in the calf muscles, coming on after walking a certain distance, and disappearing with rest. The pain becomes so intolerable that he is obliged to stand or sit still until it passes off. As time goes on the distance he can walk in comfort becomes progressively shorter. Examination of the affected limbs reveals nothing obvious; they are well nourished, powerful, and normal in regard to sensation and reflexes. Probably, however, the observer will fail to detect pulsation in the arteries of the foot, and perhaps he may not feel the popliteal artery behind the knee-joint. The femoral artery can usually be felt to pulsate in a normal manner. After the exertion of walking, the foot may appear unduly pale. With rest, the returning flush of normal colour spreads gradually over its surface. In several cases of this kind the writer has noticed myokymia of the calf muscles; that is to say, slow worm-like contractions of individual muscle bundles without any movement of the foot. The ankle-jerk may be diminished or absent. The condition is not very uncommon, and its diagnosis is not difficult if the characteristic history of pain coming on during the act of walking is borne in mind and leads to the search for the signs referred to above. The importance of its recognition needs no emphasis in view of its tendency to go on to gangrene.

Raynaud's Disease.—The pain attendant on the local syncope and local asphyxia which characterize this disease may be severe, but the diagnosis is obvious owing to the onset of symmetrical pallor or cyanosis of the toes preceding the acutely painful stage (see GANGRENE, p. 255). The hands are nearly always affected at the same time.

Erythromelalgia.—In this condition, which may affect various parts of the body, but which is seen most commonly in the feet, pain may precede any other phenomenon. The pain is more or less continuous, with paroxysmal exacerbations, and it is aggravated by the dependent position of the limb as well as by warm applications. The raising of the foot to, or above, the level of the body, and the application of cold, are attended by alleviation. Local patches of cutaneous flushing follow, or sometimes precede, the pain, and are often found about the ball of the big toe or along one edge of the foot. These patches are generally rose-pink, but may become purplish-red in severe paroxysms. The local temperature is raised, and pulsation of the vessels may be observed. Superficial and deep tenderness is also present, but no changes in the reflexes are noted. In long-standing cases a certain amount of œdema results. Erythromelalgia occurs in persons who are apparently healthy in other respects; on the other hand, it may be an early symptom of, or be associated with, some disease of the spinal cord, such as disseminated sclerosis, tabes dorsalis, or syringomyelia.

The following table is drawn up with a view to summarizing the chief points in the differential diagnosis of intermittent claudication, Raynaud's disease, and erythromelalgia:

	INTERMITTENT CLAUDICATION	RAYNAUD'S DISEASE	ERYTHROMELALGIA
Age	40 and over	All ages	20 to 60
Sex	Males more than females	Females more than males	Males more than females
Site	As a rule symmetrical in calf muscles	Symmetrical in toes	Asymmetrical in feet, rarely bilateral
Pain	Onset while walking	During syncopal phase or absent	Precedes vasomotor phenomena
		Unaffected by position	Aggravated by dependent posture
	Worse in cold weather	May be excited by cold	Cold beneficial
	Only with exercise	Paroxysmal	More or less continuous, with exacerbations
	No sensory change	Anæsthesia and analgesia during paroxysm	Superficial and deep tenderness
Vasomotor changes	No change or slightly pale	Pallor and lividity	Pink to purplish flush
	Absence of pulsation in arteries. Feet sometimes cold	Ischæmia and local cold	Increased pulsation and local heat
	Gangrene occasional	Gangrene common	Gangrene rare
Associations	Arteriosclerosis Tobacco Syphilis	General vasomotor disturbances	Functional and organic disease of the central nervous system

3. **Referred Pain in Visceral Disease.**—In the lower extremity the referred pain of visceral disease is not recognized so often as is that of cardiac disease in the upper extremity. Disease of the *rectum*, *bladder*, *prostate*, or *uterus* may, however, give rise to pain and cutaneous tenderness, chiefly in the fifth lumbar and sacral areas. Head quotes a patient who suffered from prostatitis and whose complaint was as follows: "My life is a burden, for I cannot stand owing to the pain in the soles of my feet, I cannot walk owing to the pain in my calves, or sit on account of the pain over the ischial tuberosities and in the perineum, or even lie owing to the pain in my loins and side." A careful examination of the abdominal and pelvic viscera is necessary, therefore, in all cases of pain referred to the legs without obvious local cause. *E. Farquhar Buzzard.*

PAIN IN THE EXTREMITY (UPPER).—Pain in some part or other of the upper extremity is a common complaint, and one for which relief is often sought. This article makes no pretence to deal with the diagnosis of cases in which there is some obvious local source of pain, such as acute arthritis or a tumour, but is intended to serve as a guide for the diagnosis of cases in which the pain is more obscure in origin.

First, it is always essential to inquire into the character of the pain, its exact site, its duration, and, if paroxysmal, its usual time of onset, its relation to movement, rest, etc. Secondly, a careful examination must be made, not only of the offending limb, but of the functions of various organs and of the nervous system in particular. It must be remembered that the arm is innervated by branches of the brachial plexus, and that the latter is made up of nerve fibres derived from the fifth cervical to the second dorsal spinal segments through their corresponding roots. Consequently, complete examination may necessitate an investigation of the spinal functions, and an inquiry into the condition of the cervical vertebral column and the cervical meninges; it may even be desirable to take a skiagram of the neck or to make a lumbar puncture for the purpose of a correct diagnosis. Attention may be drawn especially to the fact that pain in any situation may be a forerunner, the first symptom of a nervous or spinal lesion which ultimately leads to more serious disorders of function, such as paralysis, loss of sensibility, and alteration of reflexes.

The following are various pathological conditions of which pain in the arm is often a prominent symptom:—

Brachial Neuralgia.—This, like neuralgia in other parts, is characterized by pain and tenderness in the distribution of one or more nerves. The pain may be referred to the course of all the branches of the brachial plexus, but sometimes is limited to that of one or two nerves, such as the ulnar, musculospiral, or internal cutaneous. It may occur only in paroxysms, but more commonly there is a constant aching discomfort, with occasional severe exacerbations excited by exertion, cold, or mental worry. The patient is generally glad to rest the limb or to carry it in a sling, in order to avoid the more acute attacks; on the other hand, the continuous aching drives him to find temporary relief in frequent changes of position. Pressure over the affected nerves is accompanied by tenderness, especially over the brachial plexus in the posterior triangle of the neck, over the musculospiral as it winds round the humerus, and over the ulnar along its superficial course in the region of the elbow. The tenderness so produced may be associated with pain or tingling referred to the more peripheral course of the nerve. The skin may be hyperæsthetic and show vasomotor changes in the way of flushing or hyperidrosis.

In making a diagnosis of brachial neuralgia it is desirable to seek for some cause to which it can be ascribed, such as a *rheumatic* or *gouty diathesis*, or a history of some preceding toxic condition, such as *influenza*, *malaria*, or *alcoholism*. In some cases no satisfactory explanation beyond unusual worry or work in a neuropathic individual is forthcoming. The urine should be examined for sugar, as neuralgia is sometimes of *diabetic* origin. The presence of muscular atrophy or anæsthesia removes the case from the category of neuralgia, and the diagnosis of neuritis or of some more gross organic affection must be substituted. On the other hand, it must not be forgotten that a *cervical rib* (*Fig.* 187) may produce many of the symptoms of brachial neuralgia without any definite muscular atrophy or sensory loss. In contradistinction to some of the conditions about to be described, brachial neuralgia is practically always unilateral.

Brachial neuralgia may be diagnosed, therefore, if there is pain and tenderness in the distribution of the brachial plexus without paralysis or sensory loss, and if no gross lesion can be found to account for the symptoms. So-called *muscular rheumatism* differs from brachial neuralgia in that the pain is generally less acute and the points of local tenderness are to be found over muscular insertions rather than over the nerve trunks. At the same time it must be admitted that the muscles as well as the nerves are hypersensitive in severe cases of neuralgia.

Brachial Neuritis.—When muscular atrophy and sensory loss are found in addition to pain and tenderness, the condition must be regarded as one of neuritis. Unilateral brachial neuritis is very uncommon except as a result of some gross lesion, such as pressure on, or irritation of, the nerve-trunks. Bilateral brachial neuritis is common enough, but is then a part of a multiple peripheral neuritis due to alcohol, arsenic, lead, diabetes, etc., in which the lower extremities also are generally involved.

Before making a diagnosis of one-sided brachial neuritis, careful search must be made for evidence of such conditions as *cervical rib*, *tumour in the posterior triangle of the neck*, *glands in the axilla*, *aneurysm of the subclavian artery*, *malignant disease* or *caries of the cervical vertebræ*, *cervical pachymeningitis*, *spinal tumour*, or *spinal gliosis*. *Neuromata* or *fibroneuromata* are generally widely distributed about the peripheral nerves, but cases have been recorded in which they have been limited to the brachial plexus and have given rise to a brachial neuralgia or brachial neuritis. Such tumours may be so small as easily to escape observation unless looked for. *Adiposis dolorosa* (p. 410) is another rare condition which may give rise to neuralgic pain in the arm, but it is not limited to one limb.

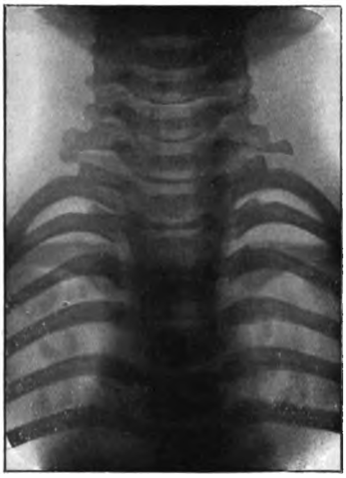


Fig. 186.—Skiagraph showing cervical ribs in the case of a child.
(*Skiagram by Dr. Gilbert Scott.*)

Cervical Ribs.—A supernumerary seventh cervical rib, unilateral or bilateral, is a frequent congenital abnormality. In a small proportion of cases it may give rise to symptoms, especially in adults who use their arms and hands continually in the course of their employment. Women suffer more often than men. Pain radiating from the root of the neck to the tips of the fingers, more often than not along the ulnar border of the arm, is usually the first, and may be the only, symptom. The pain is of an aching or dull boring character, and is much influenced by rest and position. For instance, if a woman, who has been suffering much when at work, takes a holiday, and ceases to use her arms for scrubbing, lifting, etc., she may lose the pain altogether until she resumes her occupation.

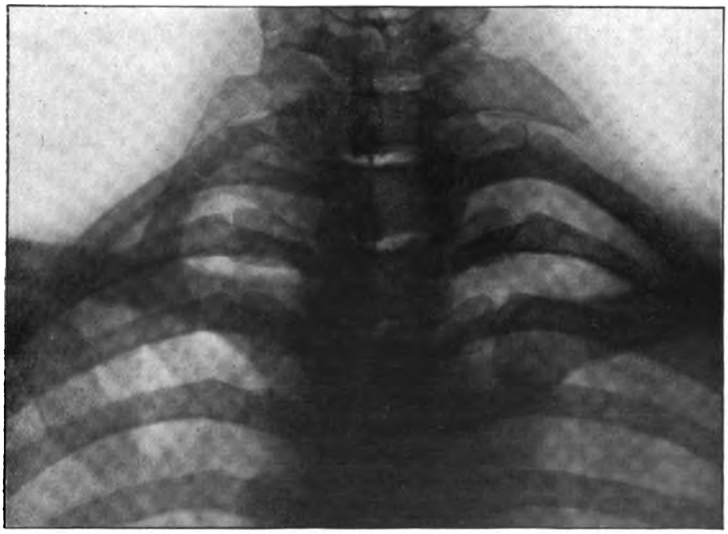


Fig. 187.—Skiagram of cervical ribs in an adult. On the left side of the skiagram the rib is fully developed; on the right side only a part is bony, so that it forms a false or buttress rib continued by a fibrous cord, which does not show with the *x*-rays.
(*Skiagram by Dr. S. Gilbert Scott.*)

Similarly the pain is worse at night after a day's work, and may be influenced favourably by keeping the arm in certain positions. Lying in bed with the hand behind the head is a favourable attitude in many cases. Very occasionally the pain spreads into the scapular region along the course of the suprascapular nerve. There is rarely any tenderness along the peripheral parts of the nerves, but pressure in the posterior triangle of the neck, just above the inner part of the clavicle, may give rise to pain radiating down the arm.

In addition to pain there may be disturbances of motor, sensory, and vasomotor origin. Atrophic palsy of the intrinsic hand muscles and of the flexor muscles in the forearm are the common motor disturbances, and may lead to deformities such as CLAW-HAND (p. 109). Anæsthesia along the ulnar border of the forearm, and perhaps extending on to the inner fingers, is sometimes observed. In one bilateral case the writer has observed intense vasomotor disturbances without definite muscular atrophy or sensory loss. Both hands were the seat of a painful cyanosis involving the fingers, and almost amounting to the condition seen in cases of Raynaud's disease. Sometimes there is a diminution in the radial pulse on the affected side. The diagnosis of cervical rib or ribs depends on the use of the x-rays (*Figs.* 186, 187) to reveal their presence, but it must be borne in mind that the pressure on the trunk or trunks of the brachial plexus is usually exerted by a fibrous band passing from the tip of the cervical rib to the first dorsal rib, and that therefore the size of the rib shown by skiagraphy affords no guide as to the importance of its effect. The most rudimentary rib is as important from this point of view as one which is fully developed.

Acroparæsthesia.—This term is applied to a fairly common complaint, usually made by women between thirty-five and fifty-five years of age, who are continually using their hands, and especially by those whose hands are frequently immersed in waters of different temperatures. Charwomen, domestic servants, needlewomen, and washerwomen are particularly liable to suffer. Many of the victims indulge to a moderate extent in spirit-drinking. They complain of a burning pain, associated with tingling and numbness, in the fingers and palms of their hands. It is noticed chiefly in the latter part of the day after work is over, and becomes intensified when they are warm in bed. In the early morning their fingers are numb and clumsy, but the discomfort passes off while they are at work, only to return again towards evening. As a rule there is little to see on examination, but there may be redness or pallor of the affected parts, associated with a subjective feeling of heat and swelling. Sensibility is unimpaired if allowance is made for the cutaneous thickening usually present in persons whose hands are much exposed to moisture and friction. There is no definite palsy or muscular atrophy. Acroparæsthesia as a rule affects both hands, and very occasionally is accompanied by a similar condition in the feet.

Similar paræsthesiæ are sometimes complained of by patients suffering from *tabes dorsalis*, but in those cases the pains are of the lightning character, and never limited to the hands. Other tabetic signs, such as Argyll Robertson pupils, ulnar analgesia, impaired sense of position, and absence of tendon-jerks, serve to make a diagnosis. In the early stages of *subacute combined degeneration of the spinal cord*, paræsthesiæ, sometimes of a markedly painful character, are referred to the hands and feet. The presence of some ataxia or spastic paraplegia, with increased tendon-jerks and extensor plantar reflexes, differentiate this disease from the ordinary acroparæsthesiæ.

Radicular Pain.—Under this title may be included all pains in the arm which radiate through the peripheral distribution of the posterior spinal roots from the fifth cervical to the second dorsal. These pains extend from the neck towards the periphery of the limb, and are usually of a sharp, lancinating type. In the large majority of cases they are produced by some gross morbid process involving the roots within the spinal canal or in their course through the intervertebral foramina. The morbid processes most commonly responsible are *intravertebral tumour*, *cervical pachymeningitis*, *cervical caries*, and *malignant disease of the cervical vertebræ*. In all these conditions the radicular pain may precede all other symptoms, with the result that the diagnosis is often difficult and sometimes impossible until further phenomena develop. The pain is occasionally unilateral, more often bilateral; there may be tenderness on pressure over the vertebræ, especially in the case of vertebral caries or malignant disease. Movements of the neck will intensify the pain in the latter conditions. The diagnosis is arrived at by careful attention to the following points: (1) Evidence of deformity, rigidity or tenderness of the cervical vertebræ, supplemented by an x-ray examination; (2) The presence of other root symptoms, such as localized atrophic palsy, anæsthesia, and loss of tendon-jerks in the arms; (3) Evidence of pressure on the spinal cord, producing spastic paralysis of the trunk and lower limbs, together with anæsthesia, loss of abdominal reflexes, increased knee-jerk, ankle-clonus, and extensor plantar reflexes; (4) The occurrence of oculo-pupillary phenomena when the eighth cervical and first dorsal roots are involved; and (5) The condition of the cerebrospinal fluid obtained by lumbar puncture (p. 304).

In addition to the gross extrinsic processes affecting the spinal roots, there are other cases in which a spinal root is the site of an intrinsic inflammatory or vascular lesion. *Herpes zoster* is a common result of such a lesion, and may be found in the peripheral distribution of any of the posterior roots which go to form the brachial plexus. Pain in the upper extremity often precedes the eruption, and post-herpetic neuralgia is sometimes long persistent and associated with marked hyperæsthesia in the corresponding root area. Uniradicular pain, followed by atrophy of the muscles supplied by the efferent root fibres and by sensory loss in the region innervated by the afferent fibres, with or without the development of an herpetic rash, also occurs in rare instances as the result of an inflammatory or vascular lesion of the spinal nerve in the neighbourhood of the posterior root ganglion.

Referred Pain in Visceral Disease.—In disease of the heart and aorta, especially with syphilitic disease of the aortic valves, or with atheroma or aneurysm of the first few inches of the aorta, attacks of pain in the left arm are often complained of. These may be confined to the arm, or may be associated with fully developed angina pectoris (p. 433). The pain is radicular in distribution, referred to the first and second dorsal root areas—the ulnar border of the arm—sometimes extending into the little finger. During the attacks cutaneous hyperæsthesia may be present over the same areas. In all cases of paroxysmal pain referred to the left arm a very careful examination of the thoracic viscera is therefore indicated.

Occupation Neuroses.—The upper limb is the common site of occupation neuroses—termed writer's cramp, typist's cramp, and so on, according to whether it has to do with writing, typing, needlework, telegraphy, hair-cutting, etc. These neuroses are mainly characterized by some form of muscular spasm, but pain of a cramp-like character is a frequent accompaniment of the spasm. The diagnosis is easy, because careful inquiry will elicit the fact that the pain and spasm are evoked by the employment of the limb in a particular occupation, and that other manipulations involving the use of the same muscles may be carried out with impunity. The acute pain associated with the spasm may be followed by a dull aching for some hours after the occupation has been indulged in.

Finally, the term **Psychalgia** may be applied to pain referred to the arm, as well as to other parts of the body, by patients whose nervous and mental resistance is undermined or exhausted. Neurasthenic pain of this kind is rarely limited to the arm; it is referred more commonly to various parts of the head and to particular regions along the course of the vertebral column.

E. Farquhar Buzzard.

PAIN IN THE EYE is not by itself pathognomonic of any particular lesion; but it may be complained of under very diverse circumstances, which may be ranged into the following groups:—

1. **Pain associated with Visible Inflammatory Changes,** due to—

Foreign body
Entropion
Conjunctivitis
Ulceration of the cornea
Interstitial keratitis
Iritis
Glaucoma
Ocular herpes.

The differential diagnosis between these is discussed in the article on EYE, ACUTE INFLAMMATION OF (p. 231).

2. **Pain without Visible Changes in the Eyeball, but with Acute Loss of Sight** in one eye only, in both eyes together, or in one eye after the other: Retrobulbar neuritis.

The pain is generally referred to the back rather than to the front of the eye. The diagnosis is suggested at once if considerable loss of sight comes on acutely in an eye which on examination proves not to be affected by glaucoma, intra-ocular hæmorrhage, detachment of the retina, or any visible or palpable lesion, especially if the degree of vision power waxes and wanes, owing to varying degrees of engorgement of the vessels in the optic nerve where it is inflamed between the eyeball and the brain. After a day or two, or a few days the pain may disappear and sight return to normal; on the other hand, in severer cases, the inflammation in the optic nerve may come forward to the back of the eyeball and become visible as optic neuritis (*Plate XIX*, *Fig. k*, p. 416). The cause of the mischief may be difficult to determine; often it remains obscure; sometimes it is traced successfully to plumbism (p. 34), syphilis, or to some acute infection such as influenza.

3. **Pain without Inflammation and without Blindness,** but associated with errors of refraction: Eyestrain.

The commonest cause of eyestrain is some error of refraction, especially hypermetropia, astigmatism, or presbyopia, and it arises mainly in persons whose occupation entails much reading of small print, fine needlework, or close attention to minute details near to the eyes; or in those whose work has to be carried on in too dim or too strong a light —especially electric or strong sunshine. It is not so much the big degrees of error of refraction that cause the mischief—for these are generally so obvious to the patient that they have been corrected already by appropriate glasses; it is rather the minor or even quite small degrees of hypermetropia or astigmatism that are responsible, for these have very likely not by themselves led the patient to seek ocular advice; and they need considerable care in their detection even at the hands of a skilled ophthalmic surgeon The patient may consider that he has exceptionally good sight, especially in the case of minor degrees of hypermetropia, which may none the less cause not only severe pains in the eyes, but also serious headaches and attacks simulating migraine. Frequently it is only when the patient has become tired from excessive work that the strain of accommodating for near objects begins to tell; and the symptoms may be attributed to overwork when this is but partly true; or presbyopia may produce pain in eyes which were not subject to eyestrain when the faculty of accommodation was more lissom in youth, notwithstanding the error of refraction. Pain in the eyes from eyestrain becomes more common as age advances, and some elderly persons are scarcely able to read or work at all on this account, even when glasses have been prescribed.

On the other hand, work carried out under exceptional circumstances of light or closeness may cause eyestrain even in those whose ocular refraction is normal or fully compensated by glasses; microscopists are apt to suffer in this way for instance, or those who are exposed to the glare of sunshine upon snow; and so on. The circumstances of the case are likely to suggest the diagnosis.

4. **Pain in the Eyes due to Febrile or other Constitutional Causes.**—The most familiar example of cases which come under this heading is afforded, perhaps, by *influenza.* The pain is generally referred rather to the backs of the eyeballs than to the eyes themselves, but nevertheless the complaint is one of pain in the eyes. The trouble occurs both as an early symptom of the disease and as a sequela when the fever has subsided. The diagnosis is made from the course of the pyrexia and the general symptoms; it depends little upon whether there is pain at the back of the eyes or not. But influenza should not be regarded as certain without bacteriological confirmation, for it is usually guessed at rather than diagnosed—and often wrongly. In a similar way, pain in the eyes may form a part of the general clinical picture in many other fevers, notably small-pox, typhus, typhoid fever, measles, secondary syphilis, and malaria. The diagnosis is influenced very little by the fact that the patient complains about his eyes, except when there is coryza as well as pain, for instance in the early stages of measles. In other conditions there is PHOTOPHOBIA (p. 524) rather than pain in the eyes, and reference may be made to the article upon that symptom.

Herbert French.

PAIN IN THE FACE.—The distinction between pain in the face and pain in the head, though to some extent artificial, is sufficiently marked in most instances; the latter, with its diagnostic significance, is discussed under HEADACHE (p. 293). There are certain etiological points, however, at which faceache and headache overlap; for example, the supraorbital pain and the headache which may both originate from ocular errors of refraction. Pain in the face, as elsewhere, may be due to very obvious causes, which need no discussion, such as an inflamed parotid gland, a gumboil, or an acute conjunctivitis. On the other hand, pain in the face may be complained of when superficial, and perhaps minute, examination fails to discover an adequate basis. Guidance towards the correct diagnosis of such cases may be obtained by a consideration of the course, signs, and symptoms of the following clinical types of facial pain.

Major Trigeminal Neuralgia (tic douloureux or epileptiform neuralgia) may be regarded as a distinct disease, owing to the general similarity of one case to another. Its pathology is unknown, but in each case the pain is attributed in its early stages to some local defect, such as a carious tooth, and many sound, as well as many diseased, teeth are removed in a vain endeavour to arrest the malady. Beginning usually after thirty-five years of age, tic douloureux is characterized by paroxysms of acute pain in the distribution of one or more of the divisions of the trigeminal nerve, usually of one side only. The intervals between the paroxysms vary from seconds to months, and may be influenced in their length by many factors, such as the general state of health, mental worry, and exposure to cold. The intervals tend to become shorter and the paroxysms more severe and more extensive in their distribution. The pain is described as beginning in spots beneath the skin, and radiating along the peripheral branches of the nerve. These spots correspond to points where the nerve bundles penetrate the deeper tissues to reach the superficial structures, and may be recognized as places pressure upon which is particularly liable to start an attack. In severe cases, the lightest touch, a breath of wind, attempts at articulation or mastication, and even the act of defæcation may be sufficient to provoke an agonizing spasm in which the violent reflex contraction of the muscles of the corresponding side of the face affords some evidence of the suffering endured. During the paroxysm the patient may endeavour to obtain relief by firm pressure with his hand over the starting-point of the pain. The attack may be accompanied by cutaneous flushing, photophobia, lachrymation, and salivation, as well as by a subjective sensation of swelling in the affected tissues. When the tongue is affected a metallic taste is sometimes described by the sufferer. Trophic changes in the hair and skin are also observed as a result of repeated attacks. The diagnosis of major neuralgia depends chiefly on the following points: (1) The age of onset; (2) The absence of relief or only temporary alleviation, afforded by removal of possible exciting causes, such as defective teeth; (3) The presence of definite starting-points of the pain corresponding to exits of branches of the fifth cranial nerve, and the spread of the pain along the corresponding nervous paths; (4) The paroxysmal character of the pain, its intense severity, and its unilateral distribution; (5) The excitability of the attacks by peripheral stimuli; and (6) The various reflex, vasomotor, secretory, and trophic phenomena to which the attacks of pain give rise. From a practical standpoint the most important task in diagnosis is to discriminate between cases of idiopathic major neuralgia and those which belong to the next group.

Trigeminal Neuralgia due to Organic Lesion of the Nerve or its Roots.—This form of neuralgia may simulate tic douloureux in every particular, and its diagnosis can be made only by careful systematic examination of the patient, with the possibility of an organic lesion being the source of pain before the physician's mind. *Tumours at the base of the brain* in the middle fossa, *tumours growing from the base of the skull* in the neighbourhood of the foramen ovale and foramen rotundum, as well as *tumours of the cranial nerves* themselves, are amongst the causes of trigeminal neuralgia. *Gummatous meningitis* and *gummatous periostitis* may be mentioned in the same connection. In every case of trigeminal neuralgia, therefore, headache and vomiting should be inquired after, and optic neuritis looked for. Examination of the functions of each cranial nerve must be carried out, and in particular those of the fifth nerve carefully tested. Any impairment of sensibility in the cutaneous territory of this nerve must be regarded as evidence that the case is not one of idiopathic neuralgia, and the same may be said when there is impaired motor power in the muscles of mastication. In several cases of severe trigeminal neuralgia I have found atrophic palsy of the masseter and temporal muscles on the same side, with slight anæsthesia on the face, and these cases have always proved to be instances of growth involving the structures at the base of the skull. In one patient the neoplasm originated in the splenomaxillary fossa.

Trigeminal neuralgia may also occur as the result of intrinsic disease of the Gasserian ganglion, e.g., in cases of *herpes zoster*. This condition is fairly common in the distribution of the first division of the trigeminus, much less common in that of the second and third. The pain usually precedes the herpetic eruption by some days, and is associated with constitutional malaise and sometimes with pyrexia, two important points in diagnosis. The latter becomes clear with the development of the rash, but even then it is necessary to bear in mind the possibility that the Gasserian ganglion may be affected by gross external disease, such as neoplasm or gumma, or an extension of bony disease. In persons over fifty years of age it is found frequently that pain of a neuralgic character persists after the herpes has disappeared, and may last for months and even years. Careful examination may discover cutaneous marks corresponding to the site of the previous vesicular eruption.

Neuralgia Minor.—Under this heading may be classed the varieties of facial pain which are secondary to disease of various local structures, such as the teeth, the eye, the ear, the nose, and the tongue. The pain can be distinguished by certain features as belonging to one or other of two types. The first is a true neuralgia, that is to say a pain which is distributed along the course of one or more divisions of the trigeminal nerve, usually starting in the neighbourhood of the diseased structure. The second is a visceral pain, referred to some spot which may be at a distance from the disease, and which is usually the site of superficial hyperæsthesia or tenderness.

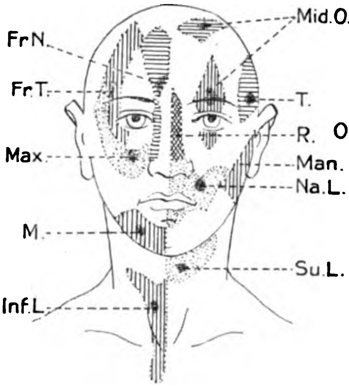


Fig. 188.

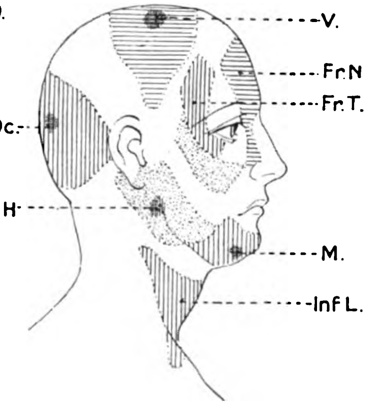


Fig. 189.

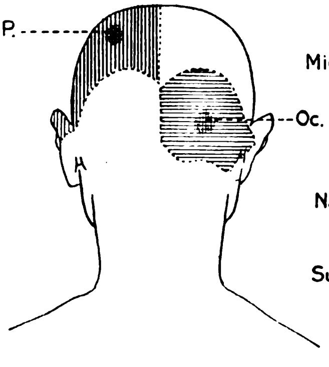


Fig. 190.

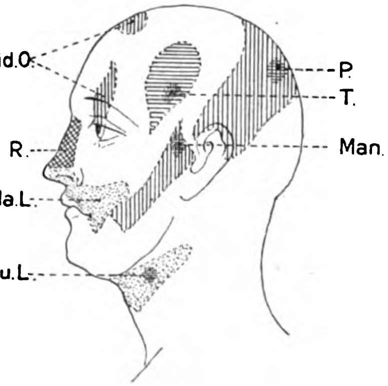


Fig. 191.

SENSORY AREAS OF THE FACE, HEAD, AND NECK.

Fr. N. Fronto-nasal; Fr. T. Fronto-temporal; H. Hyoid; Inf. L. Inferior laryngeal; M. Mental; Man. Mandibular; Max. Maxillary; Mid. O. Mid-orbital; Na. L. Naso-labial; Oc. Occipital; P. Parietal; R. Rostral; Su. L. Superior laryngeal; T. Temporal; V. Vertical.

The history of a *decaying tooth* affords an example of how these types of pain may arise. In the early stages of caries the pain is limited to the tooth. With inflammation and destruction of the pulp, pain is referred to a segmental area on the surface of the face varying with the particular tooth implicated; thus a diseased canine tooth is associated with pain and tenderness in the naso-labial area (*Fig.* 188, Na.L). Finally, after the pulp is dead, local suppuration may start a neuralgia which may not only spread along the nerve branch which supplies the tooth socket, but may extend into neighbouring branches and into other divisions of the trigeminal nerve. The maximal points in the segmental areas referred to are shown in the accompanying diagrams, and the general relationship between individual teeth and their segmental areas may be described as follows :—

UPPER JAW.

Incisors	Fronto-nasal	1st molar	Maxillary
Canine	Naso-labial	2nd „	Mandibular
1st bicuspid	„ „	3rd „	„
2nd „	Temporal or maxillary		

LOWER JAW.

Incisors	Mental	1st molar	Hyoid
Canine	„	2nd „	„
1st bicuspid	„	3rd „	Hyoid or superior laryngeal.
2nd „	Doubtful		

The value of this knowledge in relation to diagnosis lies in the fact that pain, with tenderness, referred to any one of these segmental areas should lead the observer to seek for its cause in disease of the corresponding viscus.

The headache, sometimes called neuralgia, which results from *errors of refraction*, especially astigmatism, is referred to the mid-orbital area, where superficial tenderness may often be discovered on examination. This form of pain comes on in the morning as soon as the eyes are opened, and is intensified by reading or sewing. It disappears under the use of atropine, and wears off of itself if the eyes are not used for near work. Occasionally it takes on a paroxysmal character without any particular relationship to the use of the eyes. In *iritis* and *glaucoma*, referred pain may be intense, and it is usually situated in the temporal and maxillary segmental areas as well as in the eyeball itself. The occurrence of referred pain in chronic glaucoma without pain in the eyeball is a point which may be of great diagnostic importance, as it may draw attention to the unsuspected ocular disease.

In *ear disease* the hyoid area is that to which pain is referred and in which hyperæsthesia of the skin may sometimes be found. In the more severe types of disease, such as suppuration in the middle ear, the pain may also be referred to the vertical and temporal areas.

Lesions of the tongue may produce, in addition to local pain in the organ itself, referred pain in three other areas : the mental area when the disease affects the anterior portion of the tongue ; the hyoid area when the lateral portion is involved ; and the occipital area when the dorsum is the site of the lesion.

In inflammatory affections of the *nose* and *frontal sinuses*, pain is referred to the fronto-nasal and mid-orbital areas on the forehead.

The various forms of pain in the head associated with *disease of the thoracic* and *abdominal organs* are discussed under HEADACHE (p. 293), and the same article deals with the aches which accompany general constitutional diseases.

In *tabes dorsalis* pains are sometimes described in the face, and have the same characteristics as those in other parts of the body. They are paroxysmal, sudden, severe, and lightning-like. They are rarely limited to the face. They may be accompanied by a more continuous dull, boring kind of pain. The diagnosis is easy if a systematic examination of the nervous system is carried out.

Pseudo-neuralgias, or psychalgias, which are complained of by hysterical and neurasthenic individuals, are vague in their distribution, not limited to the trigeminal area, and often bilateral. They tend to disappear when attention is drawn in other directions, and are less intense during eating and talking. They are diagnosed by the method of exclusion of other causes.

E. Farquhar Buzzard.

PAIN IN THE FOOT.—(See PAIN IN THE EXTREMITY, LOWER, p. 438.)

PAIN IN THE FOREARM.—(See PAIN IN THE EXTREMITY, UPPER, p. 442.)

PAIN, GIRDLE.—(See GIRDLE PAIN, p. 260.)

PAIN IN THE HAND.—(See PAIN IN THE EXTREMITY, UPPER, p. 442.)

PAIN IN THE HEAD.—(See HEADACHE, p. 293.)

PAIN IN THE HYPOCHONDRIUM (LEFT).—Pain in the left hypochondrium may proceed from :—

The Stomach.—Any painful condition of the stomach may cause pain to be felt below the left costal margin. In particular, a new growth or an ulcer towards the cardiac end may produce it. For the differential diagnosis, see INDIGESTION, p. 315, and PAIN IN THE EPIGASTRIUM, p. 436. Flatulent distention of the fundus may also be a cause, which can be diagnosed by the fact that the pain disappears on eructation.

The Gall-Bladder.—In cases of cholelithiasis the pain is sometimes referred to the left hypochondrium (see PAIN IN THE EPIGASTRIUM, p. 436).

The Spleen.—Some enlargements of the spleen are painful (see SPLEEN, ENLARGEMENT OF, p. 628) ; or the pain may be caused by perisplenitis, in which case a friction-sound can sometimes be heard on auscultation over it.

The Left Kidney.—Stone in the left kidney may cause pain which has the characters described in the section on pain in the right hypochondrium (see below). A movable left kidney is rarely a cause of pain. A perinephric abscess may cause pain, as it does in the right hypochondrium (see p. 451).

The Colon.—A new growth in the splenic flexure of the colon, or obstruction of it lower down, may cause pain in the left hypochondrium. In the former case a tumour can usually be felt on bimanual palpation ; in the latter, signs of chronic obstruction will be present (see CONSTIPATION, p. 121). Apart from growth, a mere *accumulation of fæces* in the transverse and descending colon may cause a feeling of pain and weight in the left hypochondrium. The disappearance of the pain after the administration of a few large enemata will establish the diagnosis.

Pleurisy, Intercostal Neuralgia, and **Herpes Zoster** may all cause pain in the left hypochondrium. In the first of these a friction-sound will be heard ; in intercostal neuralgia there will be tender points over the course of the intercostal nerve. In the case of herpes, the cause of the pain will be cleared up by the appearance of the eruption, but pain may persist long after this has disappeared.

Subdiaphragmatic Abscess.—(See p. 451.)

Robert Hutchison.

PAIN IN THE HYPOCHONDRIUM (RIGHT).—The differential diagnosis of the cause of pain in the right hypochondrium is often a matter of great difficulty, or even of impossibility, as it may proceed from any of the following organs : (1) Liver and gall-bladder, (2) Duodenum, (3) Head of the pancreas, (4) Right kidney, (5) Appendix vermiformis, (6) Colon, (7) Uterine appendages. Intrathoracic disease, affections of the spine or chest wall, and subdiaphragmatic abscess may also cause pain in this situation. The diagnosis is rendered still more difficult by the fact that disease may easily be present in more than one of these situations at the same time.

Liver.—Various forms of enlargement of the liver are apt to be attended by pain in the right hypochondrium, e.g., hepatitis, passive congestion, hepatic abscess, and carcinoma (see LIVER, ENLARGEMENTS OF THE, p. 366).

Disease of the gall-bladder must also be thought of, e.g., gall-stones, cholecystitis, and carcinoma. In these it will usually be found that there is tenderness on pressure over the gall-bladder, with the characteristic catch in the breath when the patient is asked to take a deep inspiration while the fingers of the observer are pressed in over the organ. In acute cholecystitis there will be pyrexia, and probably rigors.

The pain of *biliary colic* may be felt chiefly in the right hypochondrium, but tends to radiate through to the back and up towards the right shoulder. It may be simulated closely both by the kinking of a movable kidney and by renal colic (see below). When the attacks occur during the night as well as in the day, this is in favour of biliary colic.

It must be noted specially that the absence of jaundice in no way contra-indicates a diagnosis of gall-bladder disease.

Duodenum.—A *duodenal ulcer* may cause deep-seated pain in the right hypochondrium, which usually has the character of hunger pain (p. 271). It must be remembered, however, that pain due to chronic cholecystitis, or appendicitis, may also have this character, and an exact differentiation of them may not be possible without exploration. The pain in duodenal ulcer, however, occurs in more definite attacks with intervals of freedom ; it is often nocturnal, waking the patient in the small hours of the morning.

Duodenal ulcer is commoner in men, disease of the gall-bladder in women, whilst appendicitis may occur with almost equal probability in either sex. The percentage of free HCl in the stomach contents is more persistently high in duodenal ulcer than in either appendix dyspepsia, or gall-stones. A history of melæna, or the presence of occult blood in the fæces, would determine one in favour of ulcer.

Pancreas.—Malignant disease of the pancreas may cause pain in the right hypochondrium. In such a case a deep-seated tumour may be felt, and there is often jaundice along with a distended gall-bladder. On the other hand, when gall-stones lead to jaundice, the gall-bladder is not usually distended (see JAUNDICE, p. 324).

Right Kidney.—A freely *movable right kidney* may, by ureteral kinking or dragging on the bile-duct, cause sudden attacks of pain in the right hypochondrium which may exactly simulate gall-stone colic. Indications of intermittent hydronephrosis should be looked for, e.g. the appearance of a renal tumour, and the occasional discharge of large quantities of urine ; urinary symptoms, however, may be entirely absent. The attacks tend to occur by day, whilst biliary colic often begins in the night.

Stone in the right kidney may cause chronic pain in the right hypochondrium and back. The kidney is often found to be enlarged and tender on bimanual palpation in such a case, but it must be remembered that the urine may furnish no diagnostic indication. The x-rays, however, may make the diagnosis clear (*Fig.* 133, p. 279), although a negative result does not exclude the possibility of stone.

The pain of *renal colic* may be difficult to diagnose during an attack from gall-stone colic, lead colic, or appendicitis, but it begins below the lower ribs and has a characteristic tendency to pass downwards into the groin. It may be attended by vomiting and fever. During or after the attack there may be blood and gravel in the urine ; but it must be remembered that the urine may be heavily loaded with urates after an attack of biliary colic.

Pyelitis may also be the cause. The urine will then furnish diagnostic indications (see PYURIA, p. 574, and BACTERIURIA, p. 69) ; and the kidney may be felt to be enlarged on bimanual palpation. The patient is often a pregnant woman, and the pain may begin acutely, starting in the loin and right hypochondrium and passing downwards towards the iliac fossa and pelvis. There is a high temperature, rigidity of the muscles, and hyperæsthesia both in the loin and in the right side of the abdomen.

A *perinephric abscess* may cause pain in the right hypochondrium and lumbar region. A tumour will be felt, and the loin may be filled out, and there will be the usual signs of deep-seated suppuration.

Appendix.—The pain of chronic appendicitis may be felt chiefly in the right hypochondrium, and, as already remarked, may be of the nature of a hunger pain. Tenderness over McBurney's point should be looked for. When an acute attack of appendicitis simulates gall-stones, it may be of help to remember that indicanuria is common in the former, but is usually absent in the latter.

Colon.—*New growths* in the neighbourhood of the hepatic flexure may cause pain in the right hypochondrium ; but in that case a tumour can usually be felt, and signs of chronic intestinal obstruction are present.

Uterine Appendages.—*Salpingitis*, a *twisted ovarian pedicle*, and a *ruptured extra-uterine gestation*, may all cause pain in the right side of the abdomen which, however, has usually its maximum intensity rather below the hypochondriac region. A careful pelvic examination will usually make the diagnosis clear.

Pleurisy, Intercostal Neuralgia, and **Herpes Zoster** may be causes of pain in the right hypochondrium.

Subdiaphragmatic Abscess.—In this case there will be a history pointing to precedent gastric or duodenal ulcer, appendicitis, or hepatic abscess. The onset of the pain may be sudden or gradual. There will be pyrexia and leucocytosis, pointing to deep-seated suppuration. There is usually an abdominal swelling, which does not move with respiration. The note over this may be tympanitic, from the presence of gas in the abscess, and in that event the coin-sound will be obtained on percussion. There are usually indications of pleurisy at the base of the corresponding lung, but the liver is *not*, as a rule, pushed down. The use of the x-rays may help in locating the abscess ; but the exploring needle should not be used except when the patient is on the operating-table, and one is prepared to open the abscess at once if found. *Robert Hutchison.*

PAIN IN THE ILIAC FOSSA (LEFT).—Although many of the causes of pain complained of mainly or entirely in the left iliac fossa are the same as those which cause similar pain in the right iliac fossa, there are certain differences, as will be seen on comparing the table on p. 454 with the following :—

CAUSES OF PAIN IN THE LEFT ILIAC FOSSA.

1. *Causes of Acute Pain :—*

Acute diverticulitis
Ureteral calculus
Acute ureteritis
Twisted left ovarian cyst pedicle
Salpingitis
Oöphoritis
Pelvic abscess
Retained left testis
Suppurative periostitis of the ilium
Appendicitis (exceptional cases)
Coli bacilluria
Local injury
Stitch
Volvulus of the sigmoid colon
Strangulated retroperitoneal hernia.

2. *Causes of Subacute, Chronic, or Recurrent Pain :—*

Most of the conditions mentioned under Group 1, and also :—

Carcinoma of the sigmoid colon
Carcinoma recti
Massive impaction of fæces
Chronic diverticulitis
Spastic constipation
Psoas abscess
Sacro-iliac joint disease
Tuberculous hip
Osteo-arthritis of the spine
Infective arthritis of the lumbar spine
Herpes zoster
Inflamed iliac glands
Tuberculous iliac glands
Periproctal abscess
Periprostatic abscess
Dysentery
Ulcerative colitis
Aneurysm of the left iliac artery
Tumours of the left iliac bone
Tuberculous left kidney.

Acute Lesions.—What is said on p. 454, et seq., in regard to *ureteral calculus, acute ureteritis, twisted ovarian cyst pedicle, salpingitis, oöphoritis, pelvic abscess, retained testis, suppurative periostitis* of the ilium, *injury* and *stitch*, applies in the case of the left iliac fossa as it does to the right, so that here we need discuss only *acute diverticulitis, appendicitis, coli bacilluria, volvulus of the sigmoid*, and *strangulated retroperitoneal hernia.* Of these, the last two call for immediate operation on account of urgent symptoms of intestinal obstruction—especially persistent constipation and vomiting, which becomes fæculent if operative measures are not adopted soon. The precise nature of the obstruction may not be certain until the abdomen has been opened. Abdominal distention is apt to be general, and there is visible peristalsis of the oblique or transverse type in the case of strangulated retroperitoneal hernia, a rare condition in which a coil of small intestine becomes herniated through the normally small retrosigmoid pouch of peritoneum; whereas in the case of sigmoid volvulus the distention is at first much more marked in the left iliac fossa, before general colonic dilatation with vertical peristaltic waves appear. In either case the abdominal wall remains supple as a rule until, if the case should be left unoperated upon, general peritonitis supervenes.

Coli bacilluria is much less commonly a cause of pain in the left iliac fossa than it is of corresponding pain on the right side (p. 455) ; and when it does cause left-sided pain, it nearly always causes an even worse pain on the right side also. It is a clinical fact that coli bacilluria affects the right kidney and ureter very much more commonly than the left, especially perhaps in pregnant women, in whom its incidence on the right side first or solely is an almost constant rule ; why this should be so is not clear, although various theories have been put forward to account for it.

Appendicitis is perhaps almost the last thing that occurs to one as the cause of acute pain referred entirely to the left iliac fossa, just as it is the first thing in one's mind when the pains are on the right side ; but it should not be omitted altogether from consideration : first, because in some cases, in which the vermiform appendix is very long, and inflammation starts at its tip and spreads to the left, as it sometimes does, the symptoms and even the swelling may be to the left of the middle line instead of in the right iliac fossa as usual ; secondly, because in a few cases pains produced on one side of the body are referred to the corresponding region on the other side—just as some patients with a right renal calculus complain of pain in the left loin, so do some with appendicular trouble confined to the right side complain of pain in the left iliac fossa ; and thirdly, because very occasionally one comes across a patient with transposition of the viscera, in whom the cæcum and vermiform appendix are on the left side.

Acute diverticulitis has been described as 'left-sided appendicitis,' and this nickname is a good one in that, if one imagines acute appendicitis developing in the left iliac fossa, one has a very good idea of what the symptoms of acute diverticulitis, and its degrees and results, may be. In some it causes an acute abscess needing surgical measures for its cure; on the other hand, there may be an acute attack without suppuration, spontaneous resolution occurring just as it often does in the case of acute appendicitis. The patient is seized almost suddenly with acute pain in the left iliac fossa, and generally vomits. It hurts him to walk, so he lies down or goes to bed. His temperature and pulse-rate rise, appetite fails, the tongue is coated, there is generally either diarrhœa or constipation, or the two may alternate. Micturition is often frequent because there is pain if the urine is held long; locally there are acute tenderness and pain, with rigidity of the muscles over the lower left quadrant of the abdomen, generally palpable fullness in the same region, and often an actual tumour difficult to define well; on rectal or vaginal examination pain is complained of when the examining finger is pressed upwards and to the left. After a day or two these symptoms may begin to abate, and within a fortnight they may have disappeared; on the other hand, they may increase rapidly, and call for urgent surgical measures; or they may subside considerably without clearing up altogether, and may recur after a few weeks or months. At the operation the cause of the persistence of symptoms will be found to be a local thick-walled abscess in the left side of the pelvis, possibly suggesting a pyosalpinx if the patient is a woman. General peritonitis may supervene at any stage, just as it may with any form of appendicitis. The cause of the disease, which is not so very uncommon, though it is not always recognized, is the development of exaggerated sacculations of the colon with narrowing of their mucus-aspect orifices, so that if the interior of the intestine is seen it looks as if it had been punched with a series of small holes into which the end of the little finger may just pass, each such hole leading into a more or less dilated pouch or sacculus, generally with an appendix epiploica attached to its free end; such a diverticulum seems liable to inflammation just as the vermiform appendix is, and the result is spoken of as acute diverticulitis. Long-continued constipation, together with chronic colitis, seem to be predisposing factors in the origin of these diverticula, and acute diverticulitis is a disease of the second half of life rather than the first. Either sex may be attacked.

Subacute or Chronic Lesions.—*Carcinoma* of the *sigmoid colon* or of the *rectum* may cause pain in the colon generally, owing to its distention with accumulated fæces; sometimes this pain is complained of chiefly over the descending colon, and thus in the left iliac fossa. As a rule increasing constipation will be a more prominent symptom, or alternatively a constantly repeated desire and necessity to defæcate without satisfaction in the result. If blood and mucus are passed per rectum, if the patient is over forty, has had no bowel symptoms at all until the last two or three months, and has been losing weight, the carcinoma and its locality will suggest themselves at once, and an actual tumour may be felt in the left iliac fossa; the chief difficulty arises in cases who have long been habitually constipated, so that it is difficult to assess the importance of the increased difficulty complained of. Rectal examination, the sigmoidoscope, *x*-rays after a bismuth meal (*Fig.* 53, p. 125) or after a bismuth enema (*Fig.* 54, p. 126), may all be needed to exclude simpler conditions such as *impacted fæces* or *spastic constipation*, which are discussed under Constipation (p. 121).

Chronic diverticulitis is referred to under the heading of acute diverticulitis above; when it has given rise to a chronic thick-walled abscess in the left side of the pelvis, it may produce symptoms very like those of carcinoma of the pelvic colon on the one hand, and of other forms of pelvic abscess on the other, especially pyosalpinx, or periproctal or periprostatic abscess. Vaginal and rectal examination should be made, and then perhaps a primary source for one or other of these may be found; but not infrequently even when operative measures are resorted to, a chronic diverticular or a chronic periproctal abscess and its resulting matting and thickening are mistaken for new growth, and the patient is regarded as dying of a cancer unless a full post-mortem examination is undertaken to verify the diagnosis.

All the other conditions mentioned in the table above are discussed in the article on Pain in the Iliac Fossa (Right) (p. 454), and what is said there applies as much to the left iliac fossa as to the right.

Herbert French.

PAIN IN THE ILIAC FOSSA (RIGHT).—When a patient complains of pain in the right iliac fossa, probably the first thing that occurs to one as a possible if not even probable diagnosis, is appendicitis. As, however, there are a large number of other conditions which may produce the same symptom also, it is important to consider the possibility of each before concluding that the patient really has appendicitis. The pains may be either acute and severe, or they may be subacute or chronic; they may be complained of by a patient now for the first time, or there may have been previous attacks. These characters, however, do not distinguish any one cause with certainty from the rest, though they may serve as a basis for classification as follows:—

Causes of Pain in the Right Iliac Fossa.

1. *Acute and severe pain* may be produced by:—

Acute appendicitis
Acute salpingitis
Acute distention of the cæcum with gas
Calculus impacted in the right ureter
Acute ureteritis
Coli bacilluria
Twisted pedicle of right ovarian cyst
Pelvic abscess
Retained right testis
Suppurative periostitis of the ilium
Acute stitch
After local injury.

2. *Subacute, chronic, or recurrent pain* in the right iliac fossa may be caused by:—

Most of the conditions already enumerated in Group 1.
Ileocæcal kink (Lane)
Peri-appendicular adhesions
Pericæcal adhesions
Psoas abscess
Sacro-iliac joint disease
Tuberculous hip
Inflamed iliac lymphatic glands
Tuberculous iliac lymphatic glands
Tuberculous cæcum
Actinomycosis of the cæcum
Carcinoma of the cæcum
Movable right kidney
Tuberculous right kidney and ureter
Intestinal obstruction at a point further on in the intestines, due to any cause, such as carcinoma of the sigmoid flexure, etc.
Obturator hernia
Herpes zoster
Osteo-arthritis of the lumbar vertebræ
Infective arthritis of the lumbar vertebræ
Dysentery
Ulcerative colitis
Typhoid fever
Aneurysm of the right iliac artery
Sarcoma, osteoma, or chondroma of the iliac bone
Lobar pneumonia, pleurisy, or other chest conditions.

Formidable though the above list may seem, in the great majority of cases, when a patient complains of pain in the right iliac fossa, the first point to be decided if possible is whether that patient has appendicitis or not. In an acute case, in which the pains have come on rapidly and have become severe, are associated with an increased pulse-rate, and some rise of temperature; vomiting at the beginning; a coated tongue; local rigidity over the right iliac fossa; perhaps with, in addition to a sense of resistance, a diffuse palpable fullness in the right iliac fossa, or even a more or less localized tender swelling, together with tenderness of the right side of the rectum on rectal examination; the great probability will be that the patient has *acute appendicitis*, and although the circumstances of the case may prohibit operation, in most instances surgical measures will be employed to cure the condition, and at the same time the cause will be verified. The pain in cases of acute appendicitis is often associated with a remarkably localized acute tenderness referred to McBurney's spot, which is situated at the outer point of trisection of a line joining the umbilicus to the right anterior superior iliac spine. Another sign which is sometimes helpful is the presence of congestion of the right superficial circumflex iliac vein; this may be obvious at a glance, and it is suggestive of active inflammation of the underlying appendix.

Occasionally, when acute appendicitis has been the apparent diagnosis before operation, some other focal suppuration will be found when an operation is performed, and it is generally in this way that *acute suppurative periostitis of the inner surface of the ilium* is discovered, a somewhat rare but very important condition which simulates acute appendicitis very closely, and which can only be cured by immediate surgical treatment.

A *ureteral calculus* generally becomes impacted in the lower end of the ureter close to the bladder (*Fig.* 192), and it sometimes gives rise to little pain; occasionally, however, it produces acute ureteral colic, the pain being referred to the right iliac fossa in a way which simulates the pain of appendicitis closely; there may be local rigidity of the muscles but no tumour can be felt, and as a rule the patient is much less ill than he is with appendicitis. In a first attack of such pain, however, an operation for supposed appendicitis may very easily be performed ; it is when the patient has had recurrent attacks, associated perhaps with transient hæmaturia, that the real cause is suggested, or more often still perhaps, the diagnosis is arrived at as the result of routine examination, including the use of the *x*-rays. The only conditions likely to simulate a stone in the ureter when the *x*-rays are employed are either calcareous iliac glands or a phlebolith in formerly thrombosed iliac veins. Sometimes it is possible to tell the difference between these three conditions by the relative situations of the shadows ; more often there will remain some doubt, however, as to what the *x*-ray appearances signify, and it may be necessary to use the cystoscope and a ureteral bougie or catheter to determine whether there is a stone in the ureter or not.

Acute ureteritis produces symptoms almost exactly like those of an actual stone in the ureter, and in some instances at any rate it is due to inflammation of the lower end of the ureter resulting from a stone which has already passed. Occasionally, however, it arises apparently as a primary condition or as part of a bacillus coli infection of the urinary passages, and in some such cases operation for supposed appendicitis has been performed ; the vermiform appendix proving perfectly normal, but the ureter being seen to be thickened and inflamed. There can be no doubt that some such cases, diagnosed and operated upon for appendicitis, escape recognition altogether, for it is not always easy to tell whether the lower end of the ureter is inflamed when the tube is inspected merely from outside, but the writer's post-mortem experiences show that the lesion is commoner than might be expected.

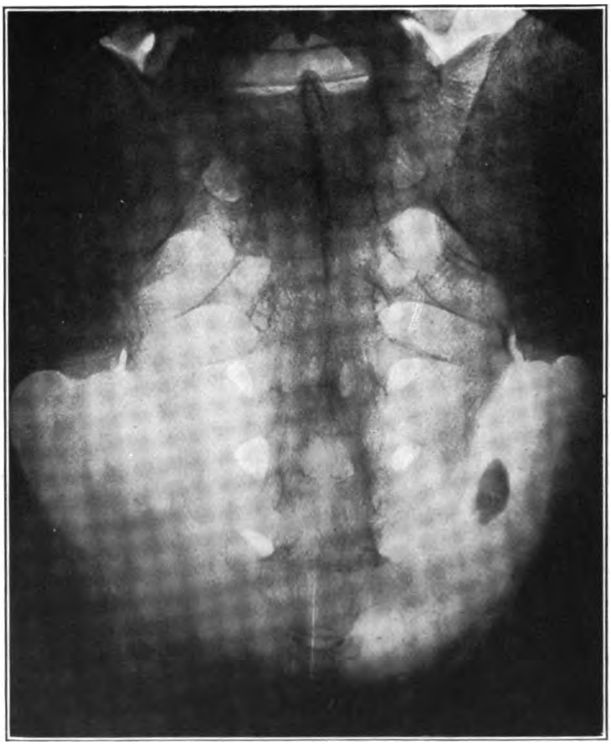


Fig. 192.—Skiagram of calculus impacted in lower end of right ureter. The diagnosis was confirmed at operation by Mr. Thelwall Thomas.
(*Skiagram by Dr. C. Thurstan Holland.*)

Coli bacilluria is now a familiar difficulty in the differential diagnosis of appendicitis ; although theoretically the pain would be referred mainly to the kidney, generally the right, it is quite common for patients suffering from this condition to refer their pain not to the back or loin at all, but to the front of the lower part of the abdomen, and particularly over the right iliac fossa, in such a way that acute appendicitis is simulated very closely. Even though the urine be examined and a haze of albumin found, together with an excess of leucocytes, the acuteness of the condition may be such that the surgeon may not feel justified in waiting for cultures of a catheter specimen of the urine to be made, to see whether bacillus coli communis is present in pure culture ; and not a few patients of this kind are unavoidably operated upon for acute appendicitis when the appendix is perfectly normal. The condition of coli bacilluria is recognized most readily when one has become familiar with previous cases ; the main features of the condition are described on page 69, and *Fig.* 193 is a temperature chart from a case in which appendicitis was simulated so closely that operation was performed and the diagnosis of coli infection of the right urinary passages, including the kidney, confirmed by the surgeon.

Twisting of the pedicle of an ovarian cyst upon the right side generally produces symptoms analogous to those of strangulated hernia, and the diagnosis may only be established when urgent laparotomy is performed, unless the patient is already known to have an ovarian cyst. As a rule the pain starts in the lower part of the abdomen before it becomes general, and in the case of a cyst upon the right side it may be referred particularly to the right iliac fossa, so that appendicitis may be simulated, especially as the patient may be very tender in the right lower quadrant of the abdomen, and a diffuse swelling may be felt here. Effusion of fluid into the general peritoneal cavity takes place rapidly, so that there may be dullness in the flanks, though the peritonitis which produces this is generally non-suppurative. The emergency is one which calls for laparotomy at once, and the diagnosis is confirmed after the abdomen has been opened.

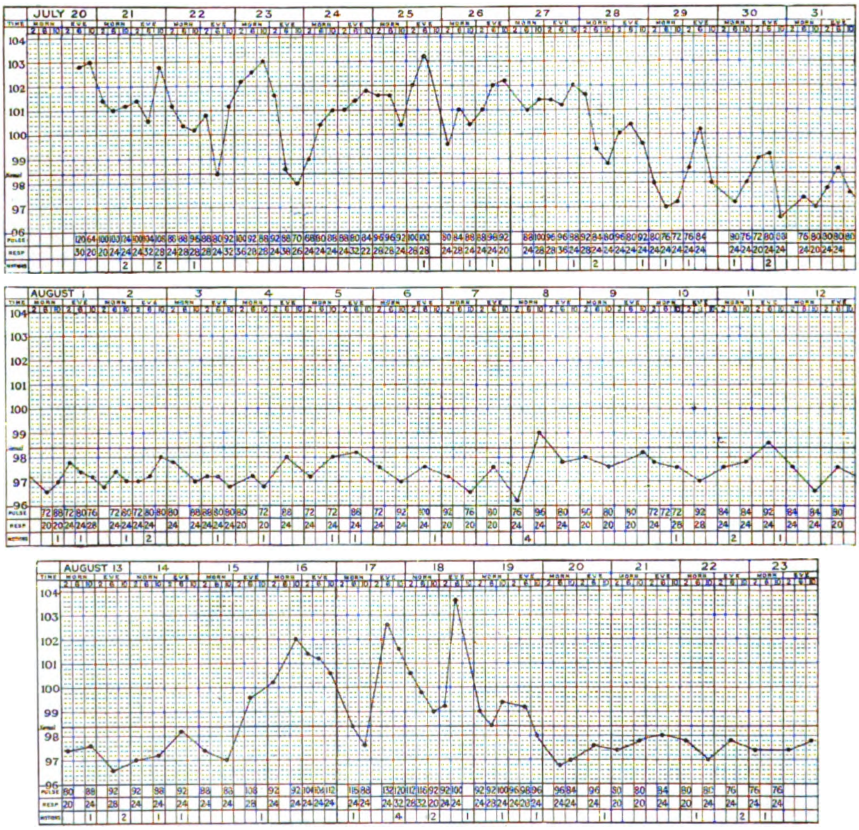


Fig. 193.—Temperature chart (four-hourly) from a case of acute coli bacilluria; it shows one recrudescence of the acute trouble after an apyrexial interval.

Acute salpingitis or *inflammation of the right ovary* are generally secondary to some other pelvic inflammation which has been diagnosed on account of other symptoms such as a vaginal discharge (p. 185), or pain in the pelvis (p. 467), or some menstrual irregularity such as menorrhagia (p. 385). The skilled gynæcologist may, by vaginal palpation, be able to determine the cause, though doubts may very often persist as to whether the condition is one affecting the right uterine appendages on the one hand, or the vermiform appendix on the other, unless laparotomy is performed. This procedure, as a rule, is much less urgent in the case of salpingitis or ovaritis than with appendicitis, and the doubts may therefore remain unresolved even for a period of months. Should the pains in the right iliac fossa be relatively slight in the intermenstrual periods, but severe at the time of the periods themselves, this fact will suggest that the mischief is pelvic and not appendicular; but even this conclusion is open to fallacy, because when there has been preceding appendicitis with extensive adhesions which may very likely include the uterine appendages, the pains, though primarily the result of appendicitis, may be recurrently worse at each monthly period. An important point, however, is that salpingitis and ovaritis, though possibly unilateral, are much more commonly bilateral, so that the distribution of the pains is likely to be much wider in the case of ovarian or Fallopian tube mischief than it is with or after appendicitis.

Acute stitch generally affects the flank in the lower costal region on one side or the other, but sometimes the muscles over the right iliac fossa are involved instead of the abdominal muscles higher up, or the diaphragm, and acute pain results in the right iliac fossa and the patient may fear appendicitis. The general character of the pain, however, the fact that it appears to be definitely in the abdominal wall, and that it is unassociated as a rule with acute hyperæsthesia or with any swelling or pyrexia, will generally serve to distinguish stitch from appendicitis.

Distention of the colon with fæces or with gas seldom produces very acute pain in the right iliac fossa, but rather a severe grumble; and although in a first attack appendicitis may be feared, recurrence of similar attacks without any obvious illness on the patient's part, and the way the pains are almost immediately relieved if the patient can pass flatus abundantly, or has his bowels evacuated, either naturally, by medicines, or by means of an enema, will generally serve to exclude acute appendicitis at any rate, though doubt may remain as to whether there may not be appendicular or perityphlitic adhesions. In some such cases examination of the bowel with bismuth and the *x*-rays may serve to show the distended condition of the cæcum, or on the other hand evidence of deformity from adhesions.

Retention of the right testis is a thing which should not be forgotten, especially when a lad at about the time of puberty complains of acute pain in the right iliac fossa, suggesting appendicitis. The testis at this time swells, and if it is situated at the upper end of the inguinal canal, distention of it may cause acute pain of a character very similar to that of appendicitis, especially as at the same time there may be local resistance suggesting a swelling in the iliac fossa. It is important therefore to examine the scrotum to see whether both testicles are present there or not. There is no pyrexia.

Injury to the right iliac fossa may be followed by acute pain. In most instances the history will indicate the diagnosis clearly, especially if there is local evidence of bruising. Sometimes, however, the patient may have injured himself when he was unaware of doing so, for instance during times of great excitement, or perhaps when he was under the influence of drink, or again during a nocturnal attack of epilepsy, and it may then be very difficult sometimes to tell whether the pains are due to injury or not. The absence of pyrexia and of an increased pulse-rate will be points against acute appendicitis, though there may be local swelling from a deep hæmatoma, and the injured muscles may be rigid.

Passing on now to a consideration of the differential diagnosis of conditions which may produce subacute, chronic, or recurrent attacks of pain in the right iliac fossa, it is clear that most of the conditions described above need to be borne in mind again, though one need not recapitulate what has already been said in regard to them. Many, if not all, of the additional causes mentioned under the second heading, however, may simulate or be confused with subacute or recurrent appendicitis.

The *ileocæcal kink* of Sir Arbuthnot Lane is now familiar to most physicians and surgeons as a cause of constipation by interfering with the free passage of the intestinal contents from the lower end of the ileum into the cæcum, and as a result of this kink discomfort, or a dull grumbling pain, or even an occasional acute pain, may result in the right iliac fossa; it may be very difficult to tell whether there is not some degree of appendicular trouble at the same time, but the diagnosis of the kink itself is relatively easy if a serial examination of the alimentary canal is made with the *x*-rays after the administration of bismuth. The actual narrow point between the bismuth kept in the lower end of the ileum beyond its proper time and the bismuth which has got past the kink into the cæcum can generally be seen very clearly; though it is important in making the examination that the patient should not have any paraffin, aperient, or enema during the period of the successive *x*-ray examinations.

Adhesions round the appendix itself, or round the cæcum, may be very difficult to diagnose unless the patient has recurrent pains in the iliac fossa subsequent to a preceding attack of appendicitis but without any recurrence of signs of inflammation, pyrexia, or raised pulse-rate. In some such cases, however, it is only when operation is performed for the relief of the symptoms that the adhesions can be diagnosed with certainty.

Tuberculosis of the cæcum is being recognized nowadays with increasing frequency (*Fig.* 194); it is nearly always associated with chronic phthisis, of which there will be either definite physical signs or *x*-ray shadows, and the sputum will generally contain tubercle bacilli. When the phthisis is active and extensive the number of bacilli swallowed are generally so numerous that if ulceration of the bowel occurs at all the ulcers are diffused widely through the ileum, cæcum, and ascending colon; but even in such a case the post-mortem evidence shows that the maximum incidence of the bowel tuberculosis which results from the swallowing of tuberculous sputum is in the region of the ileocæcal valve, presumably because some delay occurs here in the passage of the motions and gives the bacilli a better opportunity of attacking the mucous membrane. In cases which are much less acute the bowel tuberculosis is sometimes confined entirely to the ileocæcal valve region, involving perhaps the last inch or two of the ileum, the ileocæcal valve itself, the caput cæci, and the first inch or two of the ascending colon. There may be diarrhœa as a result of this chronic ulceration, but as a rule there is none, and the patient has become so accustomed to his lung condition that he presents himself to the physician complaining of a sense of dull pain in the right iliac fossa with occasional exacerbations. There may or may not be pyrexia owing to the phthisis. On examination an indeterminate fullness or even a definitely palpable mass may be felt in the right iliac fossa, and the first impression will probably be that the patient has chronic appendicitis, or even possibly carcinoma of the cæcum. It is the concomitant lung trouble which gives the diagnosis, and possibly tubercle bacilli may also be detected in the fæces by the antiformin process. A certain number of these cases have now been cured of their bowel trouble completely by excision of the affected parts, accompanied by anastomosis between the healthy lower end of the ileum and the healthy large bowel beyond the ulceration in the cæcum.

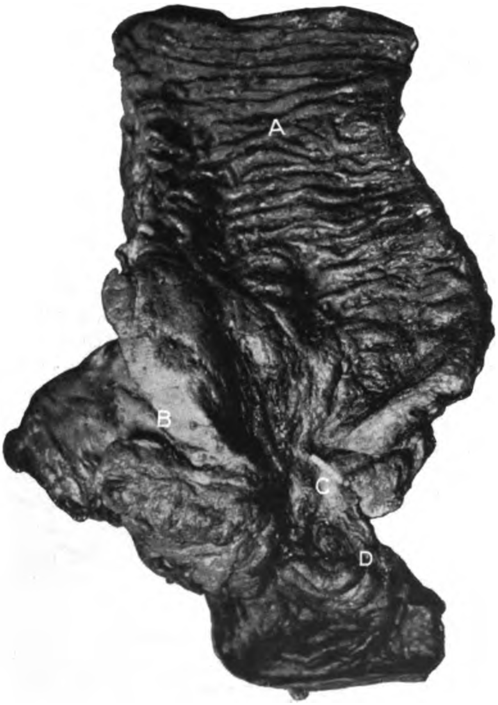


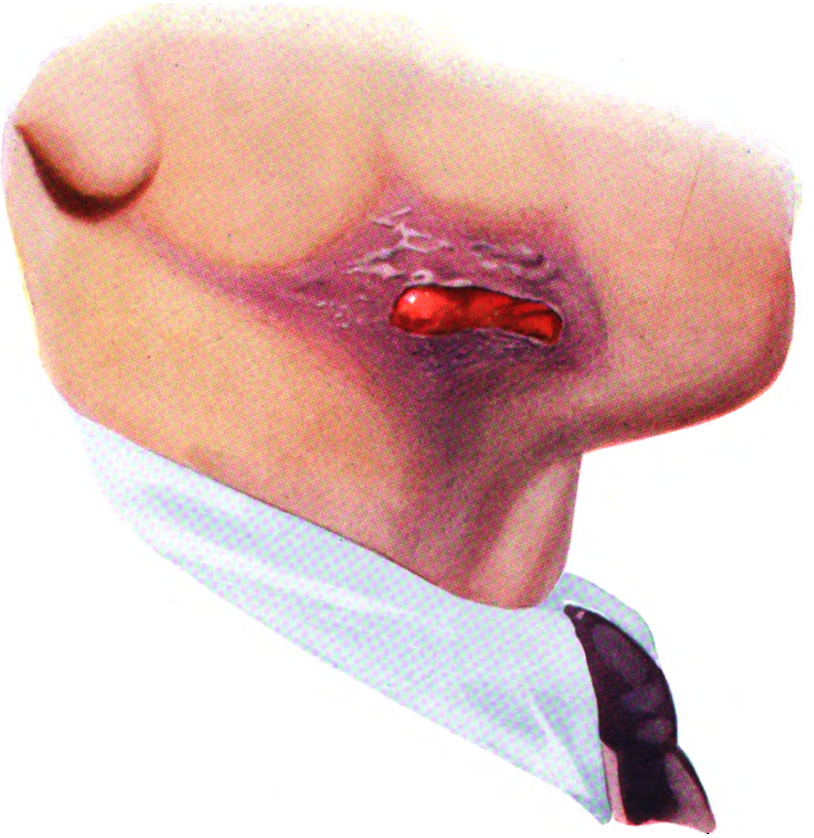
Fig. 194.—Photograph of chronic tuberculosis of the lower end of the ileum: ileocæcal valve and cæcum successfully excised in a case of chronic phthisis with symptoms suggesting chronic appendicitis. Patient alive and well seven years after the operation.

A Normal ilium. B. Dense tuberculous infiltration of the bowel wall. C. Ileocæcal valve, deformed by the tuberculous infiltration. D. Infiltrated cæcum.

Actinomycosis of the cæcum is rare, but it is a well-recognized condition which, when it does appear, often baffles diagnosis. In some cases the nature of the mischief in the right iliac fossa is suggested by the fact that the patient has a chronic inflammatory, suppurative, or ulcerative condition either of the jaw or cheek (*Plate XX A*), or of the pleura and chest wall, or of the liver at the same time, for these, in addition to the cæcum, are the sites most usually involved by actinomycosis. The chronicity of the affection is generally pronounced, but the ultimate diagnosis depends upon the discovery of ray fungi from the affected part (*Plate XXVIII*, p. 614). Were the cæcum involved primarily the symptoms would be more or less like those of either chronic appendicitis or of carcinoma of the cæcum, and laparotomy would probably be resorted to, though if the nature of the malady were suspected large doses of iodide of potassium would be prescribed with or without actinomycotic vaccine treatment, cure possibly resulting without operation.

PLATE XXA

ACTINOMYCOTIC ULCERATION OF THE JAW



Coloured sketch (from life) of the softened area in a case of perimandibular actinomycosis. An incision had recently been made and some gelatinous material wiped away. The central red area is therefore denuded of skin and shows the underlying granulation tissue. Granules of the fungus embedded in this tissue can be seen. The surrounding livid area represents the soft actinomycotic focus covered by thinned skin. It is rare for ulcers to occur naturally in the course of the disease. Treatment by potassium iodide caused marked improvement. *R. Clyne, del.*

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Inflamed iliac lymphatic glands may cause both pain, tenderness, and obscure swelling in the right iliac fossa, and appendicitis may be simulated. There will generally be pyrexia; if the inflammation in the internal glands is due to spread from the femoral or inguinal glands there will generally be some sore place upon the skin in the area drained by the latter to indicate the diagnosis, but the inflammation of the iliac may also occur independently of the inguinal lymphatic glands when the source of the infection is in the pelvis or secondary to periproctal or periprostatic inflammation. Rectal examination will assist the diagnosis.

Tuberculous iliac lymphatic glands generally form part of tabes mesenterica or of tuberculous peritonitis (p. 47), but sometimes the tuberculous deposits occur mainly, or even solely, in the glands in the region of the cæcum, in which case it may be very difficult to be sure without operation that the patient has not got chronic appendicitis or alternatively tuberculous lesions in the cæcum itself (see above). It is only when one can feel multiple small but firm, tender swellings, as is sometimes possible in a thin person, that the diagnosis of tuberculosis of these glands may be guessed at; as a rule the patient is a child suffering from general delicacy, and the condition is not one in which the symptoms call for urgent laparotomy. Von Pirquet's skin reaction may be positive, though even when it is the diagnosis of tuberculosis of these glands will often remain one of conjecture only. Sometimes the *x*-rays show multiple shadows in the position which the glands normally occupy, and thus assist the diagnosis.

Carcinoma of the cæcum is generally characterized much less by pain in the iliac fossa than by a definite, usually irregular firm mass, at first movable, later more fixed. This mass is distinguished from an accumulation of fæces in the cæcum by its greater firmness, by the fact that it cannot be moulded by the fingers, and that it does not disappear when the bowels are thoroughly evacuated. It may enlarge very slowly; in cases of doubt, operation with a view to possible excision will probably be resorted to, and the diagnosis thus confirmed. Sometimes carcinoma of the cæcum may be simulated by accumulated fæces, however, when there is obstruction to the bowel further on; there may for instance be adhesions obstructing the sigmoid colon, or a partial volvulus, or a carcinoma of the rectum, or of the sigmoid colon, leading to partial intestinal obstruction and preventing the fæces from leaving the cæcum thoroughly even when evacuant measures have been employed. The pain which the patient then complains of may be not at the site of obstruction so much as in the right iliac fossa. A bismuth and *x*-ray examination may assist materially in detecting the cause of the symptoms in such a case, and careful examination of the rectum with the finger, or of the sigmoid colon by means of the sigmoidoscope, may be required. It is noteworthy that when there is obstruction to the colon in its distal part, it is particularly in the cæcum that *stercoral ulcers* are apt to occur, and these may be associated with perityphlitis and pain in the right iliac fossa, more or less simulating appendicitis.

In *dysentery* and *ulcerative colitis* the abdominal pains are usually general, or at least referred now to one, now to another part of the whole colon, so that in these conditions the patient seldom complains mainly of pain in the right iliac fossa; occasionally, however, pain in the right iliac fossa may be more pronounced than over other parts of the colon, though the diagnosis of a more widespread infection will generally be indicated by the history, especially as to residence in the tropics in the case of dysentery, or of recurrent intractable diarrhœa with the passage of blood and mucus in one suffering from ulcerative colitis.

In *typhoid fever* the general symptoms will nearly always be out of all proportion to any pain in the abdomen, but occasionally so mild a case of typhoid fever is met with—the ambulatory type—that the patient has not been ill enough even to go to bed, and in some of these acute pains in the right iliac fossa have been the first thing to call attention to the nature of the disease; in some indeed the patient has had a perforation low down in the ileum, so that a local abscess in the right iliac fossa has formed, and the patient, though suffering from typhoid fever, has been operated upon as an ordinary case of appendicitis until the subsequent course of the pyrexia has indicated that something else was wrong, and it has occurred to one to have the blood tested for a Widal's reaction, which has then been found positive.

Discomfort or actual pain in the right iliac fossa may be the first complaint of patients suffering from a *tumour of the iliac bone*; this tumour may be simple—*osteoma* or *chondroma*—or it may be malignant *sarcoma*. In either case the diagnosis is arrived at by careful deep palpation, when the tumour will be felt to be firm or even bony hard, and, unlike other tumours in the iliac fossa, completely fixed to the deep parts.

An *aneurysm of the iliac artery* is very uncommon; when it does occur, pain in the iliac fossa may be considerable, and this pain may radiate thence down the right thigh; the diagnosis would be made by careful palpation and the discovery of a tumour with expansile pulsation.

Although both *movable right kidney* and *other lesions of the right kidney*, especially *tuberculosis*, cause pain in the loin to a more pronounced extent than pain in the right iliac fossa, some patients, especially those suffering from unduly movable right kidney, complain of much more pain in the lower right abdominal quadrant than in the loin, and the difficulty of distinguishing between such pains when due to movable kidney only, and a subacute appendicitis occurring in a person who has at the same time a movable kidney, may sometimes be considerable. Careful urine examination should be made; with renal tuberculosis pus cells and tubercle bacilli may be detected; with movable kidney the urine is generally normal, though sometimes there may be a little albumin or occasionally a trace of blood. With movable kidney, however, there will be no pyrexia, such as one would expect were there also appendicitis, and on careful palpation the patient will tell the examiner that she experiences the pain when he has the kidney between his two hands and not when he presses into the right iliac fossa without touching the kidney.

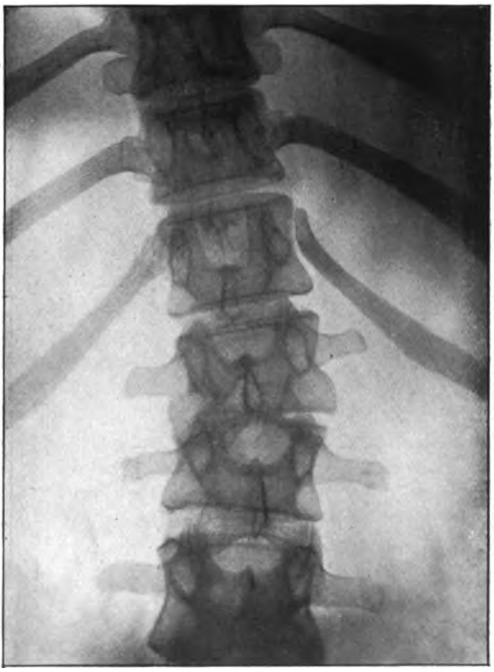

Fig. 195.—Skiagram showing tuberculous disease of 1st and 2nd lumbar vertebræ.
(*Skiagram by Dr. C. Thurstan Holland.*)

Herpes zoster is a condition which always needs bearing in mind when pains are unilateral without objective signs, and these pains due to herpes zoster may be present some time before the actual eruption appears, and also in a few cases for weeks or months after the eruption has subsided. It is even probable that in some cases there are pains from herpes zoster without any eruption at all, though then the diagnosis is almost impossible. As a rule, with herpes zoster occurring in such a way as to produce pain referred to the right iliac fossa, the pain will not be confined to this fossa, but will be referred also to the inner side of the upper part of the thigh and to some point in the right loin. The characteristic vesicles will also be looked for, or the slight scabs that may be left for some time after the vesicles have dried up.

The remaining conditions in the list above are some in which pain may be referred to the right iliac fossa by lesions at a distance, and it is only by bearing them in mind and looking for evidence of them that they can be recognized. Thus, the fact that a patient who is suffering from *acute pleurisy* at the base of the right lung, or from *pneumonia* in the lower lobe of the right lung, may complain of pain in the lower part of the abdomen rather than in the chest, is familiar; and sometimes it may be very difficult to decide whether the patient's lesion is abdominal or thoracic. Even when there are definite lung signs there may be doubt as to whether there may not be appendicitis as well; acute pleurisy, for example, may result from the rapid tracking up along the posterior abdominal wall of infection from the appendicular region. In such cases it is only by careful judgement that a decision can be come to. As a rule, in such cases there is neither rigidity nor tenderness in the right iliac fossa upon examination, although the patient may put his hand over the right iliac fossa when indicating where he has the pain. In a similar way chronic pain suggestive of ureteral calculus or of appendicular colic or even a subacute appendicitis may be complained of by persons whose posterior nerve roots on the right side are being irritated by bony or other changes in connection with the lower dorsal or upper lumbar vertebræ, for instance, from *spondylitis deformans* (p. 648) in an early stage; *osteo-arthritis* of the spine; infective changes in the spine of the same nature as *rheumatoid arthritis; spinal caries* with or without *psoas abscess*. Such cases will be of chronic duration, and it may be only after much deliberation and repeated examinations that a conclusion will be come to that the pains are referred from the spine and not due to primary trouble in the right iliac fossa itself. The *x*-rays will sometimes be of assistance in detecting the osteophitic or other bony changes in the vertebræ (*Fig.* 195), and if the patient's complaints can be analyzed successfully, it will generally be found that he has definite localized pain in the back as well as in the iliac fossa. The difficulty of being sure of the cause of the pains in such a case, however, may sometimes be very great, and even after weeks of observation their nature may still remain one of doubtful opinion only. *Herbert French.*

PAIN, INTERSCAPULAR.—(See also PAIN IN THE BACK, p. 427.) Pain referred to by the patient as being " in the back, between the shoulder blades," is due in the great majority of cases to simple indigestion, flatulence, or biliousness. Sometimes it is in the middle line, with a maximum intensity at the 5th, the 6th, the 7th or the 8th dorsal vertebra; sometimes it is referred more to one side of the mid-line than the other; often it varies in position, being sometimes near the mid-line, at other times right under the blade of one scapula—particularly on the left side in the case of indigestion and stomach conditions, on the right side when the patient is 'bilious' or his liver is out of order. It is very important, however, not to conclude forthwith that a patient who has this symptom is merely bilious or suffering from dyspepsia, for pain of a similar kind may be the result of more serious lesions, amongst which one must think of:—

Epithelioma of the œsophagus
Mediastinal sarcoma
Spinal caries
Sarcoma of the vertebræ
Carcinoma of the vertebræ
Infective arthritis of the spine }
Fibrositis } 'Chronic
Myositis } rheumatism'
Spondylitis deformans }

Aortic aneurysm eroding the vertebræ
Gastric ulcer
Duodenal ulcer
Gastric carcinoma
Gall-stones
Hepatic abscess
Carcinoma of the gall-bladder
Carcinoma of the liver.

The first step in arriving at a diagnosis will be to examine the bare back very carefully by inspection in a good light, and by palpation. Broadly speaking, the conditions mentioned above divide themselves into two main groups—namely, (1) Those in which the pain is not produced locally, but is referred to the interscapular region from a distance (*Fig.* 294, p. 716); (2) Those in which the pain is definitely of local origin, so that there is tenderness on palpation as well as pain. When the pain is due to local causes, there will generally be some deficiency in the free movement of the spinal column when the patient bends forwards and backwards, or from side to side, or when he twists round; and attempts at such movements will increase the pain in a way which is not the case when the cause is gastric or hepatic.

The maximum amount of rigidity is found in cases in which the bones themselves are involved, whether by *spinal caries*, *new growth*, an *aneurysm*, *spondylitis deformans*, or *arthritis*. The diagnosis of each of these may be clinched by *x*-ray examination in all but a few cases. Skiagraphy will also serve to detect *mediastinal new growth*; or, with the help of a bismuth meal, *epithelioma of the œsophagus*, which is generally suggested by the progressive DYSPHAGIA (p. 194) long before it produces pain between the shoulders.

Infective arthritis of the spine, *fibrositis*, and *myositis* are all associated with local tenderness and local stiffness or deficiency in movement as well as with local pain; in only a few cases will the parts affected be the mid-dorsal region of the vertebral column alone; in most instances there will be pains in other parts of the body as well, referred to vaguely as 'neuritic,' together with more or less affection of many joints of the multiple rheumatoid arthritis type (p. 341). All three are closely related to one another, and where fibrositis or myositis ends and infective arthritis begins is usually impossible to define. Local infiltration may sometimes be recognized on minute palpation, especially by those who have had training in massage. The diagnosis should not be made, however, before caries, aneurysm, and other serious deeper lesions have been excluded by skiagraphy.

When there is no local thickening, tenderness, or rigidity, and no local abnormality to be made out with the *x*-rays, it will be probable that the interscapular pain, or the pains beneath the blade of one or other scapula, is a referred pain, either of gastric or hepatic origin. In many such cases the diagnosis of FLATULENCE (p. 240), or simple INDIGESTION (p. 315), or of biliousness is guessed at rather than made, and the chances are that the patient will be suffering from one or other of these ill-defined 'simple' conditions rather than from something more serious,—such as gastric or duodenal ulcer, gastric carcinoma, gall-stones, hepatic abscess, or carcinoma of the liver or gall-bladder; especially if he has had similar pains on many previous occasions, losing them completely in the intervals. The severity of the scapular or interscapular pain is no guide, for it may be extreme even when the patient is suffering from nothing worse than flatulence or biliousness. It is important, however, not to jump too readily to the conclusion that the pain is due to these simple conditions, especially if it does not yield easily to simple treatment, such as bicarbonate of soda or a little calomel. Careful abdominal palpation is indicated, lest a gastric or hepatic tumour escape detection; with these the pain will seldom be solely interscapular, however, and here, just as in the case of gastric or duodenal ulcer, hepatic abscess, or gall-stones, there would be pain in the epigastrium or hypochondrium to direct attention to the nature of the lesion, which would be suggested further, perhaps, by such other symptoms as VOMITING (p. 763) or JAUNDICE (p. 324). Nevertheless, in some cases it may only be by watching the patient for a time that an exact diagnosis can be made; and occasionally that which at first appears to be no more than the interscapular pain of dyspepsia or biliousness, proves to have been really due to gall-stones or a carcinoma.

Herbert French.

PAIN IN THE JAW (LOWER)—unaccompanied by any swelling (see SWELLING OF THE JAW, LOWER, p. 683)—is generally due to *dental caries*, i.e., toothache, and it is with this thought in the mind that an examination should first be made. The decayed tooth may be obvious at once, or it may be so hidden as to call for the services of a skilled dentist. Occasionally an unerupted molar may be the cause of the pain, and a skiagram may be needed to complete the diagnosis.

Neuralgia.—Here, pain is the essential feature, and it may be of two kinds. It either follows the course of a nerve such as the inferior dental in the lower jaw, or it affects a considerable part of the jaw without special reference to any nerve. It varies greatly in severity, being sometimes slight, at other times so severe as to call for all the fortitude of the patient to bear it. Usually neuralgia of the inferior dental nerve is combined with neuralgia of the other branches of the fifth nerve, and this in conjunction with the spasmodic character of the pain makes the diagnosis easy. Some cases of neuralgia are embarrassing, especially when sources of irritation in decayed teeth are present as well, and it may be that the true condition can only be settled after all the teeth have been extracted.

Other causes, such as epithelioma or other neoplasm, do not produce pain as a rule until the diagnosis has been arrived at on other grounds. *George E. Gask.*

PAIN IN THE JAW (UPPER).—What has been said above as to pain in the lower jaw being caused by dental caries and neuralgia applies equally to pain in the upper jaw, but there is an important additional cause to be sought for in the latter, and one easily overlooked, namely, inflammatory affections of the antrum of Highmore.

Abscess of the Antrum of Highmore.—The presence of pus within the antrum is indicated by local pain generally dull in character, but sometimes acute. On examination of the jaw, the gums will often be found tender and swollen, and a carious tooth is frequently the source of the infection. So far, the signs are compatible with those arising from a septic tooth, without implication of the antrum, and further evidence is required; the most certain is the periodical discharge of pus, which may run from the corresponding nostril when the head is bent forwards, or trickle down the pharynx when the patient is lying on the back. If the normal opening of the antrum into the nose becomes closed, as it may from inflammation, this valuable sign is lost, and though local signs of inflammation and general febrile disturbances are present, it may be difficult to arrive at a diagnosis, for the condition is simulated by inflammation in the nasal fossæ or suppuration in the ethmoidal and frontal sinuses. It must also be remembered that a growth, either innocent or malignant, starting in the antrum and not yet big enough to cause a swelling, may easily be mistaken. Recourse, therefore, should be had to the method of transillumination, and the antra on the two sides compared (see *Fig.* 84, p. 180). The position of the antrum should be shown by a bright red area, and if instead a shadow is thrown, there is presumably some affection of the antrum. It does not mean necessarily that there is an abscess, for a growth or a thickening of the bone may cast a shadow equally well. In these conditions a skiagram may help. A growth may indicate its presence by pain before the appearance of any swelling, but the differential diagnosis is discussed under SWELLING OF THE JAW, LOWER (p. 683). The only certain method of diagnosis of an antral abscess is by tapping the antrum with an exploring syringe. This can be done through the nose immediately under the anterior part of the inferior turbinate bone. The fluid withdrawn may be subjected to microscopical and bacteriological examination. *George E. Gask.*

PAIN IN THE JOINTS.—(See JOINTS, AFFECTIONS OF THE, p. 337.)

PAIN IN THE LEG.—(See PAIN IN THE EXTREMITY, LOWER, p. 438.)

PAIN IN THE LIMBS (General).—In the great majority of cases pains in the limbs are the result of some general or systemic disease : in but few instances can they result from symmetrically distributed local lesions. For clinical purposes they may be classified by their duration, according as they are acute or chronic.

1. **Acute General Pains in the Limbs** occur in—

Rheumatic fever
Muscular overstrain
Myositis—
  Acute polymyositis
  Neuromyositis
Trichinosis
Peripheral neuritis
Neurasthenia
Hysteria
Acute infections such as—
  Acute coryza
  Tonsillitis
  Febricula or chill
  Influenza
  Acute specific fevers
  Rat-bite fever
Inflammations of the lungs, kidneys, etc.
Secondary syphilis
Tropical fevers—
  Dengue, Malta fever, cholera, yellow fever, dysentery, malaria. etc.

2. **Chronic General Pains in the Limbs** occur in—

Peripheral neuritis
Tabes dorsalis
Chronic rheumatism
Myalgia
Pernicious anæmia
Osteo-arthritis
Rheumatoid arthritis
Gonococcal arthritis
Chronic wasting disease, as pulmonary tuberculosis
Gastritis
Cirrhosis of the liver
Bronchitis
Emphysema
Morbus cordis
Severe anæmia
Malignant disease
Nephritis.

These general pains may be felt most acutely sometimes in one tissue or part of the limbs, sometimes in another. The muscles, for example, may be the chief seats of pain in a child with rheumatism ; in a rheumatic adult the pains are usually worst in and about the joints ; in a patient with secondary syphilis the pain is often deep in the bones, the so-called osteocopic pain. General limb-pains are usually made worse by movement, particularly when they are accompanied by inflammatory changes in the joints ; but the general pains of chronic rheumatism, or the stiffness and pains left after muscular overstrain, will often pass off if the movements be persisted in for a little time. As a rule, general pains in the limbs are least felt when the patient is at rest, especially when he is at rest in bed ; but in some cases rest leads to stiffness and increased discomfort, change of position giving temporary relief ; in others—particularly the muscular pains of rheumatism and the osteocopic pains of specific disease—the pains are at their worst as soon as the patient gets warm in bed.

Acute Pain.—General pains in the limbs are common in *rheumatic fever*, occurring mainly in the limbs in which there is acute inflammation of the joints. In severe cases the cause will not readily be overlooked : it is in the comparatively mild cases in children that failure to make the proper diagnosis is likely to occur, when the general pains in the limbs may be set down as merely 'growing pains.' There is no doubt that 'growing pains' occur in healthy children, quite independently of rheumatism; but any complaint of growing pains should lead to a careful investigation of the patient's history, and of the condition of his heart; a family history or past personal history of either rheumatism or chorea will often be made out; and not rarely, examination of the heart will lead to the discovery of valvular disease. The joint-affections of rheumatism are far more prominent in adults than in children; in children the non-arthritic lesions are the most conspicuous—endocarditis, pericarditis, pleurisy, chorea, inflammations of the fibrous tissues generally and of the skin, sore throats. The way in which both the pains and the pyrexia are relieved by salicylates may often assist the diagnosis; if both are relieved within forty-eight hours after salicylate treatment is begun, acute rheumatism is probable, whilst conversely, if the pains persist in spite of salicylates, acute rheumatism is not probable (*Fig.* 150, p. 338).

Few people can be unacquainted with the general pains and stiffness due to *muscular overstrain*, the result of some violent and unusual muscular exertion—walking, running, playing games, etc.—undertaken when the body was out of training. The pains are accompanied by local tenderness of the affected muscles, and there may be slight fever.

Myositis, or inflammation of the muscles, is a comparatively rare cause of general pain in the limbs. *Acute polymyositis*, also described as dermatomyositis and as pseudo-trichinosis, is characterized by pain, rigidity, and tenderness in the muscles, œdema of the extremities, and a rash resembling one or other of the exudative erythemas. In addition, there are the general symptoms of malaise, anorexia, general debility, and fever. It must be distinguished from trichinosis, in which the affected muscles are found to contain *Trichinella spiralis*, the face and eyes are œdematous, and the blood shows eosinophilia. A second rare form of myositis is the *neuromyositis* described by Senator, in which the nerves are involved as well as the muscles. In this, sensation is lessened, the reflexes are lost, and vasomotor phenomena are seen in the extremities. The affected limbs are tender on pressure, and painful when movement is attempted.

Trichinosis, or infection with *Trichinella spiralis*, is very rare in Great Britain, though common in countries where pork is eaten uncooked. Its symptoms are due to gastro-enteritis, and to invasion of the tissues of the body, particularly the muscles, by young trichinellæ. For the first week or ten days the main symptoms are gastro-intestinal, and may suggest cholera. Then the second stage comes on with pains and swellings in the muscles, particularly the flexors. The face, neck, and trunk are affected as well as the limbs; the face and eyes become œdematous; profuse perspirations are common, and high fever is not rare; eosinophilia and leucocytosis are usual. If not fatal the symptoms last a month or more, subsiding gradually into the third stage, that of convalescence, as the larval trichinellæ become encysted in the muscles. The diagnosis of trichinosis is likely to be difficult because of its rarity; it is most likely to suggest itself when it occurs in epidemic form. In the early stages, acute gastro-enteritis, enteric fever, or even cholera, will be suspected, the main symptoms arising from the irritation of the alimentary canal set up by the parent trichinellæ breeding in it. Later, rheumatism will be simulated; but the pain and swelling are in the muscles, not the joints, and the occurrence of œdema and of eosinophilia should help in the diagnosis. It may be added, that adult trichinellæ may be found in the stools of a patient with trichinosis, and larval trichinellæ in portions of the affected muscles removed *intra vitam* for microscopical examination: encysted larvæ will also be seen in the infected meat that gave rise to the attack, should any of it have been preserved.

Aching pains all over the limbs or body, or both, are quite common at the onset of many of the *acute infectious disorders*, or of acute diseases that mainly affect one or another of the organs of the body. Associated with these pains are other general symptoms, in most instances, such as malaise, headache, anorexia, and more or less fever. Thus, a severe *acute coryza* or *tonsillitis* may be ushered in by general pains in the limbs; so may the obscure and elusive acute attack known as a *febricula* or a *chill*, in which the fever and general symptoms persist for a day or two, but no localizing signs or symptoms can be detected to give evidence as to "where the chill has settled." Such febriculæ may really be abortive attacks of pneumonia or rheumatism, the onset or recrudescence of pulmonary tuberculosis, instances of undetected sore throat, acute gastro-intestinal upsets, cases of larval enteric, scarlet fever, measles, or what not. If they are associated with much pain or prostration, there is a great tendency to apply the term 'influenza' to them indiscriminately, quite apart from considerations of fact—evidence of infection with Pfeiffer's bacillus; or of probability—the detection of any source whence influenzal infection could have been derived. But, however satisfactory it may be to the patient, the diagnosis of *influenza* should not be made without further evidence, such as is furnished by the discovery of Pfeiffer's bacillus in the patient's nasal or bronchial secretions, or by the occurrence of the attack as one of many in an influenzal epidemic. Influenza is well known to be a protean disorder. In many instances its main symptom is a severe coryza, with headache, lachrymation, pyrexia, and much prostration. In others the type is respiratory, bronchitis with cough and viscid expectoration taking the place of the coryza, and leading up to a bronchopneumonia or lobar pneumonia that not infrequently results in death. A third variety of influenza is the abdominal and gastro-intestinal; abdominal pain, vomiting, diarrhœa, and perhaps jaundice, being the main phenomena. In all of these the pains, depression, and prostration come on very rapidly, and appear severe out of all proportion to the objective signs of the disease, while the fever is usually of short duration. The diagnosis of epidemic cases should not be difficult, but in the sporadic cases it may be far from easy, and must be made on the general lines indicated above.

It is not necessary to refer in detail to the many other acute infections or inflammatory processes in which general pains occur in the limbs. In *measles*, *scarlet fever*, or *small-pox*, for example, the pains often occur at the outset, but the diagnosis will be made on the other symptoms, and confirmed by the appearance of the characteristic rash. Recurrent attacks of high fever with pains in the limbs are characteristic of the little known and rarely recognized *Rat-bite Fever* (see p. 598). Various febrile disorders of the lungs, such as *bronchitis*, *tuberculosis*, or *pleurisy*, may begin with similar pains; so may *gastro-intestinal* infections, or acute inflammations of the *kidneys*. The diagnosis in these instances will be made from the special symptoms developed in each; the pains in the limbs will rarely be the only or the most prominent complaint.

In *peripheral neuritis* of the symmetrical multiple type, the amount of pain is very variable—great in some cases, little in others. The peripheral nerves contain motor, sensory, and vasomotor fibres; in peripheral neuritis, therefore, motor and vasomotor symptoms are habitually present, as well as sensory. Alcoholism is the commonest cause of multiple symmetrical peripheral neuritis; the chief complaints are of numbness and tingling in the extremities, 'pins and needles,' sensations of 'dead fingers,' cramps in the legs, and severe gnawing or aching pains in the limbs. Beginning in the hands and feet, they tend to spread to the trunk; motor weakness comes on, the skin develops hyperæsthesia, the limbs become very tender to pressure. The deep reflexes, originally increased, are now lost; the sphincters are hardly ever involved in alcoholic neuritis unless the mind is affected. Mental symptoms are common in alcoholism, taking the form of Korsakow's psychosis; memory for recent events is lost: the patient may forget his name and address, and not know where he is; and in the endeavour to make good the lacunæ in his recollections, he is likely to lie freely, and quite without any definite wish to deceive. The physical signs of *arsenical neuritis* are similar to those of the alcoholic form, but inco-ordination and the cutaneous and deep hyperæsthesiæ are more marked, and muscular paresis and wasting come on earlier; and there may be other arsenical symptoms (p. 64). In the neuritis due to acute *lead poisoning* the sensory signs are entirely subordinated to the motor, and pains in the limbs are absent. Peripheral neuritis is a fairly frequent legacy of influenza, and may then be characterized by great severity and persistence; it may also occur as a complication of other infectious disorders—such as diphtheria, tuberculosis, or syphilis. The diagnosis of peripheral neuritis will be suggested, speaking generally, if the pains in the limbs are associated with marked sensory changes—anæsthesia, paræsthesia, hyperæsthesia—with tenderness of the skin, muscles, or along the course of the nerves, and with weakness, atrophy, and the reaction of degeneration in the muscles.

Hysterical and *neurasthenic* patients sometimes suffer acutely from pains in the limbs that lack any objective basis on examination, and may give rise to much trouble in diagnosis. It is of great importance that organic disease of every kind should be excluded before the diagnosis of hysteria or neurasthenia is given out. The hysterical patient is generally a woman, and is likely to exhibit several of the many phenomena common in hysteria, such as functional aphonia, globus or clavus hystericus, stocking-and-glove anæsthesia, hemianæsthesia, variable paralyses often due to the contraction of antagonistic muscle-groups, hysterical seizures, and the like. The signs and symptoms of hysteria change from time to time, the recovery from any particular affection often being as sudden as its onset. The neurasthenic patient, on the other hand, is oftener a man than a woman, usually overworked, run down in general health, and worried. The symptoms are those of 'brain-fatigue' for the most part; inability to attend to or take interest in either work or pleasure; the bodily strength is lessened, and subjective sensations of all sorts may be felt in the back or limbs. Headache is a prominent feature in some neurasthenic patients; dyspepsia or palpitation in others; imaginary sexual disorder in others. Exaggerated knee-jerks accompany plantar reflexes that are still normally flexor, and the temperature is often subnormal.

General pains in the limbs are common in certain diseases of hot countries, of which only two need be considered here. Both occur in Southern Europe, as well as in more tropical regions.

Dengue is an epidemic infectious disease, much like influenza in many respects. Its onset is sudden, with headache and pains all over, fever, sore throat, an initial erythematous rash, and rapid pulse. The pains may be in the joints mainly, or diffused throughout the muscles of the limbs, and are made worse by movement. After two or three days the patient feels better, and begins to get about again; but after an interval of a day or two a slight or severe relapse occurs, with pains as before, fever, and a secondary roseolar rash, which begins on the hands and wrists, later spreading in patches over the whole body. The relapse is soon over; but convalescence may be slow, with persistence of the general pains in the limbs. The diagnosis should be easy in epidemics of dengue; the sudden onset, extent of the pains in limbs, head, and loins, and the characteristic course, should suffice to distinguish sporadic cases from other acute disorders such as measles, scarlet fever, rheumatic fever, etc.

Malta fever occurs mainly in the Mediterranean and on its shores: it is a chronic fever, characterized by perspirations, constipation, and rheumatic pains in the limbs; arthritis, orchitis, and enlargement of the spleen are common. The early symptoms are obscure; but pains in the limbs and general debility, gastric derangements, headache, bronchitis, and continued fever, are the general characteristics when the disease is established. The diagnosis would turn on the discovery of exposure to infection, the milk of goats that are carriers of the *Micrococcus melitensis* being the actual vehicle of infection: the patient's serum shows the specific agglutinating reaction.

Chronic General Pains in the Limbs will often remain after several of the disorders mentioned under the former heading. Thus, the pains due to *peripheral neuritis* may become a chronic affection in cases of chronic alcohol, arsenic, or lead poisoning, after influenza, or in gouty, diabetic, or syphilitic patients. Usually only one or two of the limbs will be affected in these cases; and the diagnosis will not have to be made from the occurrence of the pains, but will have become evident from the development of other signs of disease: a blue line on the gums (p. 34), tophi, and previous attacks of acute gout, sugar in the urine, and so forth.

To certain uncommon cases of tabes dorsalis the name *tabes dolorosa* has been given, owing to the severity and extent of the pains. The patient presents the usual symptoms of tabes (p. 609); in addition he has frequently repeated lightning pains in the limbs, so severe as to form the dominating element in his disorder from the subjective point of view. The diagnosis will be made from the suddenness and shocking intensity of the pains on the one hand; and on the other, from the discovery of further signs of tabes—Argyll Robertson pupil, loss of knee-jerk, ataxia, sphincter troubles, areas or zones of anæsthesia. The pains will have a radicular distribution, and the nerve-trunks and muscles will not be tender on pressure.

General pains in the limbs are common in *chronic rheumatism*, occurring particularly in consonance with changes in the weather. In some instances, the muscles are the chief seat of the pain; in others, the joints or the fibrous tissues round them. In most cases, exercise, massage, or movement tend to diminish these pains, if the patient can be induced to submit himself to the discomforts of motion or exertion. Occurring in children, these chronic pains are usually set down as 'growing pains'; but their association with acute rheumatism is so frequent that the patient should always be examined for other evidences of the rheumatic infection (see p. 464). In adults, on the other hand, chronic rheumatism is less often an inheritance from acute rheumatism, and is not so frequently combined with valvular disease of the heart; but it gives rise to pseudo-ankylosis of the joints, inability to work, and much impairment of the general health. The possibility of gonorrhœal arthritis, miscalled gonorrhœal rheumatism, must not be overlooked.

Myalgia, or the so-called 'muscular rheumatism,' is a common affection of certain groups of muscles, and may in some instances affect the limbs generally. It is due to chill, exposure to cold after sweating, sitting in a draught, and the like. Its commoner forms, such as lumbago, stiff neck, pleurodynia, stiff back, need only be mentioned; in the rare cases where the limbs are attacked, the diagnosis of muscular rheumatism will probably be made *faute de mieux*, although there is nothing to show that the affection is rheumatic, and no proof that it is the muscles (and not their sensory nerves, for example) that are affected primarily. It happens occasionally that severe pains in the limbs, or pains all over the body, are felt by patients with *pernicious anœmia*. In some instances these pains are associated with great tenderness of the bones of the limbs and trunk to pressure, which may be connected with the hypertrophic changes taking place in the marrow within them. Similar aches, pains, and tenderness may be found in any of the severe anæmias or leukæmias.

There remains for consideration the large class of diseases characterized by *chronic wasting* or *cachexia*, in which general pains in the limbs are often prominent. These pains are due to widely different causes in different instances. In some they may be due to nothing more than exaggerated muscular fatigue or overstrain; the debilitated patient has but little muscle, and that little is exhausted by exertions that would be trifling for a normal subject, so that the cachectic patient becomes the victim of general pains by the mere fact of being up and about. In other cases the pains are connected with peripheral neuritis, set up by the circulation of toxins in the patient's blood, though few or none of the other signs or symptoms of neuritis may be detected on investigation. In others, again, the pains seem to be connected with the occurrence of fever, being lessened or absent when the patient's temperature is normal. In the great majority of cases these pains are lessened by rest, or by any treatment that builds the patient up and increases his strength. Either the lungs, the heart, the liver, and gastro-intestinal system, or the kidneys may be the organs primarily at fault, and bodily wasting and weakness will be among the main symptoms. In cases where the organic disease is deep-seated and out of reach, there is danger lest the patient who is really seriously ill should be suspected of nothing more than functional disease and treated for such. Thus, patients with carcinoma of the stomach may be treated for hysterical vomiting or anorexia nervosa; the victim of a carcinoma or aortic aneurysm invading the spinal canal may receive the treatment usually meted out to the malingerer. It is important, therefore, that the most thorough examination should be made, and deep-seated organic disease of every sort excluded as far as is possible, before the diagnosis of functional disease be made in a cachectic patient. This is all the more necessary because there is no doubt that purely functional disease of long standing may reduce nutrition or bodily strength to a very low ebb. *A. J. Jex-Blake.*

PAIN IN THE NECK.—(See SORE THROAT, p. 613; and STIFF NECK, p. 647.)

PAIN IN THE PELVIS.—In practice, pelvic pain can usually be classified under four headings, namely: (1) *Deep-seated pain*; (2) *Superficial pain in the skin*; (3) *Spasmodic pain*; (4) *Backache or sacralgia.*

Deep-seated Pain is aching in character, continuous, and may be acute in onset, or may be chronic in duration. It is associated with tension in the pelvic organs, usually the result of overfilled vessels, or, in other words, of congestion. If the result of actual inflammation, i.e., congestion due to infection, it is acute, and very severe. It is elicited by pressure, and thereby made worse. In its worst form it is of peritoneal origin; but it may be due to simple congestion of the uterus, tubes, or ovaries, without infection or evidence of actual inflammation. The presence of adhesions between the pelvic organs is an important factor in the differential diagnosis of this type of pain, making it abundantly clear that there has been a past peritoneal inflammation, and that the tension in the organs is the result of the binding and pressure of new fibrous tissue. Thus it may be caused by :—

Local peritonitis due to infection, recent or remote, caused by salpingo-oöphoritis, infection after labour or abortion, ovarian cyst with torsion of the pedicle, extra-uterine gestation, appendicitis.

Simple congestion, caused by retroversion and flexion of the uterus, prolapsed ovaries, sclerosed ovaries, hæmorrhagic corpus luteum cyst, endometritis.

Superficial Pain in the Skin.—This is elicited bv pinching or touching the skin with the head or point of a pin. It is essentially a referred pain, and may radiate very widely over the abdominal area, down the groins, over the crest of the ilium, and down the thighs. The area on the skin in which referred pain is felt in connection with uterine, tubal, or ovarian disease, is that to which the tenth dorsal nerve is distributed ; and the area is that which is commonly known as the 'ovarian region.' It is not, however, ovarian only, and it is not even uterine and tubal only, but may be affected also by lesions of the kidney, ureter, gall-bladder, and some parts of the intestines. Consequently, referred pain in the skin in this so-called ovarian region cannot be taken to indicate disease of the generative organs at all, unless other lesions can be eliminated. The region of the tenth dorsal segment is simply a horizontal band spreading behind from the first to third lumbar spines, and extending round the body with its upper level in front at the umbilicus. All parts of the region are not necessarily affected equally, and there may be points of maximum intensity ; one, notably, is midway between the umbilicus and anterior superior spine. This spot, especially on the left side, has often been taken erroneously to indicate pain due to ovarian inflammation. It is interesting to note that referred pain is commonly more marked on the left side of the body, the explanation of which is not quite clear. Referred pain in this segment may not be due to any local lesion at all, but may be a marked manifestation of hysteria in its graver forms. When extreme hyperæsthesia of this area on the left side is accompanied by anæsthesia of the skin of the legs and feet up to the level of the knees, with brisk knee-jerks and absence of the palate reflex, the diagnosis of hysteria is almost certain.

Spasmodic Pain in the pelvis is nearly always due to painful uterine contractions when it is of genital origin. The exception to this is the pain, certainly spasmodic in character, which occurs in connection with *tubal gestation*, as a rule in the week or two preceding tubal abortion or rupture of the tube. In this case it is supposed to be due to contraction of the muscle-coats of the tube, but there is no real evidence that this is a fact. There can be no doubt that, even though a part of the pain is muscular, some of it at least must be due to peritoneal irritation. The only way to diagnose between this tubal pain and that due to uterine contractions, is by a careful consideration of the history of the case, and the finding of a definite tubal swelling by the bimanual method. Even then the diagnosis is exceedingly difficult and often impossible. Spasmodic pain due to *uterine contractions* is caused by : The onset of abortion or labour ; deficient development of the uterine muscle in spasmodic dysmenorrhœa (p. 192) ; expulsion of a growth from the uterus such as a fibromyoma ; 'after-pains' following labour ; gauze packing of the uterus after operations.

The differential diagnosis of these conditions is fortunately easy ; but a much greater difficulty is sometimes met with when spasmodic pain has to be diagnosed which is due to causes which may not be of genital origin at all. The possible *extraneous causes* of spasmodic pain have already been outlined (see DYSMENORRHŒA, p. 192), and are : Appendicitis, intestinal, renal, or hepatic colic, leaking gastric ulcer, ruptured tubal gestation, twisted ovarian pedicle, hæmorrhage into a Graafian follicle, rupture of an ovarian cyst or pyosalpinx, dyspepsia, and flatulent distention of the bowels.

Backache, or **Sacralgia,** is a very common symptom in all classes of pelvic disorders ; and may be present at the same time as deep-seated pain and superficial skin tenderness. It is associated especially with chronic uterine congestion and endometritis, backward displacement of the uterus, downward displacement (prolapse), and impacted uterine or ovarian tumours. Sometimes the only lesion to be demonstrated is a chronic cervical catarrh or a cervical erosion. It is a very difficult pain to explain in all cases ; but it is usually regarded as one referred to the roots of the actual nerves which supply the uterus, tubes, and ovaries. In cases of impacted tumours it is possible that the pain is due to actual pressure on the sacral nerves at their exit from the bone, in which case pain will also be felt down the inner side and backs of the thighs. In cases of *carcinoma of the cervix* backache is complained of, but is always associated with pain in the 'ovarian regions,' inguinal region, and also radiating down the legs. It must not be forgotten that this form of backache is not necessarily of genital origin, but may be the result of many other lesions. Thus, it may be the result of some irritating urinary constituent, like excess of urates and phosphates; also it may accompany a calculus in the ureter or some lesion of the renal pelvis. As a rule, in renal cases, the pain is situated rather higher up. Further, caries of the spine low down, growths of the spine, or of the spinal cord membranes, may give rise to it. Inflammation of the sacro-iliac joint, rectal growths, hæmorrhoids, and ulcers, may be its originating cause. It is clear that a correct diagnosis in any case cannot be made without a complete examination of all these structures, combined with careful urinary analysis.

Thos. G. Stevens.

PAIN IN THE PENIS—is a symptom which occurs frequently in urinary surgery, not only in association with lesions of the penis or urethra, but also as a referred pain with disease of the prostate, bladder, or kidney. The symptom is one which is common to many diseases, so that in the diagnosis of any case due consideration must be given to the other symptoms accompanying it, without placing too much reliance on a single symptom which may point strongly to the urethra or bladder.

Penile pain may be present either during or immediately after micturition, or may be entirely independent of the act. It may be said generally that if pain is felt only during micturition there is some inflammatory lesion of the urethra or prostate; whilst if it occurs immediately after the flow of the urine, it suggests some lesion in the urinary bladder. On the other hand, pain may be present quite apart from micturition, due to various diseases of the penis, bladder, ureter, or kidney.

The term 'pain,' too, is a relative quantity, varying with the nervous susceptibility of the patient, for what is pain in one may be merely discomfort in another, so that the patient's account may have to be discounted to a certain extent by the clinician.

I. CAUSES OF PAIN IN THE PENIS EXPERIENCED DURING MICTURITION.

1. *Diseases of the Urethra*—
    - Acute inflammations
    - The passage of a calculus or the impaction of the latter
    - Stricture of the urethra
    - Injury of the urethra.
2. *Diseases of the Prostate*—
    - Acute prostatitis
    - Prostatic abscess
    - Prostatic carcinoma.
3. *Diseases of the Bladder*—
    - Acute cystitis
    - Vesical calculus
    - Villous papilloma
    - Pedunculated carcinoma.

Diseases of the Urethra.—By far the most common cause of pain in the penis occurring *during* micturition is acute inflammation of the urethra, usually gonorrhœal, but occasionally septic. In the earliest stages of an acute urethritis, before any marked urethral discharge is apparent, there is usually a sense of smarting or tingling in the terminal urethra, more marked as the discharge increases, when it is of a burning or scalding character. The occurrence of this pain during micturition within a few days of sexual connection is frequently the earliest symptom of urethral infection, whilst a purulent discharge from the urethra is usually present when the case comes under observation.

The *passage of a calculus* through the urethra causes a sharp, cutting pain along the urethra, the cause of which is apparent when the calculus is voided. Occasionally it may happen that micturition occurs in these cases in the dark, or that urine is not passed into a vessel, so that the calculus is not actually seen by the patient; but if there is a history of previous renal descent of a stone or symptoms pointing to vesical calculus, the sharp urethral pain during micturition occurring upon one single occasion is significant of the passage of a calculus. A stone may, however, pass on to the urethra during micturition and become *arrested* at some narrowed portion of the canal, usually at the membranous portion or at the distal end, when a sudden, sharp pain is felt in the urethra, and at the same time the flow of urine is partially or completely stopped before the bladder has been emptied, whilst further efforts fail to re-start the stream. In these cases the presence of a stone should be suspected, and the whole length of the urethra examined by passing the finger along its course, when a stone may be actually felt, or the canal may be illuminated by an endoscope and the calculus seen.

Urethral stricture occasionally causes pain in the urethra during micturition, especially if the calibre is small, and if there is septic infection or ulceration of the urethral mucous membrane behind the stricture. The forced efforts at urination may cause pain in the urethra during micturition, but as a general rule stricture causes but little pain. The gradually increasing difficulty in micturition, the feeble stream, and the dribbling of urine from the meatus after the stream has terminated, are symptoms pointing to stricture of the urethra; the diagnosis will be confirmed readily by the obstruction offered to the passage of a full-sized bougie, or better, by direct observation of the urethra by means of the endoscope.

Injury of the urethra may cause pain during micturition. The urethra may be injured by a fall on the perineum, by a kick or blow, or by the faulty or careless passage of instruments; it may also be injured or lacerated in association with a fracture of the pelvis. The urethra may be merely bruised, may be lacerated on one aspect, or may be completely ruptured. If the urethra be injured, there is usually an appearance of blood at the external urinary meatus, together with a contusion in the perineum or along the course of the urethra, if the laceration is caused by direct injury. Any attempt at micturition causes pain in the penis, whilst urine may or may not be expelled from the meatus, depending upon the extent of the injury, or may be extravasated into the perineal or scrotal tissues. As a rule, no difficulty will be experienced in the diagnosis, but in any suspected case the greatest care should be exercised in passing an instrument into the urethra.

Diseases of the Prostate.—*Acute prostatitis* and *prostatic abscess* both give rise to pain during micturition, in addition to increased frequency and difficulty during the act. Both are usually sequelæ of an acute urethritis, and whereas an acute prostatitis is accompanied by a temperature raised to 100° F. or 101° F., a prostatic abscess causes the usual rise and fall in temperature common to septic processes. The diagnosis of the two conditions is made readily on careful rectal examination, when the acutely inflamed gland presents a much enlarged, smooth-surfaced prominence in the rectum; whilst if an abscess be present, a softer area in the inflamed gland can usually be detected. An acute prostatitis may accompany a hæmatogenous bacterial urinary infection as distinct from a venereal urethritis.

Adenomatous enlargement of the prostate gives rise to no penile pain during micturition; neither does the prostate containing tuberculous deposits, but pain in the penis is present during micturition occasionally in cases of *prostatic carcinoma*, owing to the direct infiltration of the urethral mucous membrane. Prostatic carcinoma is by no means uncommon, and whilst in its general symptoms it resembles those of prostatic adenoma, there is a marked difference found on digital examination of the gland per rectum. The carcinomatous gland presents rounded areas of densely infiltrated tissue, in contradistinction to the elastic, uniform feel of the adenomatous variety; the whole gland is fixed and immovable, and in advanced stages distinct infiltration of the lateral pelvic lymphatics may be felt extending laterally from the affected organ.

Care must be taken not to mistake the hard nodules felt in a prostate containing *calculi* for carcinoma. With calculous disease, the gland is not fixed and is only slightly enlarged, whilst on gentle pressure with the examining finger the calculi may be felt to grate upon each other. During the passage of a catheter through the prostatic urethra, distinct grating may be felt if any calculus has ulcerated the urethral wall.

Diseases of the Bladder may cause penile pain during micturition under certain circumstances, although it is much more common to find that pain in vesical disease follows the completion of micturition. In *acute cystitis*, penile pain is present throughout micturition, due to the intense congestion of the vesical mucous membrane of the trigone and around the internal urethral orifice. The other symptoms of acute cystitis, namely, suprapubic pain, pyrexia, increased frequency of micturition, and the presence of pus and blood in the urine, are sufficient for the diagnosis.

Pain during micturition in other vesical lesions is caused whenever there is any sudden obstruction to the normal flow of urine by the implantation of some body against the internal urethral orifice. This may occur with a small *calculus* or with a *pedunculated tumour*, whether simple or malignant, when during micturition the flow is arrested suddenly, accompanied by a shooting pain in the urethra, whilst after an interval of a few seconds the stream may be re-established. With vesical calculus, the urine may be normal or may contain pus and blood if the bladder has become infected ; there is penile pain after micturition, and the stone may be felt with a sound. With a simple villous papilloma there is no pain unless part of the fimbriated portion of the tumour engage in the urethral orifice during micturition, but there are usually recurrent attacks of profuse hæmaturia, whilst with a villus-covered carcinoma there is increased frequency of micturition, with pain following the act, more or less constant hæmaturia, and usually pyuria. Upon rectal or vaginal examination, the base of the bladder may be felt to be infiltrated, but by far the most valuable means of diagnosis between the three conditions is cystoscopy, when a calculus or villous tumour is seen readily, whilst a pedunculated carcinoma appears as a dark red tumour covered with stunted processes. (See *Plate XVI*, p. 284.)

II. PENILE PAIN FOLLOWING MICTURITION.

This symptom is common to many lesions of the urinary bladder, more especially those in which there is ulceration or infiltration of the basal areas. The particular pain felt by the patient is described as a sharp pricking or tingling at the terminal part of the penis on the cessation of micturition, lasting some minutes and causing a desire to squeeze the glans. It has often been described as typical of vesical calculus, but this is far from being the case, for it may be due to almost any affection of the trigone.

The common causes of pain in the penis following upon micturition are :—

1. *Vesical*—
   - Calculus
   - Tuberculosis
   - Tumour—carcinoma
     - papilloma
   - Acute cystitis
   - Bilharzia.
2. *Ureteric*—
   - Calculus in lower end
   - Descending ureteritis
   - Descending tuberculosis.
3. *Prostatic*—
   - Acute inflammation
   - Abscess
   - Calculus.
4. *Vesicular*—
   - Acute spermato-vesiculitis
5. *Rectal*—
   - Carcinoma.
6. *Anal*—
   - Fissure
   - Inflamed hæmorrhoids.

In Diseases of the Bladder.

A *calculus* in the bladder, unless it is trapped in the pouch behind an enlarged prostate, causes pain in the glans penis after micturition. It may exist without causing cystitis, although commonly there is some degree of pyuria when the case is first seen. There is increased frequency of micturition during active exercise or during the jolting of travelling, but not during complete rest unless cystitis is marked. The terminal drops of urine during micturition are often tinged with blood, and on some occasions there may have been a sudden stoppage of the stream during micturition. In some cases there is a history of the descent of a stone from the kidney without the subsequent appearance of a calculus in the urine. Patients subject to vesical stone have usually reached the later part of life, and although the symptoms are as a rule sufficiently marked to render the diagnosis easy, sometimes they may be so few that vesical calculus is quite unexpected, or the symptoms are so like those caused by other lesions of the bladder that error is easy. In such a case it is advisable to examine the interior of the bladder with a cystoscope rather than by the usual vesical sound ; with a sound a small calculus, or one contained in a vesical pouch, may be missed, whilst with a cystoscope it is seen readily, its approximate size determined, and any other condition of the bladder accompanying or simulating calculus may be diagnosed with certainty. (See *Plate XVI*, p. 284, and *Fig.* 135, p. 282.)

Vesical tuberculosis may be a primary affection, but is more frequently secondary to tuberculous disease in some other part of the genito-urinary tract. It causes marked penile pain after micturition, together with pyuria and a tinge of blood in the terminal drops of urine ; the frequency of micturition is increased during both day and night, and is uninfluenced by rest, thus differing from the increased frequency of calculous disease. Vesical tuberculosis usually occurs in young adults, but it must be distinguished carefully from other vesical infections, and more particularly from renal tuberculosis, in which symptoms referable to the bladder are commonly present before the bladder is attacked by disease. In a young patient in whom increased frequency of micturition, pyuria, and penile pain are present, a search should be made for any tuberculous focus, especially in the testes, prostate, and seminal vesicles, or for marked thickening of the terminal ureter as felt per rectum, whilst a careful search should be made for tubercle bacilli in the urine. A cystoscopic examination may be necessary to determine the extent of the disease (*Plate*

XV, *Fig. E*, p. 282), but, speaking generally, the less instrumentation that is carried out in these cases the better.

Vesical Tumours.—Carcinoma of the bladder occurs in two forms: the infiltrating epithelioma and the villus-covered carcinoma. Either begins most commonly in the basal portion of the bladder, the muscular planes of which become infiltrated. For this reason, the contraction of the bladder wall during micturition causes pain which is referred to the terminal portion of the urethra. Both forms occur in elderly patients, and give rise to increased frequency of micturition during both day and night, and to hæmaturia. They also often give rise to renal pain when the infiltration has extended to the ureteric orifice in the bladder. The base of the bladder may be found per rectum to be infiltrated, or enlarged glands may be felt in the lateral pelvic space, and a cystoscopic examination will usually clear up the diagnosis (*Plate XVI*, p. 284).

Whereas the carcinomatous growths of the bladder give rise to penile pain after micturition from the direct infiltration of the vesical walls, the pedunculated villus-covered carcinoma and the simple villous papilloma may give rise to sharp penile pain during micturition, from the blocking of the internal urethral orifice with a process of growth. The occurrence of this, together with attacks of profuse hæmaturia, are evidence of a pedunculated growth. On cystoscopic examination the carcinomatous pedunculated tumour is seen to be covered by blunt, stunted processes; it is often multiple, whereas the innocent villous papilloma is single and presents much more delicate fimbriæ.

Acute cystitis causes tingling pain in the penis after micturition from the inflammatory infiltration of the trigonal area. The mode of onset, the character of the pain, and other symptoms of cystitis will point to the cause of the pain.

Bilharzia hæmatobia gives rise to clinical symptoms very similar to those of vesical tuberculosis. The history of residence in an infected district, the microscopical examination of the urine for ova (see *Fig.* 26, p. 79), and the typical cystoscopic appearance of the bladder (see *Plate XVI*, *Fig. K*, p. 284) will render the diagnosis apparent.

Ureteric lesions not infrequently produce pain in the glans penis after micturition, and may cause considerable difficulty in the diagnosis from vesical disease.

When a *calculus* becomes impacted in the narrowed terminal or intramural portion of the ureter, symptoms are produced almost exactly similar to those of vesical calculus or tuberculosis, namely, increased frequency of micturition, pain in the glans penis after micturition, and a small amount of pus and blood in the urine. Intimate knowledge of the history of the illness will often be of value in these cases; the first attack of pain is usually described as being sudden, and felt in the renal angle posteriorly, passing forward above the iliac crest and spine, and finally becoming localized at the situation of the external abdominal ring. The calculus may become impacted in the terminal inch of the ureter, when, in addition to this pain, increased frequency of micturition and penile pain are added. In a recent case under the author's care, in which a small oxalate calculus was impacted in the terminal part of one ureter, there were frequent attacks of fairly profuse hæmaturia, suggesting a villous papilloma, but this is probably infrequent. With ureteric calculus there is usually pain in the kidney of the affected side from the dilatation of the pelvis of the latter, due to the increased renal tension. The diagnosis of these cases is not so difficult if a careful inquiry is made into the history and symptoms, and so long as it is remembered that increased frequency of micturition and penile pain may be caused by ureteric impaction of a calculus. A good skiagraphic examination of the pelvic areas may show the shadow of a stone (*Fig.* 135, p. 282), whilst the latter may be felt occasionally as a small, painful nodule above the seminal vesicles upon examination per rectum. A cystoscopic examination also affords valuable information, not only in excluding vesical lesions, but by giving a distinct indication of ureteric calculus by the marked congestion and dilatation of the blood-vessels in the immediate vicinity of the ureteric orifice. A small bougie passed into the ureter may meet with obstruction in its passage, whilst a wax-tipped bougie may be grooved or indented by the stone.

Ureteritis descending from infection of the renal pelvis may give rise to slight penile pain and to increased frequency of micturition, and thus simulate vesical disease before the bladder is actually affected. This is most commonly seen in the *tuberculous* form, but is present in a less marked degree with infection by other organisms, of which the most common are the *Bacillus coli communis* and the *staphylococcus*. In the non-tuberculous form, the ureter may be felt per rectum to be slightly thickened, but the cystoscopic appearance of the inflamed ureteric orifice is quite distinctive (*Plate XV*, *Fig C*, p. 282). In *descending tuberculosis* from the kidney, the ureter may be felt as a firm, infiltrated cord on the bladder base, the penile pain and increased frequency of micturition are more marked, the kidney may be felt enlarged and tender, and tubercle bacilli will be found in the urine. Apart from this, typical changes in the ureteric orifice are seen on cystoscopic examination, the orifice being pulled up or retracted or horse-shoe shape, and usually occupying a position slightly above and outside the situation of the normal orifice, due to the actual shortening of the duct by infiltration of the submucous coats (*Plate XV*, *Fig. D*, p. 282).

Diseases of the Prostate often cause pain in the penis immediately following micturition. This is most commonly seen with acute inflammation or abscess in the gland as a sequela of acute gonorrhœa or septic urethritis. In either case there is penile pain, sometimes associated with erection, but little difficulty will be experienced in the diagnosis on due consideration of the symptoms and upon rectal examination.

Prostatic calculi are not uncommon, and there may be a single calculus or a nest of them in the prostate. They tend to ulcerate into the urethra, so that small calculi may be passed in the urinary stream, or some may pass back along the dilated prostatic urethra into the bladder. If a calculus projects from the prostate into the urethra, it causes pain in the penis after micturition. A diagnosis of prostatic calculus is often made by the grating sensation imparted to a catheter in traversing the prostatic urethra, whilst on rectal examination the calculus may be felt as an isolated, hard nodule in the gland, or, if more than one is present, by the crepitation of one upon another on digital pressure in the rectum.

Diseases of the Seminal Vesicle are seldom present without accompanying disease of the prostate or bladder. Acute vesiculitis may follow a urethritis and give rise to pain after micturition, but in most cases will be associated with prostatitis. Similarly tuberculous nodules in the vesicle will be associated with foci in the epididymis, prostate, or bladder.

Diseases of the Rectum and **Anus** may occasionally give rise to penile pain following micturition, apart from any infection of the bladder or prostate. Thus, a carcinoma in the anal canal, a rectal fissure, or an inflamed hæmorrhoid may occasionally cause pain in the penis, but in each the local symptoms of the trouble will be the more marked, and little difficulty will be found in the diagnosis if a local examination is made with care.

III. PAIN IN THE PENIS APART FROM MICTURITION.

Under the above divisions the symptom penile pain has been considered in relation to the act of micturition, and it remains to consider some conditions giving rise to pain in the penis *apart from urination*. These include certain local lesions of the penis and urethra, and also the pains referred from disease elsewhere. Although a local lesion may cause little more than discomfort in many patients, in some it is described as pain, the degree of which depends upon the nervous susceptibility of the patient. Thus, penile pain may be present with *acute urethritis*, with *balanitis* in association with *phimosis*, with *paraphimosis*, or with the *lymphangitis* of the organ due to a septic sore or abrasion of the skin or mucous membrane. In some instances *herpes* of the prepuce or penile skin causes distinct pain. Any infiltration of the cavernous tissue of the penis causes pain during erection of the organ ; thus during an attack of acute urethritis the common symptom known as *chordee* arises from this cause, whilst in a chronic form, *cavernitis* may be due to infiltration in association with tertiary syphilis or the gouty diathesis, so that erection of the organ is only partial or confined to the proximal part, and causes pain. Another condition causing the same condition arises from the organization of a *hæmatoma* in the cavernous tissues of the penis following upon a local injury, either from external violence or during forcible attempts at coitus.

Epithelioma of the penis occasionally gives rise to pain in the organ.

Pain may be felt in the penis in some cases of *renal colic*, in which case it is classed as a referred pain. Thus, in the acute colic accompanying the passage of a calculus, blood-clot, or débris of caseous material, aching pain may be felt in the penis quite apart from the increased desire to pass urine. Penile pain is, however, only a minor detail in the presence of the severe pain in the loin, and is often only lightly alluded to.

Pain in the penis was a prominent early symptom in two recent cases of *acute appendicitis* under the writer's care. In neither case was it associated with micturition, nor was there any increased frequency of micturition, but in both the appendix was found to occupy a very low position, turning down into the pelvis, which in one case contained a foul abscess.

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PAIN IN THE PERINEUM—is a symptom often mentioned by patients in giving their history of some affection of the genito-urinary apparatus or of other organs, but usually only as a dull aching, of which little notice is taken, as it is generally of minor consequence in comparison with other more striking symptoms. The complaint of perineal pain *per se* does not convey much information to the clinician, and it is practically never present as the only symptom in a case.

Aching in the perineum is frequently present in diseases of the following organs :—

- *Prostate*—
  - Acute or subacute inflammation
  - Abscess
  - Tuberculosis
  - Calculus
  - Adenomatous enlargement
  - Carcinoma.
- *Seminal Vesicles*—
  - Acute inflammation
  - Tuberculosis.
- *Urinary Bladder*—
  - Cystitis
  - Tuberculosis
  - Calculus
  - Carcinoma
- *Urethra*—
  - Injury and rupture
  - Stricture with extravasation or urethral abscess
  - Fistula
  - Calculus impacted in bulbous portion.
- *Testicle*—
  - Congenital misplacement in perineum.
- *Anal Area*—
  - Hæmorrhoids
  - Fissure
  - Boil
  - Carbuncle
  - Ulcer
  - Carcinoma.
- *Vagina*—
  - Acute inflammation
  - Inflammation or abscess of Bartholin's glands
  - Cystocele
  - Epithelioma.
- *Cutaneous Diseases*—
  - Intertrigo
  - Eczema, gouty and diabetic
  - Condylomata.

From the foregoing list it will be seen that aching in the perineum occurs with numerous different lesions, but other symptoms discussed elsewhere are in almost every case more marked.

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PAIN, PRECORDIAL.—(See PAIN IN THE CHEST, p. 430.)

PAIN IN THE SHOULDER (see also PAIN IN THE EXTREMITY, UPPER, p. 442) may be due to two entirely different main groups of causes, namely : (1) *Direct causes*, in which the shoulder-joint itself is involved, or the nerves, ligaments, muscles, fasciæ, bursæ, close to it ; and (2) *Indirect causes*, in which the pains are referred to the shoulder region when the real seat of disease is at a distance, as in the case, for instance, of angina pectoris, or gastric or hepatic disorders. The conditions to be thought of include the following :—

1. Direct Causes :—

- Injury
- Arthritis
- Synovitis
- Fibrositis
- Myositis
- Neuritis
- Effects of exposure to cold or damp
- Effects of occupations
- Subacromial bursitis
- Muscular paralysis, local.

2. Indirect Causes :—

(*a*). *Cardiovascular lesions.*

- Angina pectoris
- Aortic valvular disease
- Aortic atheroma (syphilitic)
- Aortic aneurysm.

(*b*). *Pleural, pulmonary, or mediastinal lesions.*

- Pleurisy
- Pneumonia
- Phthisis
- Intrathoracic new growth
- Pneumothorax.

(*c*). *Gastric lesions.*

- Flatulence
- Indigestion
- Gastritis, acute or chronic
- Gastric ulcer
- Gastric carcinoma

(*d*). *Duodenal lesions.*

- Catarrh, with or without jaundice
- Duodenal ulcer.

(*e*). *Hepatic lesions.*

Biliousness
Gall-stones
Cholecystitis
Nutmeg liver
New growth
Hepatitis, acute tropical
Abscess.

(*f*). *Nervous lesions.*

Hemiplegia
Herpes zoster
Acute bracheitis
Cervico-dorsal spinal caries
New growth in cervico-dorsal spine
Pachymeningitis, syphilitic cervico-dorsal
Cervico-brachial neuralgia.

The term shoulder is not explicit, and a patient may complain of pain in the shoulders when careful inquiry shows that it has widely different situations in different cases ; in one it may be mainly in the root of the neck or in the central clavicular region ; under the deltoid region in a second ; under the lower part of the blade of the scapula in a third ; and so on. The first step in making the diagnosis is to locate the pain complained of as definitely as possible.

It is often easy to diagnose its cause with some certainty from this point alone. If, for instance, the pain is mainly under the left shoulder-blade, its origin is likely to be gastric or intrathoracic ; if mainly under the right shoulder-blade, duodenal or hepatic. (See PAIN, INTERSCAPULAR, p. 461.) If on the other hand the pain is definitely localized to the region of the shoulder-joint, the mischief is probably within the joint itself, or in the fibrous tissues, ligaments, bursæ, muscles, or nerves around the joint.

The next point to investigate is the condition of the parts at and round the shoulder ; if there is definite tenderness as well as pain, increased pain on attempted movement, local deformity from swelling or from wasting, or impaired mobility at the shoulder-joint, the trouble is probably local and the pains are not referred from the viscera. It may be difficult to be sure that there is no local tenderness as well as pain, for the patient may have suffered so much that he winces on palpation, from an expectancy of pain, when palpation or attempts at moving the joint are really painless ; considerable judgement is required in deciding just how much tenderness is present as well as pain, especially in nervous subjects. If, however, there is definite tenderness as well as pain, the trouble is most likely local ; and the same applies when the pain is made materially worse by local movements.

In testing the mobility of the shoulder-joint, it is important not to rely on the patient's statements or on inspection only ; careful palpation is required. Frequently the patient may seem to move his shoulder well when, if the angle of the scapula is grasped whilst the patient moves his humerus, it will be found that the shoulder-blade moves synchronously with abduction of the arm, there being little or no play at the glenoid fossa. Normally, when the humerus is abducted, the scapula should remain stationary until the arm is at a right-angle with the trunk ; if the upward movement of the extended and abducted arm is continued beyond the right-angle, the scapula normally moves with it. If it begins to move before the right-angle is reached, there is something the matter in or around the shoulder-joint. More often than not the mischief is due to peri-arthritic changes rather than to osteo-arthritis or other intra-articular inflammation ; the fixation is due to the muscles being on guard to prevent any movement that would produce pain. Under an anæsthetic mobility may be found to be nearly perfect.

The differential diagnosis between the various conditions that may affect the joint is discussed in the article on JOINTS (p. 337). Fibrositis and myositis are in most respects equivalent to infective peri-arthritis, and it is often a matter of opinion whether any particular infective inflammatory condition around the joint is to be styled infective synovitis, or fibrositis, or myositis. Any of the three may involve one shoulder region only, but more often the patient has signs and symptoms of rheumatoid arthritis elsewhere also.

The effects of injury, exposure to cold and damp, and of occupations, when they involve the shoulder region, are only particular varieties of fibrositis, myositis, and arthritis. The stiffness and pain that may result from sleeping in a damp room with the shoulders uncovered by the bedclothes may be very severe, and they may take weeks or months to pass off. The chief question will be whether the exposure may not have caused actual rheumatoid arthritis, or have brought on a gouty inflammation or neuritis, in addition to mere stiffness and pain of a type allied to stiff neck or lumbago ; and the differentiation will often be a matter merely of opinion. The same applies to the effects of injury: in some cases the bruising or the tearing of ligaments, or the fracture of bones, or their dislocation, may be followed by pain and stiffness continuing to affect the shoulder region for months or years afterwards; in addition, however, the injury may set up actual arthritis allied to osteo-arthritis, and like the latter, more or less permanent. The *x*-rays may help in determining the exact degree of local disease the injury has produced. Occupations involving constant use of the shoulder often result in permanent intra-articular changes also—a sort of osteo-arthritis; the tendency increases with age; and very likely the occupation—such as that of porters who carry heavy weights on the shoulder—determines the site of the first joint to be affected rather than actually initiates the disease. Besides intra-articular changes, however, occupations involving strenuous and constantly repeated use of the shoulders may cause pain in them of the same nature as that of writer's cramp and other occupation neuroses. Rowing men, navvies, stokers, and so on, may suffer in this way. It is often very difficult, however, to distinguish between the occupation neurosis and organic fibrositis, myositis, neuritis, or arthritis.

One well-defined but often unrecognized cause of painful shoulder, in which both recurrent slight injury and occupation play an important part, is *subacromial bursitis*. Outside the upper part of the capsule of the shoulder-joint, between it and the under surface of the acromion process, there is a fairly large bursa. This becomes inflamed from overuse or overstrain of the shoulder in much the same way that housemaid's knee results from persistent kneeling; but with this difference, that the swollen subacromial bursa cannot be seen or grasped as an inflamed prepatellar bursa can. Pain on using the shoulder in certain positions is the chief complaint. The trouble arises from falls or blows upon the apex of the shoulder, or from occupations involving repeated sudden jerks to the shoulders—it is common in golfers, tennis players, bowlers, or in those who throw the cricket ball or put the weight; it also arises when the daily work involves the maintenance of a strain against interrupted resistance, as in the case of certain tools employed in electric steel grooving and the like. The diagnostic point is as follows: on minute palpation of the shoulder region, when the arm is at the patient's side there is a definitely and often acutely tender spot immediately below the tip of the acromion process, but if the patient now abducts the arm as far as he can, palpation of the previously tender spot no longer elicits any tenderness at all; on adducting the arm to the side the tender focus is again discoverable with ease. The explanation of the disappearance of the tenderness on abduction of the arm is that this movement takes the subacromial bursa far enough under cover of the acromial process for it to be no longer open to direct palpation. In cases of this sort *x*-ray examination generally shows a wider clear space between the acromion process and the head of the humerus on the affected than on the normal side.

In any case of doubt *x*-ray examination, not only of the shoulder region but also of the whole thorax, should be resorted to; now and again an unsuspected aneurysm of the aortic arch interfering with the lower part of the right brachial plexus will be found to be the cause of acute pains in the right deltoid region; or a mediastinal new growth, or rarities such as a hydatid cyst in the chest (*Fig.* 137, p. 291) may be discovered when mere arthritis has been alone suspected. *Angina pectoris* is responsible for very acute pain in the left shoulder region, but hardly ever here alone; the attacks, and the spread of the pain from the precordial region to the left shoulder and down the left arm are generally characteristic; and their nature will be confirmed by examination of the heart, including resort to electrocardiograms if need be, and by the influence of amyl nitrite or nitroglycerin on the paroxysms.

Pains referred to the shoulder in cases of *phthisis* are generally of the nature of dull discomfort or of aching than of acute pain; and the patient generally has had cough, wasting, night sweats, and so on, to indicate the diagnosis, which is confirmed by the existence of abnormal apical signs, by the discovery of tubercle bacilli in the sputum, or by *x*-ray examination of the chest (*Fig.* 41, p. 103). *Pleurisy*, *pneumonia*, and *pneumothorax* will hardly ever cause pain in the shoulder as their only symptom, and they will be diagnosed on other grounds.

Gastric, *hepatic*, and *duodenal* lesions causing pain in the shoulder, do so in the subscapular region rather than in the region of the shoulder-joint itself—stomach disorders on the left side, hepatic and duodenal on the right. They are discussed in the article on PAIN, INTERSCAPULAR (p. 461). The chief difficulties that remain are the nervous causes, direct and indirect. *Herpes zoster* will be recognized by the characteristic eruption (p. 754), and the pains will nearly always extend down the arm and not be confined to the shoulder region. The chief point to bear in mind is that the pains resulting from herpes may persist for months after the eruption has disappeared, and may be severe before the vesicles come out. If there is local wasting of the muscles of the shoulder region, it does not follow that the primary lesion is nervous, because precisely similar muscular atrophy results very rapidly from joint lesions; with the latter, however, there is no R. D. (p. 582), whereas with true *neuritis* there is. It is, however, only when all intrathoracic lesions have been excluded, and when physical and *x*-ray examination point to there being no affection of the shoulder-joint, that neuritis of the circumflex nerve, for instance, should be diagnosed; an inaccurate diagnosis of neuritis is so often made simply because the patient has pains and no apparent cause can be found to account for them. *Acute bracheitis* is allied to acute sciatica and acute anterior cruritis; it is diagnosed by analyzing the distribution of the pains carefully, and by excluding gross lesions of other structures in the shoulder region or in the chest. It is often associated with marked muscular atrophy, so that the humerus tends to fall away from the glenoid fossa and the patient invents all sorts of slings or splints for the better support of his arm. When there is no muscular wasting, the same lesion would often be termed *cervico-brachial neuralgia.* Some such cases are due to much more serious lesions, which may baffle diagnosis until the patient has been watched for weeks or months—*new growth* in the cervico-dorsal spine, for example; or cervico-dorsal *tuberculous caries*; *x*-ray examination of the vertebræ may assist in the earlier recognition of these conditions, but sometimes it is not until post-mortem examination is made that the exact cause of the pains—possibly regarded previously as in the main neurotic—is demonstrated. The *hemiplegic* arm is sometimes the site of great pain, especially in the shoulder; the pain is probably referred from the sensori-motor cortex of the cerebrum; the differential diagnosis will be found in the article on HEMIPLEGIA (p. 302). Cervico-dorsal *pachymeningitis* of syphilitic origin may cause no more symptoms than ill-defined though acute pains in various parts of the neck, shoulder, arm, or hand; peripheral neuritis or myofibrositis may be the diagnosis made. In most cases it is almost impossible to diagnose the pachymeningitis with certainty, though it may be guessed at; and in obscure cases of pain in the shoulder, apparently of nervous origin, it is often wise to have a Wassermann test made. If it is positive, appropriate antisyphilitic treatment may cure the patient; and it is remarkable how often syphilis is found to be at the root of obscure pains and other ill-defined nervous complaints, even when any suspicion of syphilis might seem to be out of the question. *Herbert French.*

PAIN IN THE SPINAL REGION.—(See PAIN IN THE BACK, p. 427.)

PAIN IN THE STERNAL REGION.—(See PAIN IN THE CHEST, p. 430.)

PAIN IN THE TESTICLE—of varying degree may be present in many conditions, and may be discussed under separate headings as follows:—

I. *Diseases of the body of the testis or epididymis.*
II. *Affections of the coverings of the testicle.*
III. *Affections of the spermatic cord.*
IV. *A retained or misplaced testicle.*
V. *Pain from lesions remote from the testis.*

I. DISEASES OF THE BODY OF THE TESTIS OR EPIDIDYMIS.

Inflammatory Lesions may attack the testis proper, or, as is more common, may begin in the epididymis; they rarely remain confined to one part of the organ, however, for the process tends to spread rapidly from one part to the other, so that the whole organ is involved and the result termed an "*epididymo-orchitis.*" An inflammatory affection of the testicle may be acute, subacute, or chronic, the latter being the terminal result of the former.

An acute epididymo-orchitis arises most commonly by the direct infection of the organ from the urethra *via* the vas deferens. When any inflammation has reached the prostatic portion of the urethra, the orifices of the vasa deferentia may become infected, and inflammation spreads rapidly along the duct to the epididymis and testis. Whilst formerly the occurrence of an acute inflammatory condition of the testis, following upon some form of urethritis, was looked upon as "metastatic," it has been shown that this view is no longer tenable, and that we must look upon it as a direct spread of infection *via* the vas deferens.

Causes of Acute Epididymo-orchitis :—

Causes of urethral origin :—
- Gonorrhœal urethritis
- Septic urethritis
- Passage of catheters
- Urethral instrumentation
- Ulceration behind a stricture
- Ulceration about an impacted calculus or a prostatic calculus.
- Injections into the posterior urethra.

General causes :—
- Fevers—Parotitis (mumps)
  - Enterica
  - Scarlet fever
- Injury
- Influenza
- Gout and rheumatism
- In hæmatogenous urinary infections.

Acute epididymo-orchitis begins as a painful thickening of the epididymis associated with febrile symptoms. Before any actual pain is noticed in the testis there is often a sense of discomfort and weight over the external abdominal ring and inguinal canal due to the inflammatory process extending along the vas deferens. The swelling of the epididymis increases, and with it the tubules of the testis proper become infected, causing swelling of its body and increase of pain. The whole organ thus becomes enlarged, and it is often exquisitely tender, the touch of the clothes or the most gentle examination causing pain. The swollen gland is often flattened on the outer and posterior aspect from pressure against the adductor muscles of the thigh ; the vas deferens and tissues of the spermatic cord are thickened.

By far the most common cause of an acute epididymo-orchitis is an *acute gonorrhœal urethritis*. During the third week of the disease the prostatic portion of the canal frequently becomes infected, when the orifices of the ejaculatory ducts may share in the inflammation, and infection be conveyed by the vas deferens to the testicle. Similarly, but less frequently, infection may arise from a *septic posterior urethritis*, contracted during connection with a woman the subject of a vaginal leucorrhœa. The gonorrhœal form of acute epididymo-orchitis usually resolves slowly, and shows very little liability to suppurate, whereas the inflammation resulting from a staphylococcal or a streptococcal infection may break down into a testicular abscess.

Acute epididymo-orchitis may also arise from septic processes in the urethra following upon the *passage of catheters*, of *instruments* for vesical operations, such as lithotrity, from ulceration behind a *urethral stricture* or about a *calculus* in the prostatic urethra, and occasionally after the *instillation of strong solutions* into the posterior urethra in the treatment of a chronic urethritis. In any case the onset of pain and rapid swelling of the testis should always lead to the suspicion of urethral infection, and attention should be directed to the urethra with that in view. Bacteriological examination of any urethral discharge is essential (see DISCHARGE, URETHRAL, p. 181).

Acute epididymo-orchitis occasionally arises *without any preceding urethral infection*, and uncommonly occurs as a complication of *acute specific parotitis* (mumps), *enterica*, *scarlet fever*, *influenza*, or as a complication of a urinary infection by *Bacillus coli* or other organisms. The testicle becomes painful, and enlarges rapidly in the same manner as in acute inflammation from urethral infection, and under appropriate treatment gradually resolves. Less frequently testicular inflammation may occur with *gout* or *acute rheumatism*, or after a direct injury to the organ, such as a *blow* or *squeeze*.

The pain in an acute inflammation is generally of an aching character at first, felt not only in the testis, but at the external abdominal ring, and often as a heavy dragging pain in the loin of the affected side. As the testis enlarges, the local pain becomes more severe, so that the swollen gland is exquisitely tender to pressure or to the touch. After a few days the pain subsides to a large extent, but remains as a dull ache until the swelling becomes greatly reduced, and usually disappears some time before the organ returns to the normal size. In a few cases in which a fibrous scar remains in the epididymis, pain may remain and cause some difficulty in the diagnosis from a commencing tuberculous lesion, but the earlier history of acute inflammation will help in forming an opinion. In other cases the persistence of the pain and swelling may indicate the formation of an abscess in the testicle, when, after decrease at first, the swelling increases, the skin covering it becomes reddened, and a soft area becomes evident in one or other side of the organ.

Tuberculosis of the Testicle is comparatively common, occurring as a primary disease or secondary to tuberculous disease of the kidney, bladder, or prostate. It begins as a localized deposit in almost all cases, causing a rounded, firm nodule in the epididymis. It frequently arises in the upper pole of the epididymis, whereas the inflammatory affections secondary to urethral infection begin in the lower pole. This nodule may remain unaltered for many months, or may enlarge, soften, become adherent to the skin and coverings of the testicle, or actually ulcerate through them to form a discharging fistula in the scrotum. The small commencing nodule in the epididymis is usually painless at first and may be found by accident, but later, as it gradually enlarges, it causes an aching pain in the organ. Other nodules may be formed in the epididymis, or the body of the testis may become involved, whilst commonly small shot-like thickenings may be felt in the course of the vas deferens. In the most advanced stages, nodules may be felt upon rectal examination in the seminal vesicles or prostate, or there may be some in the epididymis of the other side.

Tuberculous disease of the testicle usually presents little difficulty in the diagnosis. In an early case the occurrence of one or more nodules in the epididymis, which are painful on pressure and which have not resulted from a preceding acute epididymo-orchitis, should always suggest a tuberculous focus, and a careful search should be made for any other tuberculous lesion in the body. Should none be found, the estimation of the opsonic index of the blood to tubercle, or one of the several clinical tests for tubercle, such as von Pirquet's tuberculin skin reaction, may clear up the diagnosis. In later stages the diagnosis is less difficult ; the gradual enlargement of the nodules, their craggy or bossy feel, the infection of the vas or other genito-urinary organs with tuberculosis, and above all, the tendency of the focus in the epididymis to soften and to become adherent to the scrotal coverings, are points to be looked for ; whilst if it should have led to the formation of a fistula, tubercle bacilli may be detected in the discharge.

Syphilitic Disease of the Testis causes very little pain in the organ, but there is often a sense of dragging or heaviness, and for this reason it must be considered.

Syphilis may attack the testicle in several different ways, producing :—

In Acquired Syphilis :—	*In Congenital Syphilis :—*
Diffuse interstitial orchitis	Interstitial orchitis
Gummatous orchitis	Gummatous orchitis.
Epididymitis.	

The outstanding feature of syphilitic disease of the testicle is that it affects the body of the testis rather than the epididymis, thus differing in a marked degree from tuberculous disease. In the interstitial form there is thickening of the intertubular connective tissue, with an infiltration of spindle cells, which, forming young connective tissue, yield fibrous tissue when untreated. The subsequent contraction of this fibrous tissue may cause atrophy of the testis. The testis may, on section, show small gummata in addition to the diffuse orchitis, or if the inflammation is more localized, gummata may be the main feature, these varying in size from that of a pea to that of a walnut, or larger. The epididymis is affected but rarely, though cases are on record of a nodular swelling in the epididymis during the secondary stage of syphilis, which disappeared rapidly under antisyphilitic treatment.

In congenital syphilis, both the interstitial and gummatous forms exist ; they usually occur in childhood or in young adult life, and in many cases the affection is bilateral. Syphilitic inflammation of the testicle may be accompanied in either the acquired or the congenital form by a vaginal hydrocele.

There is a sense of weight in the scrotum rather than pain, and often an aching or dragging feeling in the inguinal or lumbar region. On palpation, the body of the testis feels enlarged and nodular with the gummatous deposits, but the epididymis can usually be distinguished from the testis and be found to be unaffected. The tissues of the cord remain unthickened.

The diagnosis of syphilitic disease of the testis is usually simple. There may or may not be a history of syphilis, but other signs of the disease should be looked for—thus, in the acquired form, any scar of previous ulceration or periosteal thickening, or in the congenital variety, signs in the teeth, eyes, or ears. If any doubt remains, a positive

Wassermann reaction of the blood or the behaviour of the swelling when treated with large doses of potassium iodide and mercury, or salvarsan, should be noted. Syphilitic disease is distinguished from *tuberculous disease* of the testis by the fact that the epididymis is usually free from infection; that the cord, prostate, and vesicles remain normal; and by the comparative absence of pain in the testicle upon pressure being made upon it. Tuberculous deposits tend to soften and to involve the scrotal coverings in spite of treatment. From *chronic orchitis* it is differentiated by the history of injury and by the absence of the history or signs of syphilis. From *malignant tumours of the testis* it is distinguished by the history of syphilis, the tendency of syphilitic disease to be bilateral, the slow enlargement, and a positive Wassermann reaction. In malignant disease, the increase in the size of the testicle is more rapid, whilst the tumour often shows areas of varying consistence; the cord is often enlarged in malignant cases.

Malignant Tumours of the Testis may give rise to pain in the organ, but as a rule pain is only experienced in the later stages of the disease. Both carcinoma and sarcoma may arise in the testis, but embryoma is more common, exhibiting both epithelial structures and a combination of several forms of connective-tissue type—cartilaginous, myxomatous, etc. Clinically, without microscopical examination, a soft carcinoma and a sarcoma can rarely be differentiated, and as their symptoms and history are so similar, they may for convenience be considered together. A testicle that is the seat of a malignant growth enlarges rapidly, but as pain is at first absent, there may be nothing to arouse the patient's suspicions. As long as the tunica albuginea remains intact the swelling retains the shape of the testis, but when perforation of the fibrous covering takes place nodular projections appear and render the tumour irregular. These projections are softer than the remainder of the growth, and form a valuable point in the diagnosis. A rapidly growing sarcoma or carcinoma of the testis may be so soft as to appear to be a fluid collection in the tunica vaginalis. Generally, however, although a growth may be accompanied by a small amount of fluid in the tunica vaginalis, the more solid mass can be felt through the fluid on careful examination; this fluid is often bloodstained. The epididymis may become incorporated in the growth so that it cannot be distinguished, and the tissues of the cord become thickened. The coverings of the testis become stretched over the tumour; the mass does not become adherent to the scrotal skin until late in the disease. In both carcinoma and sarcoma, the iliac and lumbar glands become enlarged, and may be felt in a thin subject at the brim of the pelvis, and pain due to the pressure of these glands upon nerve structures may become marked. The inguinal glands are usually not enlarged unless the scrotal skin is affected. The diagnosis of malignant disease of the testis may be quite easy in the case of rapidly growing tumours, but in others, especially in the early stages, it may present great difficulty.

Between *sarcoma* and *carcinoma* it may be clinically impossible to distinguish. In quite early life the tumour is more likely to be a sarcoma; the cord is thickened earlier in carcinoma, but with rapidly growing tumours it may be quite impossible to say whether it is a sarcoma, carcinoma, or embryoma until a piece is examined under a microscope.

Orchitis may be confused with the more slowly-growing forms of sarcoma. In both the swelling may have followed an injury, and in both there may be a syphilitic history. Orchitis is, however, either more acute or more chronic, it retains more the oval shape of the testis, and does not present the rounded, slightly raised bosses which are commonly present in a sarcomatous testis. In orchitis the epididymis is usually distinguished more easily, and the cord is not so thickened as with a growth. Finally, the result of treatment with strapping and with mercury and iodide will often show the disease to be of a non-malignant nature.

Chronic torsion of the testicle is not very uncommon amongst habitual horse-riders, and sometimes, if there is no clear history as to the relationship between the swelling and a saddle injury, the nature of the painful tumour may be so uncertain that operation and histological examination are resorted to.

Tuberculous disease is usually diagnosed easily from malignant disease by the tendency of tubercle to attack the epididymis, to caseate, suppurate, and to become adherent to the scrotal skin comparatively early. Tuberculosis occasionally attacks the body of the testicle first, however, forming an oval, smooth tumour of the organ; the epididymis and vas deferens may be unaffected for a time, and if no deposit is found in the prostate or vesicles, the differential diagnosis between tubercle and growth may be far from easy before operation.

Hæmatocele.—The diagnosis between a hæmatocele and a malignant tumour of the testis may present considerable difficulty. In both the swelling may date from an injury, whilst the indistinct fluctuation obtained in the soft areas of a growth, accompanied sometimes by some fluid in the tunica vaginalis, may simulate a hæmatocele. The latter feels heavy to the hand, but is usually softer in its whole mass and more regular than a growth. Care must be taken not to place too much reliance upon the withdrawal of a few drops of blood from the tumour by means of a trocar and cannula, a result which may happen equally with growth or hæmatocele. A hæmatocele may cease to enlarge, or even diminish in size, whereas, in growth, increase in size is progressive. The cord remains unaffected with hæmatocele, and testicular sensation is more likely to be lost in growth. If any doubt exist, it is advisable to make an exploratory incision rather than a puncture, when, if necessary, a radical operation can be proceeded with.

Hydrocele.—A hydrocele of very long standing, with an irregular, nodular surface, and absence of translucency due to the thickened tunica vaginalis and the thick contents of the sac, may simulate a new growth, but the long history of the case, and the absence of progressive increase in size of the swelling, will prevent a mistake of this kind.

Cysts of the Testis occur most frequently in connection with the epididymis, very rarely with the body of the testis. These cysts are quite different from hydrocele of the tunica vaginalis, and are often spoken of as encysted hydrocele of the epididymis or testis, or as a spermatocele, although all do not contain spermatozoa. They cause a swelling of varying degree in the scrotum, and usually an aching in the testicle, groin, or lumbar region. They may arise as retention cysts of the tubules of the epididymis or from one of the fœtal remains which occur about the globus major of the epididymis, namely, the organ of Giraldès, the hydatid of Morgagni, or the vas aberrans of Haller. These cysts are usually placed above and to the outer side of the testis, occasionally behind it. They move with the organ, and can usually be distinguished from the latter by the test of translucency. Their increase in size is very slow, but they may cause aching pain in the testicle by pressure upon, or stretching of, the tissues of the epididymis. They can be distinguished from hydrocele of the tunica vaginalis by the position of the swelling relative to the testicle, and by the fact that the fluid contained in them is colourless or slightly opalescent from the contained spermatozoa, in distinction to the straw-coloured fluid of a vaginal hydrocele.

II. AFFECTIONS OF THE COVERINGS OF THE TESTIS CAUSING PAIN IN THE ORGAN.

The only common lesions of the coverings of the testis are *hydrocele* and *hæmatocele* ; new growths of the testicular tunics are so rare as to render them surgical curiosities.

Hydrocele may occur occasionally as an acute affection accompanying an acute epididymo-orchitis, injury to the scrotum, or in the course of acute specific fevers such as small-pox, rheumatism, or mumps. Recently acute hydrocele has been described in conjunction with acute lesions of other serous membranes—multiple serositis or polyorrhomenitis. The more usual form of hydrocele is the chronic variety, which may be due to some disease of the testicle, but for which, in the majority of cases, no ascertainable cause can be found.

A hydrocele may cause some aching in the testicle, but more frequently it causes a dragging sensation in the loin from the mechanical effect of its weight. It forms a swelling on one side of the scrotum, oval with smooth uniform surface ; it gives a distinct sense of fluctuation. The swelling is limited distinctly above from the cord or external abdominal ring, and gives no sense of impulse on coughing ; with a good light it can be found in most cases to be translucent, the testicle occupying a posterior and low position in the swelling.

The diagnosis of hydrocele is usually easy, but occasionally, when in old-standing cases the walls are much thickened, difficulty may be experienced. A hydrocele must be diagnosed from (1) A scrotal hernia, (2) Hæmatocele, (3) New growth, and (4) An encysted hydrocele of the testis.

Scrotal Hernia.—Usually a hernia gives an impulse on coughing, can be reduced into the abdomen with a sudden slip or gurgle, and varies in size with the position of the patient. A hernia comes down from above and descends into the scrotum. In a large irreducible hernia, some part of it is usually resonant from the contained intestine, the swelling is not limited above, and the testis can be distinguished at the bottom of the scrotum. A hydrocele is distinctly limited above, gives no impulse, is translucent, and the spermatic cord can be distinguished easily. The testis in a hydrocele cannot usually be distinguished in the scrotum as in a hernia. Difficulty may arise between the two conditions when the hydrocele extends along the inguinal canal, and thus gives an impulse on coughing, or if the translucency is lost owing to the thickness of the walls or contents of the sac. A scrotal hernia in an infant may be translucent.

Hœmatocele is distinguished from hydrocele by the absence of translucency, the greater weight, and the suddenness of the onset, usually after an injury or puncture. If any doubt exist, an incision may be made into the swelling, permission being obtained to proceed to any form of cure that may be found desirable.

New Growths of the Testis.—A hydrocele is of much slower rate of increase in size, of smooth surface and uniform consistence, and is translucent.

Encysted Hydrocele of the Testis (see above).

Hæmatocele may occur as the result of tapping a hydrocele, from puncture of a vein in the sac or of the testicle, or by the occurrence of bleeding into a hydrocele. It may occur quite independently of a hydrocele, usually after direct injury. As a rule there is a rapid onset of swelling in the scrotum following the injury, with ecchymosis of the scrotal skin ; the resulting tumour resembles a hydrocele in its clinical symptoms, save that it is not translucent. In other cases the swelling arises more slowly, when a pyriform or oval swelling is present in one side of the scrotum covered by normal skin ; the surface of the swelling is smooth, and gives a sense of fluctuation and elasticity. There is no translucency, and, on tapping, dark blood-stained fluid is withdrawn.

The diagnosis in the less acute cases often presents a difficulty, especially with regard to *malignant disease of the testicle* (see above). From *hydrocele* it is distinguished by the absence of translucency ; from *hernia* by the same points, except translucency, mentioned above in the diagnosis between hydrocele and hernia.

III. AFFECTIONS OF THE SPERMATIC CORD CAUSING TESTICULAR PAIN.

An inflammatory affection of the cord secondary to urethral infection is not uncommon. Similarly, tuberculous infection of the cord is practically never present without corresponding infection of the testis or epididymis. New growths of the cord, lipomata, myxolipomata, and sarcomata or hydroceles of the cord, cause no pain in the testis. A *varicocele*, especially if large, in a pendulous scrotum, is a frequent cause of a dull, aching pain in the testicle. The characteristic feel of the enlarged veins of the cord in the erect position, and the slight impulse on coughing, will readily point to the correct diagnosis.

IV. THE RETAINED OR MISPLACED TESTIS.

This, in its various situations, may give rise to pain, and may cause some difficulty in the diagnosis of the condition present. A testis may be arrested in its descent at the external abdominal ring in the inguinal canal, may remain inside the abdomen, or may pass (1) into the perineum after traversing the inguinal canal, (2) to the upper part of the thigh *via* the crural ring, or (3) to the root of the penis in front of the pubes.

In the various situations in which an undescended or ectopic testicle is placed, it may be attacked by the several diseases which affect the normally placed organ, and thus give rise to pain ; but in addition, owing to the effect of constant muscular strains and the comparative immobility of the organ, it is particularly liable to recurrent attacks of traumatic inflammation, especially when the testis is retained in the inguinal canal : in the intra-abdominal position it remains protected from muscular injury, whilst ectopic testicles have a greater range of mobility than has one that is retained in the inguinal canal. The inflammation of an undescended testicle may be so acute as to lead to gangrene of the organ, with or without torsion of the cord.

The pain may be complained of first when the testes begin to swell at puberty, at which time an undescended right testicle may produce symptoms easily mistakeable for appendicitis.

The diagnosis of undescended testicle rests upon the following points : the fact that one side of the scrotum is empty, the outline and situation of a swelling in the inguinal canal or elsewhere, the testicular sensation upon pressure, and the recurrent attacks of pain. An undescended testicle may give rise to acute pain from inflammatory lesions or from acute torsion of the organ, and may, if placed in the inguinal canal, give rise to symptoms suggestive of a strangulated hernia. It must be remembered also that a partially descended testicle is often accompanied by an inguinal hernia. It is also stated that the misplaced testis is prone to become the seat of malignant disease.

V. TESTICULAR PAIN FROM LESIONS OTHER THAN IN THE TESTICLE.

It is necessary to mention this class of case, in which complaint is made of testicular pain, when on clinical examination the testis is found to be normal. After an acute inflammation of the organ, even when no palpable nodule remains, the resulting cicatrization may cause an aching in the organ, especially after *sexual excitement* or prolonged desire. Apart from former testicular disease, pain may be felt in the organ if a *calculus* be present *in the pelvis of the kidney* or *upper ureter*, with a marked degree of *oxaluria*, or from *stimulation of the peripheral nerves by carcinoma of the bodies of the lumbar vertebræ* or the pressure of an *aneurysm* in this situation. Pain in the testicle is occasionally present in *appendical inflammation*, when the appendix turns down into the pelvis.

R. H. Jocelyn Swan.

PAIN IN THE THIGH.—(See Pain in the Lower Extremity, p. 438.)

PAIN IN THE THROAT.—(See Sore Throat, p. 613.)

PAIN IN THE UMBILICAL REGION.—

Pain associated with External Swelling.—This may be due to the following causes :—

Umbilical Hernia.—This is common in young infants, and also in fat, middle-aged people, particularly women who have borne children. Attention is often called to the protrusion first by the pain. In the early stages, while the hernia is yet small, it may easily be overlooked, especially if the patient is obese. The swelling is usually globular in shape, has an impulse on coughing, and may or may not be reducible. In a very large proportion of cases the hernia is irreducible, and it nearly always contains omentum.

Sebaceous Cyst.—This is not uncommon at the umbilicus. It presents itself as a small spherical swelling, which might be mistaken for a small irreducible umbilical hernia. There is, however, no direct impulse on coughing, and the swelling is attached to the skin. There is generally but little pain unless the cyst has become injured or inflamed.

New Growth.—Occasionally the umbilicus is the seat of a small secondary nodule of growth which has been brought by the vessels in the round ligament from the neighbourhood of the liver. It may furnish a clue in making the diagnosis of an obscure abdominal ailment. *Epithelioma* is the only likely primary growth here ; it has to be distinguished from a mass of *simple granulation tissue* here, and from *tuberculous ulceration of the skin* at the umbilicus ; all three are uncommon ; in some cases the diagnosis between them may be obvious almost at sight ; in others the distinction may be so difficult that microscopical examination of a portion of the mass has to be resorted to.

Eczema Intertrigo.—Very obese people of uncleanly habits may suffer from this trouble at the umbilicus.

Divarication of the Recti Muscles.—A patient lying in the recumbent position may show no evidence of this condition. Diffuse pain is complained of about the umbilicus, and the divarication is made evident at once if the patient is asked to lift the head.

Cyst of the Omphalo-mesenteric Duct, a rare condition, may be found at birth.

Pain associated with Internal Swelling.

Tuberculous Peritonitis with formation of a localized abscess is the commonest cause of pain and a swelling situated inside the abdomen at the umbilicus. The patient is usually a child, and presents well-marked signs of chronic abdominal disease.

Carcinoma of the Pylorus or *Carcinoma of the Colon* may present as an umbilical tumour, especially if it has become fixed by adhesions, but it can usually be diagnosed without difficulty on account of the special symptoms arising in each case.

Pain without Swelling or other Localizing Symptoms.

The causes of umbilical pain are so numerous and varied, that in order to mention all it would be necessary to enumerate nearly all abdominal complaints. This cannot be done here, but one should bear in mind the following as possible causes in patients in whom no trace of abdominal disease can be found, and who still complain of pain at or about the umbilicus.

Tabes Dorsalis.—The only complaint of the patient may be of abdominal pain, often referred to the region of the umbilicus. The typical gastric crises may be replaced by a much more diffuse pain, and more than one person has been operated on, and a gastro-enterostomy performed, under a mistaken diagnosis. A systematic examination of the knee-jerks, pupil-reflexes, etc., should be made in all cases.

Lead Poisoning.—Severe attacks of cramp-like abdominal pains referred to the umbilicus may be the chief, or even the only, symptom of plumbism. The patient's occupation may suggest the diagnosis, or other characteristic signs of lead poisoning may be found (p. 34.)

Tumour of the Spinal Column or Cord, Spinal Caries, and *Compression Myelitis.*—Though a less common source of error, these must be borne in mind. Owing to its situation, a growth in the spine may be very hard to locate ; a skiagram may be of service.

Phthisis.—In this disease gastric pains are a common symptom, and the pains may even be referred to the umbilicus.

George E. Gask.

PAINFUL COITUS.—(See DYSPAREUNIA, p. 193.)

PAINFUL MICTURITION.—(See MICTURITION, ABNORMALITIES OF, p. 393.)

PAINFUL SWALLOWING.—(See DYSPHAGIA, p. 194.)

PALLOR.—(See ANÆMIA, p. 21.)

PALPITATION signifies the sensation experienced by a person who is conscious of his heart beats. It is not necessarily associated with pain. It may be due to many different causes, of which the following are the chief :—

1. **Valvular Heart Disease, especially** :—

Mitral stenosis
Mitral regurgitation
Mitral stenosis and regurgitation
Aortic stenosis
Aortic regurgitation
Aortic stenosis and regurgitation
Pulmonary stenosis.

2. **Heart Affections associated with very High Blood-pressure** :—

Arteriosclerosis.
Granular kidney.

3. **Myocardial Affections** :—

Fatty heart
Fibroid heart
Primary alcoholic heart
'Blacksmith' heart
Cloudy swelling
Pericarditis
Adherent pericardium
Senile changes with atheroma
Congenital deformity.

4. **Lung Affections leading to Failure of the Right Side of the Heart** :—

Chronic bronchitis
Emphysema
Fibroid lung
Large pleural or pleuritic effusions

5. **The Effect upon the Heart of certain Drugs, etc.** :—

Tobacco
Tea
Coffee
Alcohol
Absinthe
Morphia
Cocaine
Digitalis
Thyroid extract

6. **Heredity, Nervousness, and Allied Causes** :—

Fright or other emotion
Graves's disease
Paroxysmal tachycardia
Neurasthenia
Hysteria
Epilepsy
Tabes dorsalis.

7. **Anæmia** from whatever cause, but particularly

Chlorosis
Pernicious anæmia.

8. **Mechanical Interference with the Heart by** :—

Mediastinal new growth
Chronic mediastinal fibrosis
Thoracic aneurysm of large size
Tympanites
Ascites
Pregnancy
Ovarian cyst or other large intra-abdominal tumour.

9. **Dyspepsia** : especially that which is associated with flatulence and with stoutness from lack of exercise.

The majority of patients who complain of palpitation jump to the conclusion that they have something the matter with the heart, and, although the above list is a long one, the diagnosis resolves itself in all but a few cases into deciding whether the palpitations are cardiac or gastric in origin. A routine examination of the various systems will very often indicate the correct diagnosis at once.

Valvular heart disease will be indicated by the history of rheumatic fever, scarlet fever, chorea, syphilis, or the like, and by the alteration in the size of the heart, together with the various bruits. Mitral stenosis is sometimes more difficult than the others to diagnose on account of the possible absence of a bruit or of enlargement of the left ventricle, but it may be suggested by the patient's malar flush, by the history of acute rheumatism, and by the loud, sharp, slapping character of the first sound at the impulse. Aortic regurgitation is sometimes present without a bruit, but it can generally be detected in these cases by the typical collapsing character of the pulse ; if there is still doubt, and the patient is able to take exercise, it is frequently possible to bring out an aortic regurgitant bruit by asking him to take a few steps briskly.

High blood-pressure conditions are best detected by means of a sphygmomanometer ; the diagnosis will be confirmed by the big heart, the albuminuria with tube-casts, and perhaps by retinitis.

Of the *myocardial affections*, pericarditis and cloudy swelling are both acute conditions, often associated with fever and with sufficient general illness to confine the patient to bed, so that the palpitations are a minor part of the malady. The diagnoses of fatty or fibroid heart and of adherent pericardium are discussed on pp. 53, 54. Palpitations due to heart-muscle affection are sometimes most difficult to distinguish from similar palpitations due to dyspepsia. This applies particularly to fatty changes in the heart. Not a few middle-aged persons suffer from palpitations which, by some observers, will be attributed to gastric trouble, whilst by others both the palpitations and the dyspepsia will be attributed to fatty heart; nor can the diagnosis be made by watching the effect of slight exercises upon the pulse-beat, for in typical dyspeptic persons without fatty heart, the general condition is usually sufficiently lacking in tone for the pulse-rate to be increased readily by exercise. If material benefit results from the giving of digitalis, from the adoption of Schott's Nauheim treatment, or some modification of the latter, the argument will be in favour of some myocardial degeneration ; but in many instances of flatulence and palpitation the diagnosis between fatty heart and mere dyspepsia will remain largely a matter of opinion. Electrocardiograms may throw light upon the nature of some cases of myocardial disease the nature of which is not obvious to percussion and stethoscopic examination only ; auricular fibrillation may be found, for example (*Fig.* 196), though there may be no valvular disease.

Lung affections causing strain of the right ventricle, and thus leading to palpitations, are detected as a rule by physical examination, but here again there may be so much difficulty in interpreting the physical signs that when a stout, middle-aged person, with obvious emphysema, and with wheezing and shortness of breath on exertion, complains of dyspeptic symptoms and also of palpitations, it may be very difficult indeed, except by watching the effect of different lines of treatment, to say whether the actual cause of the palpitations is emphysema with secondary dilatation of the heart, or dyspepsia with reflex palpitations or the result of fatty changes in the heart muscle of a dyspeptic person of sedentary occupation who is both stout and emphysematous.

Tobacco is a very important cause for palpitations in a patient who may seem to be perfectly healthy : the degree to which different individuals can smoke tobacco with impunity varies enormously, and whereas some may smoke from morning to night and develop no untoward symptoms at all, others develop some ill effects from what are relatively quite small quantities. Cigarettes seem to be the greatest offenders in this respect, particularly cheap cigarettes sold in packets of considerable numbers for a penny. In bad cases the heart becomes absolutely irregular ; in all cases of the kind any extra exertion, such as trotting a hundred yards, causes a rise in the pulse-rate out of all proportion to what it should ; the pulse may rise, for instance, from 70 to 150 or 160 per minute as the result of slight exertion which in an ordinary individual would only increase it to 90 or 100. These patients may have palpitations at any time of the day or night, but particularly when they first get into bed, when violent thumpings may cause them considerable alarm. Similar thumpings of the heart, closely allied to but hardly identical with palpitations, are complained of by elderly men, particularly those of the gouty habit, probably with atheromatous degeneration of their coronary arteries. Another condition in which the heart may produce similar symptoms is *epilepsy*, in which disease, quite apart from the major attacks, there are innumerable accessory symptoms of which cardiac thumpings in bed at night are one. In that particular variety of epileptic convulsions which is associated with a sudden halving of the pulse-rate and coma—sometimes spoken of as *Stokes-Adams syndrome*—palpitations may also be a prominent symptom ; the diagnosis is established best by obtaining electrocardiographic records of partial or complete heart-block (*Fig*. 37, p. 83). *Tea*, *coffee*, *alcohol*, and *absinthe*, as causes of cardiac irregularity and consequent palpitations, can be diagnosed best by the history and by the effects of stopping the drugs in question. *Morphia* and *cocaine*, if taken over periods sufficiently long to lead to palpitations, will generally be indicated by the presence of multiple prick-marks upon the body or limbs. The palpitations and other cardiac symptoms are worse, not while the drug is being taken, but when it is being intermitted. *Digitalis* and *thyroid extract* will be recognized as the cause at once if they are being prescribed by the medical attendant.

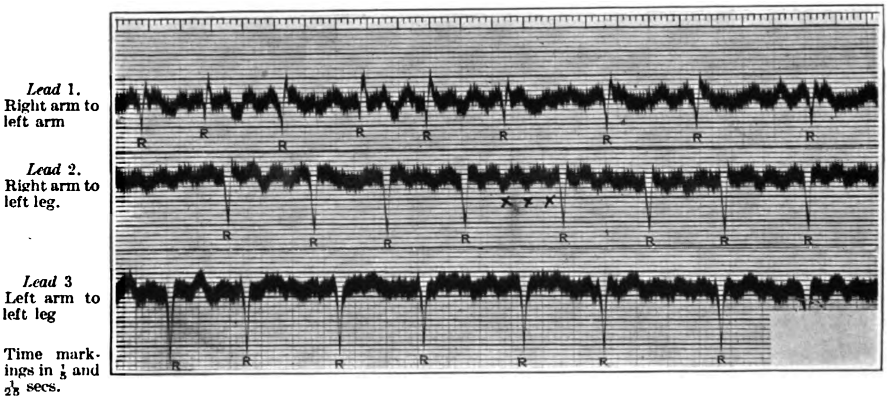


Fig. 196.—Electrocardiogram to illustrate auricular fibrillation. Ventricular beats are marked R. The auricular beats are unmarked except in the central part of the middle curve, where three of them are marked x x x. The ventricular beats (R) do not recur at equal intervals, so that the pulse was irregular in time ; but the average ventricular rate was 100 per minute. The auricles were not beating regularly ; but where marked x x x the beats occurred approximately one every fifth of a second, the auricular rate at this point being about 300 per minute.

When palpitations are due to *nervousness, fright*, or other *emotion*, they are transient and not difficult to diagnose ; if they keep on recurring from apparently trivial causes in a person who has not hitherto been nervous, it is important to bear in mind the possibility of *Graves's disease*, for although exophthalmos and enlargement of the thyroid gland are important symptoms of this disease, it is not at all uncommon for them to be absent, especially in the early stages, and for the only sign of the malady to be undue nervousness of the patient with a tendency to tachycardia and palpitations. Any condition in which the nervous system seems to be lacking in force or in control is liable to be termed *neurasthenia* nowadays, and if neurasthenia be defined in this broad sense, then one variety of it has palpitations for a chief symptom : the making of a diagnosis of neurasthenia in such cases, however, is equivalent to saying that the palpitations are of purely nervous origin, so that all one has really done is to exclude organic changes. The same applies to *hysteria*, though it should be borne in mind that modern authorities distinguish between *hysteria* and *neurosis*, confining the word hysteria to those cases in which the symptoms are directly controllable by suggestion. *Locomotor ataxia* may give rise to symptoms referable to almost any of the viscera, and there seems no reason why cardiac crises should not occur as much as laryngeal, gastric, or intestinal; they are, however, rare, and when cardiac symptoms develop in a patient suffering from locomotor ataxia, syphilitic affection of the heart would be a safer diagnosis than would cardiac crises.

Anæmia, when it produces palpitations, is usually obvious from the patient's appearance, and it can be confirmed by blood examination. As a rule, palpitations in anæmic patients are entirely absent while the patient is at rest in bed, occurring mainly when she exerts herself and causes temporary dilatation of the anæmic heart. The palpitations disappear when the anæmia is cured.

Palpitations due to *mechanical interference with the heart*, by masses of fluid or wind displacing it, can generally be relegated to their correct cause by physical examination of the chest and abdomen; mediastinal new growth, tympanites, ovarian cyst of large size, and the other conditions referred to above, generally make their presence obvious before they are of sufficient size to produce palpitations. *Herbert French.*

PAPULES may be defined as solid, circumscribed elevations of the skin, not larger than a pea. Similar formations exceeding that size are classed as nodules or as tumours. From VESICLES (p. 753) they are distinguished by their solidity; if a papule is punctured, nothing but blood exudes; but in many instances papules, especially those of an inflammatory kind, are transitional lesions, passing into (*a*) vesicles, (*b*) pustules, (*c*) scales, or (*d*) breaking down into ulcers, (*e*) undergoing hypertrophy, as warts, or (*f*) atrophying. If the transformation into pustules or vesicles is only partial, the lesions are described as papulo-pustules or papulo-vesicles, and if this is characteristic of the greater number of the lesions, the eruption is said to be papulo-vesicular, vesiculo-papular, or papulo-pustular. If the lesions, originating as erythematous macules, do not take on the full character of papules, they are said to be maculo-papular or erythemato-papular.

In *size*, papules may vary from a pin's head, as in lichen scrofulosorum, to a pea, as in lichen ruber planus. The most typical *shape* is that of the papule of lichen planus, flattened and with an irregular base; but they may be rounded or oval, as in prurigo, or cone-shaped, as in pityriasis rubra pilaris, or triangular, or umbilicated, as in lichen planus. In *colour*, they may be pink or rose-coloured, as in the inflammatory papules of urticaria papulosa, violet or purplish as in lichen planus, bright red as in eczema, dark or coppery as in syphilis, yellow as in xanthoma, whitish as in milium, almost black, as in infiltration sometimes met with in sarcoma, or simply skin-coloured, as in prurigo or verruca plana. They may be discrete, as in prurigo, or may occur in patches, as in lichen scrofulosorum; sometimes they form round a hair follicle, as in eczema folliculorum and pityriasis rubra pilaris; they are also met with in connection with the sebaceous glands, the sweat-glands, or the papillæ. They may be inflammatory, as in eczema, or non-inflammatory, as in severe goose-skin, or when they are the result of retained secretion, as in acne, or of excessive cornification round the mouths of hair-follicles. Usually, inflammatory papules give rise to itching; with the non-inflammatory kind there are seldom marked subjective symptoms.

Papules may occur in the epidermis, as in verruca plana, or in the derma, when they may be œdematous, as in urticaria, or infiltrated, as in lichen scrofulosorum; or they may affect both structures, as in lichen planus and the strophulus papule of acute prurigo. The epidermic papule may be recognized by its solidity, its hardness and dryness, and its superficial elevation; the œdematous dermic papule by its pinkish colour and its momentary yielding to pressure; the infiltrated dermic papule by its redness, induration, and elasticity; the epidermo-dermic papule by its union of some of the characters of the other varieties.

The multiple small papules which occur in *papular eczema* are usually conical with rounded base and bright-red colour; owing to rupture by scratching they are covered with a tiny dome of blood-crust; there is usually intense itching. Eczema of this type may resemble *lichen ruber planus*, but in that affection the papules, as a rule, are flat or umbilicated, with an irregular base, dark-red or violaceous colour, and glistening surface; there is no discharge or crust-formation; the papules are not transitional, and they leave brownish stains, while those of eczema frequently pass into vesicles and seldom produce discoloration. Confusion between the plaques of lichen planus and those of scaly eczema may be obviated by attention to the differences in colour and in definition ; in the former the plaques are generally dark-red or purplish, and sharply defined ; in the latter, bright-red and not clearly marked off from the surrounding skin. Under the lens, a minute vesicle can often be seen on the top of each papule in eczema papulatum, which is not the case in lichen planus. Yet another point of distinction between the two is that in lichen planus characteristic discrete papules can usually be found at the margin of the patches. This feature serves, too, to differentiate lichen planus, in its turn, from *generalized psoriasis*, which is marked also by more scaliness and less thickening.

Lichen scrofulosorum is a papular dermatosis which clinically bears some resemblance to lichen planus, but it has no right to the designation lichen, and belongs rather to the tuberculides. The papules are seldom larger than a pin's head, are usually flattish but occasionally conical, very slightly resistant, sometimes smooth and shiny but more often covered with a tiny scale which is but slightly adherent ; occasionally the summit is occupied by a pustule instead of by a scale. The prevailing colour is red, but it varies from a pale-yellow through red to violet. At first the lesions are disposed in groups, forming patches of various sizes. Others are arranged in arcs of circles, which are usually seen about the orifices of the sebaceous glands. The eruption shows a distinct predilection for the trunk—the lower part, and the back of the abdomen ; but occasionally it extends to the beginnings of the limbs and may invade the face. If the affection becomes generalized by the spread and coalescence of the scattered groups of papules, the whole skin is covered with thin scales and is of a dirty reddish-brown hue. Itching is absent, or so slight as to be negligible. The eruption begins insidiously and may last for several months ; and, having disappeared, leaving behind it no trace, it may reappear again and again over a period of several years. In the great majority of cases the disease is associated with some form of tuberculosis—phthisis, or necrosis of bone, or scrofulous ulceration of the skin ; but much more commonly with enlarged glands, submaxillary, cervical, or axillary. Its usual subjects are children and adolescents ; it is uncommon after the age of twenty. The characters of the papules—their homogeneity, situation on the trunk, flattish shape, arrangement in groups, painlessness, and chronicity—with the absence of itching, and the youth of the patient, are generally sufficient to determine the nature of the affection. In papular eczema the papules are not limited to the trunk, are bright red, and there is troublesome itching ; moreover, in many cases there are vesicular or papulo-vesicular lesions as well as papules. From *miliary papular syphilides* lichen scrofulosorum can be distinguished by the absence of any other sign of syphilitic affection, and by the usually restricted distribution of the lesions.

In *keratosis pilaris*, or *xerodermia*, the papules do not form groups or patches as in lichen scrofulosorum, and usually they appear on the limbs, most frequently the thighs ; they consist of projecting hair-follicles, which convey to the hand the sensation of a nutmeg-grater. This affection has, in turn, to be distinguished from *goose-flesh*, in which the elevations, besides being evanescent, are not rough or scaly. Keratosis pilaris is one form of *ichthyosis* ; another form, sometimes met with in association with xerodermia, but more often alone, is *ichthyosis hystrix*, in which the lesions consist of small papillary papules with horny tops, which stud the skin as with minute nail-heads, and sometimes develop into large warty masses. The condition, dating back to infancy, or at least to early childhood, is easy of identification.

The same may be said of the ordinary wart (*verruca vulgaris*). The small flat wart (*verruca plana juvenilis*) may sometimes suggest lichen planus, but it is smaller than the papule of that affection, it has not the dark colour, it gives rise to no itching, nor is there any tendency for the growths to run together into rough, scaly, infiltrated patches.

In *pityriasis rubra pilaris*, papules form at the orifices of the hair-follicles, usually following in the wake of an eruption of scaly patches, or of a dry eruption covered with eczematous-looking crusts. The papules, when they appear, are small, red, and dry, harsh to the touch, more or less conical, and the centre of each is pierced by a single atrophied hair, which is surrounded by a sheath that penetrates into the follicle. The surface of the integument, thus roughened, has been likened to the skin of a newly-plucked fowl. At first the papules are discrete, but later they tend to run together into patches which present the aspect of pale yellowish-red areas covered with papery scales resembling mica.

They mostly affect the limbs, especially the surfaces where hair is most abundant; if they encroach upon the trunk they are usually found at the waist and the lower part of the abdomen. Itching is sometimes absent, and when present is always insignificant. When the patches are covered with mica-like scales and are met with in the situations most common to psoriasis—the tips of the elbows, fronts of the knees, and the extensor surfaces of the limbs—there is some danger of confusion with that disease; but at the edge of each patch the characteristic conical papule with its single hair plugging the mouth of a follicle is always to be seen. The best place to look for the papule is on the backs of the fingers. In psoriasis, again, the lesions grow by peripheral extension instead of by the accretion of new papules. The fact that the general health is not affected suffices to mark off pityriasis rubra pilaris from other forms of *exfoliative dermatitis*, in which also there is rarely any appreciable thickening of the skin, while the colour is redder than in pityriasis and the scaliness more marked. From lichen ruber planus, pityriasis rubra pilaris is distinguished by the absence of itching and of impaired nutrition, and by its irresponsiveness to arsenic. In lichen planus, too, the papules are flattened, and often umbilicated.

In *acne vulgaris*, the papule forms the primary lesion. If the obstruction is at the mouth of the sebaceous gland-duct, the plug appears on the surface as a small black point —the comedo; if it is in the gland itself, the obstructing material is seen as a tiny whitish mass in the substance of the skin—the milium. The lesion may not develop beyond this stage, but usually it grows into a reddish papule about the size of a pea, and in many cases the papule passes into a pustule. Acne can usually be recognized by the distribution of the lesions—on the face, especially the cheeks, nose, forehead and chin, and less frequently the back of the neck, the back between the shoulders, and the chest—their discrete character, the presence of comedones or of milia, and the patient's age, for the affection is essentially one of puberty. Usually, too, the several stages through which the lesions pass are present at the same time—the comedo or milium, the papule, the pustule. *Rosacea* differs from acne in that it chiefly affects the flush area of the face, is marked by much congestion, and is most common in middle life. (See also PUSTULES, p. 557.)

In *erythema multiforme*, papules, tubercles, vesicles, bullæ, nodules, macules may all be present at the same time. Any one of these lesions may be predominant in a particular case or at a given time; but the type of eruption most frequently met with is that which consists wholly or predominantly of papules—*erythema papulatum*. This, too, is usually the first stage of the eruption in cases which go on to other types. The papules are generally, at the beginning, no larger than a pin's head, bright-red in colour, and flattish, sometimes umbilicated. If grouped closely together they may coalesce and form raised patches as large as a threepenny or a sixpenny piece; each patch has a sharply defined border, and is surrounded by an areola of congested skin; the tint in the centre soon deepens to violet, and afterwards to purple. The favourite sites are the dorsal surfaces of the hands and forearms; sometimes, too, the legs, feet, and the face are involved. Papular erythema multiforme is, as a rule, easy of recognition. Urticaria of the papular variety—the strophulus or *prurigo simplex aigu* of French dermatologists—in which the wheals are no larger than a lentil, and leave papules when they subside, occasionally offers some resemblance to it; but the two affections differ in that the lesions of erythema papulatum are much more persistent, are not white in the centre, do not give rise to itching, but do leave stains. One of the most marked of these differences is that which concerns itching, for in papular urticaria this symptom, though variable, is frequently severe. In urticaria, too, it is chiefly the covered portions of the body, and especially the lower lumbar region, that are attacked, and the affection is met with mostly in children.

Papules like those of urticaria papulosa are often met with in prurigo, both in *prurigo ferox* of Hebra, the severe form of the affection, and in *prurigo mitis*, the mild and ordinary form. The characteristic lesion is an eruption of discrete, firm, very slightly raised papules, more or less hemispherical in shape, with a roundish, sometimes oval contour, and a glistening surface. At first the papule is of the same colour as the skin, but afterwards it becomes red, yellowish, or brownish, increases in size, and is frequently covered by a blood-crust. In structure, according to Darier, it is a localized acanthosis. Most abundant on the extensor surfaces of the limbs, and rarely seen on the flexor aspects, or on the face, the papules occur not infrequently on the chest, the lower part of the abdomen, the sacral region, and the buttocks. The itching is intense, and mixed with the secondary changes produced by scratching, there may be found others not unlike those of eczema. Pustules and sores are common, often accompanied by great enlargement of the axillary and femoral glands. In prurigo ferox the papules are much larger and more numerous, and in parts the skin feels to the touch like very coarse brown paper or a nutmeg-grater. The changes secondary to the inflammatory process in prurigo are summed up by French dermatologists in the term *lichénisation* or *lichénification*. The skin is thickened and rugose, and owes its peculiar aspect to an exaggeration of the fine striæ of the normal integument, so that it becomes quadrillated into a network of which the meshes are square, lozenge-shaped, or polygonal, with a flat surface which often, as Darier says, presents the aspect of glossy and brilliant facets, as of a mosaic. Sometimes it is covered with fine scales. The skin is less supple than normal skin, and though in colour it may undergo no change, it is more often greyish or brownish. The condition has to be distinguished from the lichenization met with in other dermatoses. In eczema and psoriasis, the thickened and rugose skin is red, has no glistening facets, and the margins are clearly defined. In lichen planus the plaques are formed by confluent papules and are surrounded by characteristic papules.

Apart from these secondary characters of prurigo, the diagnosis is made by excluding other itching affections, such as *scabies* and *pediculosis*, on account of the absence of the lesions characteristic of those conditions, and by the positive characters—the origin of the affection in infancy, and its persistence, the poor general health, the preference the papular eruption shows for the extensor surfaces of the limbs, the freedom of the bends of the joints, and the glandular enlargement, especially in the inguinal region.

In *syphilis*, papules are met with frequently in association with macules, but they may occur independently. They may be divided into two main classes: (1) *Miliary papular*, and (2) *Flat papular syphiloderms*.

1. *Miliary*, or *follicular*, *papular syphilides* are the result of infiltration around and beneath the pilo-sebaceous follicles; they are rough to the touch, and feel like small shot; they vary in size, from a pin-head upward, and may be either acuminate or rounded. The sites of election are the trunk, back, shoulders, and loins, but the limbs may be invaded, and also the face. Very frequently the lesions appear in groups which run into each other; and they are sometimes disposed in rings. The characteristic colour is that of raw ham, but at first they may be pink or red. Involution proceeds slowly, the stain left behind is long in dying away, and is sometimes succeeded by a shallow depression which may last for years. There is often a slight scale on the surface of the papule, and not seldom a tiny vesico-pustule or pustule may be detected on the summit. Sometimes there is overgrowth of the papillæ; and if the lesion is situated in a moist part the warty growth is covered with sodden white epithelium, when the lesion is known as a *mucous papule*. A more marked degree of hypertrophy transforms the moist papule into a *mucous tubercle* or *condyloma*, distinguishable from a wart in that the overgrown papillæ are welded into a coherent mass by swelling of the intervening tissue, while in the wart they are free.

2. *Flat*, or *lenticular*, *papular syphilides* vary in size from a pin-head to a bean, and the small or the large lesions may predominate in a given case. They may develop directly out of macular syphilides. In contour they are almost perfectly round, with a flattened top, are but slightly elevated, and in colour usually brownish-red. The whole body is affected pretty impartially; there is little tendency to agmination; sometimes the lesions form a kind of circlet on the brow round the margin of the hair (the *corona veneris*). In some cases, ring-like patches (the circinate or annular syphiloderm) appear on the chin, around the lips or nostrils, or sometimes on the vulva; they are made up of small yellowish-red papules, with fine scales; elsewhere the papules, in the same case, are of the ordinary kind. In other instances, the flat lenticular papules of this group become seborrhœic; these are characterized by their obduracy to treatment. When there is marked scale-formation, the papules being covered with a dry, dirty-grey scale, they are styled papulo-squamous. Favourite situations for papulo-squamous syphiloderms are the palms and the soles, where they may be mixed with maculo-papular and papulo-tubercular lesions. Syphilides in these sites are often rounded or irregular in shape, have but slight elevation, are at first brownish-yellow or brownish-red, but presently become of a dirty grey, and on the disappearance of the scales have the characteristic colour of raw ham. When the scales are more abundant than usual, they form the *syphilides cornées* of French dermatologists.

The small papular syphilides may in some cases be difficult to distinguish from a widely diffused lichen ruber planus ; but in this affection the rash is uniform, the papules generally have a linear arrangement, and there is usually severe itching. The papules of syphilis are most likely to be confused with psoriasis—the squamous papules with the ordinary form of psoriasis, the papules in rings with annular psoriasis. Attention must be paid to the polymorphism of the syphilitic eruption, the coppery colour, the enlarged glands, the sore throat or tongue, and the distribution of the lesions, no such partiality for the elbows and knees being shown as is observed in psoriasis, and the papular syphilide having a preference for the flexor surfaces of the limbs, while psoriasis affects rather the extensor aspects. The syphilitic scales, too, are thin and dirty-white, while those of psoriasis are heaped up in layers and have a silvery sheen. In psoriasis, the subject will usually have a history of previous attacks to relate ; and often the affection can be traced back to early life, whereas in syphilis a particular lesion is seldom repeated. The palmar and plantar syphiloderms described above, which occur symmetrically as secondary and unilaterally as tertiary lesions, may be confused with the dry chronic eczema of those regions ; but in eczema there are heat and itching, and usually, in the case of the palms, the fingers also are involved, and at some point or other the process is vesicular or moist. Palmar syphilides may be distinguished from eczema seborrhœicum by the fact that in the latter there are coincident lesions in the common situations—the scalp and eyebrows, the neighbourhood of the beard, the naso-labial folds, the sternal and interscapular regions ; nor do they usually assume the form of crescents or segments. Whenever the diagnosis is doubtful, recourse may be had to Wassermann's serum test. *Malcolm Morris.*

PARÆSTHESIA.—(See Sensation, Abnormalities of, p. 604.)

PARALYSIS OF BOTH LEGS.—(See Paraplegia, p. 510.)

PARALYSIS, CROSSED.—(See Hemiplegia, p. 302.)

PARALYSIS, FACIAL.—This term is applied to complete or partial paralysis of the muscles supplied by the 7th cranial nerve. One or both sides of the face may be involved, the unilateral being more common than the bilateral. It is in some cases the result of a morbid process limited to the 7th nerve, known as *Bell's palsy* ; in other instances it is one of the signs of more complex, or more remote, disease. In complete unilateral facial palsy, whatever its origin, the asymmetry of the face may be so marked that the diagnosis can be made at sight. Less severe facial weakness may be overlooked unless the means for its detection are employed. It is well, therefore, to recall the evidences of facial palsy before pointing out the features which characterize its various forms.

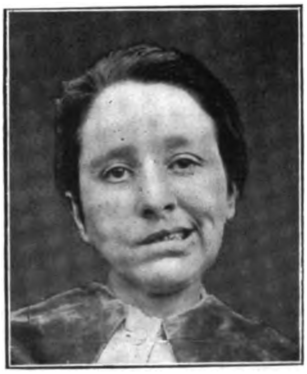


Fig. 197.—Paralysis of the right side of the face, the patient attempting to show her teeth. Note that the right palpebral fissure is wider than the left. (*Photo by Dr. S. A. K. Wilson.*)

Even with the face at rest there are certain appearances on the affected side which attract attention. The natural lines and wrinkles are less marked, and, with the obliteration of the naso-labial fold, the cheek has a somewhat flattened or, in old persons, baggy aspect. If the patient is unconscious or asleep the flabbiness of the tissues may be emphasized by the flapping of the cheek with respiration, especially if breathing is laboured or stertorous. The palpebral fissure is wider than its fellow, and the corner of the mouth may droop.

When the facial muscles are thrown into action by attempts to raise the eyebrows, to close the eyelids, or to expose the teeth (*Fig.* 197), the difference between the two sides is rendered more obvious, the movements on the paretic half of the face being carried out with less power and more slowly than those of the healthy half. The ability to whistle or to move one nostril may also be impaired, and even with slight degrees of paresis, a person who has previously been able to close the eye of the affected side, the other eye remaining open, is no longer able to perform the feat. The same difficulty is experienced in making movements of the ear, by patients who have formerly possessed that accomplishment.

Having established the presence of some facial weakness, it is necessary, in order to take full advantage of its diagnostic value, to make certain careful observations with a view to determining the site of the lesion which is responsible for the defect. Thus, facial paralysis may be brought about by : (1) A lesion anywhere in the course of the pyramidal fibres passing from the lower end of the precentral gyrus in one cerebral hemisphere to the facial nucleus on the opposite side of the pons Varolii (*supranuclear paralysis*) ; (2) A lesion involving the facial nucleus itself (*nuclear paralysis*) ; and (3) A lesion of the 7th nerve between its origin in the nucleus and the point where it divides in order to supply the various facial muscles (*peripheral paralysis*).

Supranuclear Paralysis.—Owing to the fact that the fibres of the pyramidal tract concerned with facial movements cross the mid-line of the brain-stem only a very short distance above the 7th nucleus, the facial paralysis is on the side opposite to the lesion. Occasionally these fibres are involved alone ; more often those destined to the corresponding arm and leg suffer as well, in which case the facial palsy forms part of a hemiplegia. In this type of paralysis the difference between the two sides is not nearly so marked in the upper as in the lower half of the face. For instance, the patient is able to elevate both eyebrows and to close both eyes, although it will be found, on testing, that he is not able to resist an attempt on the part of the observer to open the eye of the affected side with the same degree of success as attends his efforts on the healthy side. (*Figs.* 198, 199, 200.) In the attempt to expose the teeth, the facial asymmetry is more striking, the lip remaining immobile or retracting slowly and feebly on the paretic side, and the contrast between the depth of the naso-labial folds on the two sides becoming clearly emphasized.

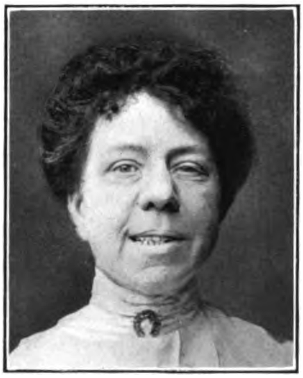


Fig. 198.—Post-paralytic contracture of the left side of the face. At a first glance the appearance suggests that the right side is the seat of paralysis.

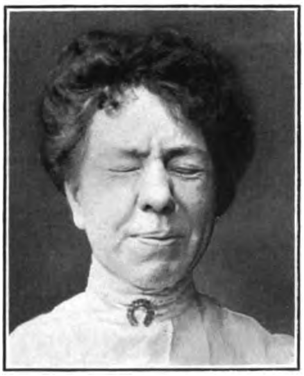


Fig. 199.—Post-paralytic contracture. The same patient closing her eyes and showing the over-action of all the facial muscles on the affected left side.

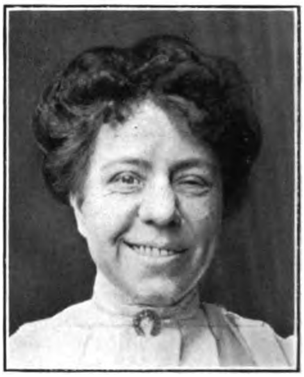


Fig. 200.—Post-paralytic contracture. The same patient smiling, with an involuntary wink.

(*Photos by Dr. S. A. K. Wilson.*)

Notwithstanding the impairment of voluntary movement on one side, the face may present perfect symmetry when it responds automatically to emotional or reflex impulses. In laughing or crying the lines and wrinkles are developed equally, while protective closure of the eyelids is accomplished as well on one side as the other in response to any threatened violence to the eyes. The preservation of these automatic movements depends on the integrity of a facial reflex centre in the mid-brain. When this centre is involved at the same time as the fibres of the pyramidal system, the emotional movements are lost or impaired along with those of voluntary origin.

The corneal reflex can generally be elicited in this form of facial paralysis, provided there is no co-existent diminution of sensibility within the area supplied by the trigeminal nerve of the same side. In sharp contradistinction to what obtains in the nuclear or peripheral types of palsy, the nutrition and electrical excitability of the facial muscles undergo no alteration when the lesion is situated above the nucleus.

Bilateral supranuclear paralysis, such as is seen in cases of cerebral diplegia and pseudo-bulbar paralysis, is characterized by a general impairment of the natural movements, and tends to the production of a stiff, expressionless physiognomy (*Fig.* 201). With the consequent defective inhibition of the reflex centre, emotional movements are often uncontrolled, and with little provocation the patient betrays in his face degrees of mirth or distress which he is very far from feeling. This condition can be distinguished from true bulbar palsy by the preservation of the nutrition and the normal electrical excitability of the muscles, and by the absence of accompanying atrophic paralysis of the tongue, masseters, etc.

Reference must be made to the occurrence of cases, the result of mid-brain lesions, in which the emotional movements are lost and the voluntary movements are preserved on one side of the face. Asymmetry is then only noticeable when the patient smiles or cries.

Finally, it must not be forgotten that in long-standing cases of infantile hemiplegia, facial weakness may be associated with spontaneous athetoid movements similar to those observed in the arm and leg.

Nuclear Paralysis.—Lesions of the facial nucleus may be slight or severe, and the resulting facial paralysis may consequently be partial or complete. As a rule, all the muscles supplied by the nerve are affected more or less equally, and the impairment of movements obtains whether they are voluntary, emotional, or reflex in origin. In cases of complete nuclear palsy certain additional effects are produced. The inability to close the eye, and the drooping of the lower lid, lead to imperfect protection of the eyeball, and to the overflow of tears on to the cheek (epiphora). Conjunctivitis and blepharitis may result. Paralysis of the lip muscles allows of saliva escaping from the corner of the mouth, and may interfere materially with the articulation of labial consonants. Paralysis of the stapedius muscle disorganizes the control of tympanic tension, so that the patient suffers from excessive sensitiveness to deep tones (hyperacusis) and perhaps from tinnitus. Unlike supranuclear paralysis, the nuclear form is accompanied by atrophy and alteration in the electric excitability of the facial muscles. In slow degenerative (usually bilateral) processes affecting the facial nucleus, the electrical response shows a quantitative modification ; in acute destructive (usually unilateral) lesions of the nucleus, the facial paralysis is followed rapidly by the reaction of degeneration.

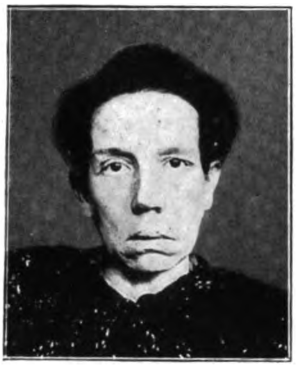


Fig. 201.—Bilateral facial palsy. The photograph shows absence of all lines, and sagging of both corners of the mouth.

Owing to the situation of the facial nucleus in the pons, unilateral nuclear palsy may be associated with paralysis of the external rectus muscle of the same side and paralysis of the opposite arm and leg (‘ crossed ’ paralysis).

Peripheral Paralysis.—The clinical picture of a peripheral facial paralysis resembles in its most important details that which has already been described under the heading of nuclear paralysis. All the muscles supplied by the nerve are affected in more or less equal degree, and the palsy is associated, within a short time of its onset, with atrophy and alterations in electrical excitability of the muscles concerned. The diagnosis between the two types depends chiefly upon the presence of additional symptoms resulting from interference with the function of neighbouring tissues, and this varies again with the exact site of the lesion in the peripheral course of the nerve.

A lesion affecting the fibres within the pons is likely to produce, in addition to the facial paralysis, external rectus palsy, together with other symptoms in proportion to the extent of the destructive process.

A lesion between the surface of the pons and the internal auditory meatus will probably interfere with the vestibular and cochlear parts of the 8th nerve, and so give rise to vertigo and impairment of hearing.

At the level of the geniculate ganglion, the chorda tympani is unlikely to escape, and the taste fibres coming from the anterior two-thirds of the tongue lose their function. At the same time, irritation of the ganglion may provoke an herpetic eruption on the auricle and around the external auditory meatus.

In the upper part of the Fallopian canal a lesion produces complete facial paralysis and loss of taste on the anterior part of the tongue; in the lower part of the canal the resulting symptoms are the same, with the exception that paralysis of the stapedius, with its consequent hyperacusis, does not occur. The nerve to the stapedius leaves the facial nerve between these two points. Involvement of the chorda tympani may also cause deficiency in the salivary secretion of the submaxillary and sublingual glands of the same side.

At the stylomastoid foramen, the effects of a lesion are limited to the facial nerve, the taste fibres being no longer in close apposition to the latter.

From the above data the site of any lesion causing peripheral facial palsy can be determined approximately, and it is only necessary to add that the term *Bell's palsy* is generally limited to cases in which the exciting cause, probably an acute inflammatory process, operates at some point within or just below the Fallopian canal.

From the diagnostic standpoint it is important to remember that a condition which often results from a long-standing Bell's palsy may produce a facial asymmetry capable of erroneous interpretation, unless the observer is familiar with it. This is the so-called *post-paralytic contracture*, which emphasizes the folds and lines on the affected side in such a way that the opposite side of the face may appear at first sight to be the weaker (*Fig.* 198). It will be noticed, however, that an attempt to close the eye is imperfectly carried out, and that the angle of the mouth is strongly, although involuntarily, retracted at the same time. Similarly, on asking the patient to show his teeth, he can only do so slowly and with effort, while the eye is almost closed on the same side by a powerful associated contraction of the orbicularis palpebræ. The only complaint of a girl suffering from a slight degree of this contracture was to the effect that she was unable to smile without at the same time giving the impression that she was winking.

Fig. 202.—Hemiatrophy of the left side of the face in an early stage. This condition is sometimes mistaken for facial palsy.

Hysterical facial spasm is another condition which may suggest weakness of the opposite side of the face, but the nature of the defect will be made obvious when the whole face is put into action.

Facial hemiatrophy often simulates facial paralysis (*Fig.* 202); it may be differentiated by the fact that not only the muscles, but all the tissues of the face on the affected side, undergo atrophic changes.

E. Farquhar Buzzard.

PARALYSIS OF THE HAND.—(See PARALYSIS OF ONE EXTREMITY, UPPER, p. 500; CLAW-HAND, p. 109; ATROPHY, MUSCULAR, p. 59; and HEMIPLEGIA, p. 302.)

PARALYSIS, LARYNGEAL.—Laryngeal paralysis is to be distinguished from interference with the vocal cords by inflammatory or ulcerative lesions, fixation of the arytenoid joints, and other affections which mechanically prevent free movements of the cords. The distinction can scarcely be made without careful examination of the parts with the laryngoscope.

In some cases, especially when bilateral abductor paralysis comes on suddenly, the symptoms may be urgent and extreme; in others, there may be no definite symptoms at all, particularly if there is but partial paralysis of one vocal cord, the other being freely movable and able to cross the middle line so as to meet its fellow for purposes of speech or coughing. In most cases the symptoms which point to paresis or paralysis of a vocal cord are a change in the character of the patient's voice, as noticed by himself and his friends, and difficulty in coughing effectually when need arises, the act of coughing being sometimes associated with a peculiar sound described by the terms 'brassy cough' or 'bovine cough.'

For purposes of differential diagnosis laryngeal paralysis may be divided into three main groups, namely: (1) *Functional*; (2) *Unilateral organic*; (3) *Bilateral organic*.

1. **Functional Paralysis of the Vocal Cords** has for its main symptom aphonia

without pain or discomfort, the patient nearly always being a young woman, or a girl over the age of puberty, who complains that she has almost suddenly become quite unable to speak otherwise than in a whisper. There may have been other functional nerve symptoms, the commonest being perhaps difficulty in swallowing owing to globus hystericus. This form of loss of speech is due to functional adductor paralysis during vocalization; but when the patient is asked to cough she does so with ease, and thus demonstrates that the adductor paralysis is not real, for one cannot cough properly without adducting the vocal cords. If the larynx is examined with the laryngoscope, the cords will be seen to move perfectly both with respiration and when the patient retches, though they may remain in the abductor position if the patient is asked to make any particular voice sound. The condition always gets well; it may pass off almost instantaneously as the result of local electrical application or of treatment by suggestion.

2. **Unilateral Organic Affection of a Vocal Cord** is obvious on laryngoscopic examination. It is nearly always the result of interference with the corresponding recurrent laryngeal nerve, and owing to anatomical differences between the two, the left is affected more commonly than the right. It may be paralyzed by pressure from, or infiltration by, an *aortic aneurysm*; a *mediastinal new growth*; *secondary deposits* in the deep cervical or mediastinal lymphatic glands—for instance, in a case of squamous-celled carcinoma of the œsophagus; *lymphadenoma*; *gumma*; or *mediastinal fibrosis*, particularly, though not very commonly, in association with syphilis, or with *fibroid phthisis* affecting the upper part of the left lung. Two rare causes are *mitral stenosis*, in which the over-distended left auricle sometimes compresses and paralyzes the left recurrent laryngeal nerve, and *lobar pneumonia* of the left upper lobe; extreme stridor has been known to result during convalescence from the latter in consequence of the cord paralysis. The differential diagnosis between these various conditions will be found discussed elsewhere; *x*-ray examination of the thorax may be helpful. In the absence of special indications, paralysis of the left vocal cord in a man of about forty-five is always suggestive of an aneurysm of the distal portion of the arch of the aorta, particularly if the patient has had syphilis, has not been an abstainer, and has undergone strenuous physical exertion.

3. **Bilateral Affections of the Vocal Cords** are seldom due to thoracic aneurysm, but some of the other diseases mentioned in the preceding paragraph may extend far enough up into the root of the neck on the right side to reach and involve the right recurrent laryngeal nerve as it passes beneath the right subclavian artery, as well as the left recurrent laryngeal nerve as it turns round the arch of the aorta to the left of the left subclavian artery. Careful examination of the chest for evidence of new growth or of syphilitic or tuberculous fibrosis is necessary, therefore, before one is in a position to diagnose the more common cause for bilateral paralysis of the vocal cords, namely *degeneration of the nerve cells in the vagus centres* in the medulla oblongata. It should also be remembered that some particularly malignant *enlargements of the thyroid gland*, and also *secondary deposits in the deep cervical lymphatic glands*, or even extensive infiltration of the latter by *tuberculous* processes, may involve both recurrent laryngeal nerves as they lie on either side in the sulcus between the trachea and œsophagus, and thus cause bilateral paralysis of the vocal cords. When the paralysis is due to degeneration in the vagal nuclei there is generally abductor before combined abductor and adductor paralysis; when the affection is symmetrical from the beginning the bilateral adductor spasm may result in acute dyspnœa simulating acute suffocative œdema of the larynx and requiring immediate tracheotomy. More often, fortunately, one vocal cord passes through the stage of abductor paralysis into that of complete paralysis before the other is affected, so that the dangerous condition of simultaneous abductor paralysis of both vocal cords is avoided. The diagnosis depends upon the alteration in, or the loss of, voice, together with the inability to cough efficiently, except with the sound which simulates the coughing of a cow (bovine cough); upon observation of the bilateral paresis of the cords with the laryngoscope; upon the exclusion of gross lesions within the thorax, or in the neck; and upon the co-existence of other indications of changes in the central nervous system. These in younger people are generally the result of syphilis, often taking the form of strabismus, or of locomotor ataxy, or general paralysis of the insane; whilst in older people there may be vascular degeneration associated with evidence of cerebral softening with or without albuminuria, glycosuria,

thickened arteries, an enlarged heart, and a high blood-pressure. Only in very rare cases is the symptom due to hæmorrhage or neoplasm in the medulla oblongata, for with these the patient does not usually survive to show signs of the laryngeal paralysis.

Herbert French.

PARALYSIS, OCULAR.—(See STRABISMUS, p. 649; PUPIL, ABNORMALITIES OF THE, p. 551; and DIPLOPIA, p. 174.)

PARALYSIS OF ONE EXTREMITY (LOWER).—The diagnosis of the conditions in which paralysis of both legs occurs is dealt with under PARAPLEGIA (p. 510); the present article refers only to cases in which paralysis of one leg is complained of. It is, however, a common experience for the clinician to find signs pointing to a bilateral affection when the patient is only aware of disability affecting one lower extremity. A notable and common example of this is afforded by many cases of disseminated sclerosis. The patient complains of weakness in one leg, and the physician finds exaggeration of both knee-jerks as well as extensor plantar responses on both sides, and is led to the conclusion that both pyramidal tracts are affected, although one may be damaged more severely than the other.

The various types of crural monoplegia may be divided roughly into two classes, one of which includes those cases without muscular atrophy, and the other those which present greater or less degrees of muscular wasting.

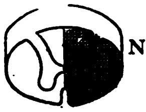
Paralysis of One Leg without Muscular Atrophy.—The cases in this class may be sub-divided into two groups, the first comprising those in which the pyramidal tract is affected, and the second those in which there is no evidence of pyramidal affection.

Spastic paralysis of one leg may result from a lesion of the pyramidal tract in any part of its course, but for anatomical reasons it is more likely that the paralysis will be confined to one side when a lesion affects the opposite cerebral hemisphere above the pons, that is to say, above the level at which the two pyramidal tracts run in close proximity. Spastic paralysis of one leg may, however, result from a lesion at any level, and the diagnosis of the level must be made from a consideration of other symptoms. In all cases the condition of the leg is qualitatively, if not quantitatively, the same. A spastic leg is characterized by a certain amount of weakness and rigidity, exaggeration of the knee- and ankle-jerks, and the extensor type of plantar response. It is useful to remember that the weakness in a spastic leg does not affect all the movements to the same extent. If the movements at the various joints are tested against the observer's resistance, it will generally be found that dorsiflexion of the ankle and flexion of the knee are affected more than other movements. It is for this reason that the patient tends to drag his toes more on the affected side than on the other, and evidence of this is often forthcoming in the fact that he tends to wear away the toes of his boot. The muscles of a spastic leg show no localized wasting, and present no alteration from the normal in their response to electrical stimulation.

In the attempt to diagnose the level of the lesion which gives rise to spastic paralysis of one leg certain considerations are of particular importance. If the lesion is situated immediately above the lumbar enlargement of the cord, the abdominal reflexes can be obtained. If the lesion is situated at the level of the 10th dorsal segment, the lower abdominal reflex on that side will be absent, while the epigastric reflex remains intact. A lesion of any of the upper dorsal segments causes abolition of all abdominal reflexes on the corresponding side. A lesion above the cervical enlargement will lead probably to some, even if slight, weakness in the corresponding upper extremity, in which the tendon-jerks will be exaggerated. A lesion of the higher part of the pons or of any level between the pons and the cerebral cortex will produce some asymmetry in the facial movements as well as weakness in the arm and leg.

Disseminated sclerosis has been mentioned already as a disease in which spastic paralysis of one leg may result from a lesion situated in the spinal cord. In all probability evidence of other patches of disease will be discovered in such cases if a careful examination is made. Some intention tremor in one or both hands, nystagmus, diplopia, optic atrophy, and sphincter troubles are among the signs which may be forthcoming. Less commonly, a one-sided affection of the spinal cord above the lumbo-sacral enlargement is due either to some intramedullary disease, such as a patch of *myelitis*, a *gumma*, or a *new growth*. When this occurs there may arise a symptom-complex to which the term *Brown-Séquard paralysis* is applied. In this condition there is spastic paralysis of the leg on the same side as the lesion, together with loss of sensibility, especially of thermal and painful sensibility, in the opposite leg. The physical signs in Brown-Séquard paralysis are represented in greater detail in the accompanying diagram :—

Zone of Hyperæsthesia



Local Effects	Atrophic Paralysis Painful and Thermal Loss Loss of all Reflexes	No Local Effects
		(4 to 6 normal segmental areas)
Remote Effects	Spastic Paralysis	No paralysis
Not Constant	Loss of sense of passive position and movement Loss of tactile discrimination	Loss of sensibility to painful and thermal stimuli Loss of tactile and pressure sensibility and localization } uncommon
	Diminished skin reflexes	Normal skin reflexes
	Increased tendon reflexes	Normal tendon reflexes
	Ankle-clonus	No clonus
	Extensor plantar reflex	Flexor plantar reflex

Fig. 203.—Diagrammatic representation of the results of a one-sided lesion of the spinal cord—Brown-Séquard Paralysis.

Hysterical paralysis of one leg usually does not afford much difficulty in diagnosis. The affected limb may be either rigid or flaccid ; in either case there is no true muscular atrophy and no alteration in the muscular response to electrical stimulation. The condition of the reflexes provides the most important information. In the hysterical form of paralysis the knee and ankle-jerks may be exaggerated, but they are never lost. A true ankle-clonus is never obtained, and the plantar reflex is either absent or of the flexor type. As a general rule the tendon reflexes in the opposite unaffected limb will be found to be equally brisk. In contradistinction to spastic paralysis resulting from a pyramidal lesion, in which it has already been pointed out that dorsiflexion of the ankle and flexion of the knee are the movements most profoundly affected, the movements of the leg in a case of hysterical paralysis are found to be more or less equally deficient at all joints and in all directions. Certain attitudes and certain types of gait are almost characteristic of hysterical paralysis of one leg. In one form the whole leg is kept rigidly extended, and the foot strongly inverted, so that the patient walks on the outer plantar edge with a stiff leg. In another form, the leg is flaccid and is dragged behind the opposite limb with the toes scraping the floor. In some cases, examination of the limb when the patient is at rest in bed reveals little or no paralysis, but in the attempt to stand or walk the limb appears to be quite useless. Hysterical paralysis of a leg may of course be associated with similar palsies of the opposite leg, or of the arm on the same side (hysterical paraplegia, hysterical hemiplegia). More often than not a leg which is the seat of hysterical paralysis also presents complete insensibility to

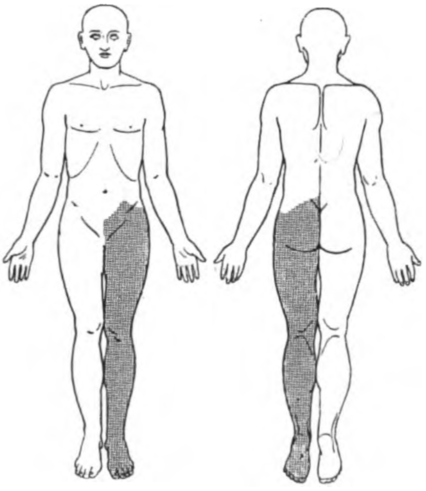


Fig. 204.—Brown-Séquard paralysis due to an intramedullary one-sided lesion of the lower thoracic cord. The shaded area was insensitive to deep and superficial pain and to all degrees of temperature, but sensitive to touch. The sense of passive movement and position and tactile discrimination were disturbed in the right foot. There was spastic paralysis of the right leg only.

all forms of stimulation, and the upper limit of such anæsthesia may correspond to the line of the knee, the groin, or the umbilicus.

In the early stages of *paralysis agitans* a patient may complain of loss of power in one leg, and the diagnosis may present considerable difficulty if the characteristic tremor of this disease has not made its appearance. Examination of the limb may show little abnormal. Some slight paresis and slight stiffness to passive movements may be detected, but no alteration in the character of the reflexes will be observed. The diagnosis must depend more upon the general aspect and the attitude and gait of the patient. Some loss of facial expression, the general slowness of his movements, and the tendency to shuffle with the affected leg, are points which may lead the observer to form a correct opinion.

Paralysis of One Leg with Muscular Atrophy.—In a case which presents atrophic palsy of one leg, the first essential for making a diagnosis is to ascertain the exact distribution of the atrophied muscles, and to review this distribution in the light of what we know with regard to the central and peripheral innervation of the muscles of the lower limb. (See table below.)

Single nerve palsies are not so common in the lower extremity as in the upper, but they may occur, especially as the result of injury. Isolated *paralysis of the anterior crural nerve* and of the *obturator nerve* are quite uncommon, and when they do occur are generally the result of compression of the nerve within the abdominal cavity, either by growths or during the act of parturition. In affections of the anterior crural nerve, the movements of flexion of the thigh on the trunk and extension of the leg upon the thigh may both be impaired or lost. Wasting of the anterior thigh muscles, and diminution or loss of the knee-jerk, are other obvious signs of this condition. When the *obturator nerve is injured*, the patient can flex his hip but cannot adduct the thigh, and so, when sitting, he can raise his knee but cannot throw it across the other leg. He can walk about with no obvious disturbance of gait, but he cannot rotate the thigh either outwards or inwards, with any degree of force.

TABLE SHOWING THE MUSCLES TO WHICH THE VARIOUS NERVES OF THE LUMBAR AND SACRAL PLEXUSES ARE DISTRIBUTED.

Nerve.	*Muscles.*	
Obturator (L. 2, 3, 4) ..	Adductor longus Gracilis Adductor brevis	Obturator externus Adductor magnus
Anterior crural (L. 2, 3, 4)	Iliacus Pectineus	Sartorius Quadriceps extensor
Sciatic nerve (L. 4, 5, S. 1, 2, 3)	Semitendinosus Biceps	Semimembranosus
External popliteal (L. 4, 5, S. 1, 2)	Tibialis anticus Extensor proprius hallucis Extensor longus digitorum Peroneus tertius	Extensor brevis digitorum Peroneus longus Peroneus brevis
Internal popliteal (L. 4, 5, S. 1, 2, 3)	Gastrocnemius Plantaris Soleus Popliteus	Tibialis posticus Flexor longus digitorum Flexor longus hallucis
Internal plantar	Flexor brevis hallucis Abductor hallucis	Flexor brevis digitorum 1st lumbrical
External plantar	Accessorius Abductor minimi digiti Flexor brevis minimi digiti Interossei	Adductor obliquus hallucis Adductor transversus hallucis Outer 3 lumbricals
Nerve of the quadratus femoris (L. 5, S. 1)	Quadratus femoris	Gemellus inferior
Nerve of the obturator internus (L. 5, S. 1, 2)	Obturator internus	Gemellus superior
Nerve of the pyriformis ..	Pyriformis	
Superior gluteal nerve (L. 4 5, S. 1, 2)	Gluteus medius Gluteus minimus	Tensor vaginæ femoris
Inferior gluteal nerve (L. 5, S. 1, 2)	Gluteus maximus	

Paralysis of the main trunk of the sciatic nerve, which would include paralysis of all the muscles supplied by the internal and external popliteal nerves, points to disease or injury affecting the pelvis. It may be brought about by a fracture of the pelvis or of the upper end of the femur, or by injuries to the hip-joint; on the other hand, the sciatic nerve may be compressed by tumours or inflammatory masses within the pelvis. Such an extensive palsy has considerable effect on the patient's gait, as he is unable to flex the knee, and consequently has to use the leg as a stiff, extended support; the disability is increased by the absence of all movements at the ankle-joint. The sensory loss in such a condition includes the outer side of the leg and the whole of the foot, except a small area on its inner and upper aspect.

Palsy of the *external popliteal nerve* is the commonest isolated nerve palsy in the lower extremity. Not only is it particularly exposed to injury in its course through the popliteal space, and as it winds round the fibula, but a primary neuritis of it is by no means uncommon, especially in cases of diabetes mellitus and lead poisoning. Isolated paralysis of the external popliteal nerve has been observed frequently in tabes dorsalis. Its most obvious result is the dropped foot to which it gives rise, and the high-stepping gait which is necessary if the patient is to clear the ground with his toes.

Injury to the *internal popliteal nerve* is very much less common, but it may be involved by tumours or the products of inflammation in the upper part of the leg. Paralysis of the calf muscles is the chief consequence, preventing the patient from extending his foot and standing on tip-toe, or from making any springing movement in the attempt to walk or run. The paralysis of the interossei and the unopposed contraction of the long extensors may lead to CLAW-FOOT (p. 109).

TABLE SHOWING THE MUSCLES INNERVATED BY THE DIFFERENT ROOTS OF THE LUMBAR AND SACRAL PLEXUSES.[*]

L. 1, 2.	*Iliopsoas. Quadratus lumborum.* Sartorius. Cremaster. Quadriceps.
L. 3.	*Quadriceps.* Sartorius. Quadratus lumborum. Adductores femoris. Obturator externus.
L. 4.	*Adductores femoris.* Quadriceps. Sartorius. Tensor fasciæ femoris. Tibialis anticus. Extensor communis digitorum. Extensor hallucis.
L. 5.	*Tibialis anticus. Extensor communis digitorum. Extensor hallucis.* Peronei. Abductors and external rotators of the hip. Gastrocnemii. Long flexors of the toes. Hamstrings. Glutei.
S. 1.	*Gastrocnemii. Hamstrings. Long flexors of the toes.* Peronei. Abductors and external rotators of the hip. Glutei.
S. 2.	*Glutei. Intrinsic muscles of the foot.* Gastrocnemii. Hamstrings. Long flexors of the toes.
S. 3, 5.	*The musculature of the perineum connected with defæcation, micturition.*

In addition to these peripheral nerve palsies of the lower limb, we have to consider paralysis due to lesions of the roots leaving the lumbo-sacral region of the cord, and forms resulting from disease of that part of the spinal cord itself (see table above): atrophic palsy of one leg is not commonly the result of *spinal caries*, although it may occur when the caries affects the lower lumbar or sacral region. On the other hand, paralysis of one leg, generally associated with acute pain of root distribution, is not a very rare early symptom of *malignant disease of the lower part of the vertebral column.* In the absence of any obvious deformity, the diagnosis in such cases is often difficult, and much may depend on the use of skiagraphy or upon the history of a growth elsewhere which may have been removed, from the breast for instance, even years previously. In some cases a good deal may be learnt from observing loss of the natural spinal lumbar curve, and from a suggestion of shortening in the stature of the patient, and particularly by noticing the diminished interval between the lower ribs and the iliac crests. These are signs of collapse on the part of the softened vertebræ, and constitute a condition to which the name 'entassement' is applied.

Syphilitic meningitis, involving the roots of the lumbo-sacral cord, is another not very uncommon source of crural monoplegia. The diagnosis depends upon the history of syphilis, a positive Wassermann serum reaction, the results of an examination of the cerebrospinal fluid, and the fact that both the motor palsy and the sensory loss follow a root distribution.

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[*] The muscles which afford the most useful landmarks are printed in italics.

Probably more common than any other cause for atrophic paralysis of one leg is the disease known as *acute poliomyelitis* (p. 512).

Tumours of the spinal cord and *syringomyelia* are very much rarer causes of paralysis of one lower extremity, although the possibility of their occurrence may sometimes need to be taken into consideration. Various forms of *progressive muscular atrophy*, of either spinal or primary muscular origin, are more important causes of PARAPLEGIA (p. 510) than of unilateral paralysis. As a general rule they are symmetrical, or approximately symmetrical, in their onset and progress, but every now and then one may meet with cases in their earliest stage, when the complaint of the patient is referred to one limb only. A good example is afforded by the case of a lad who was brought to me on account of weakness in one foot, which had appeared quite insidiously and was tending to progress. The affected limb showed atrophic palsy of the long extensors of the toes and of the peroneal muscles. The diagnosis of *peroneal muscular atrophy* (p. 60) was confirmed by the appearance of similar physical signs in the other leg some months later. In addition to their early symmetrical distribution, these progressive degenerative diseases can be distinguished from gross diseases of the spinal cord and its envelopments by the absence of pain in the course of their evolution.

E. Farquhar Buzzard.

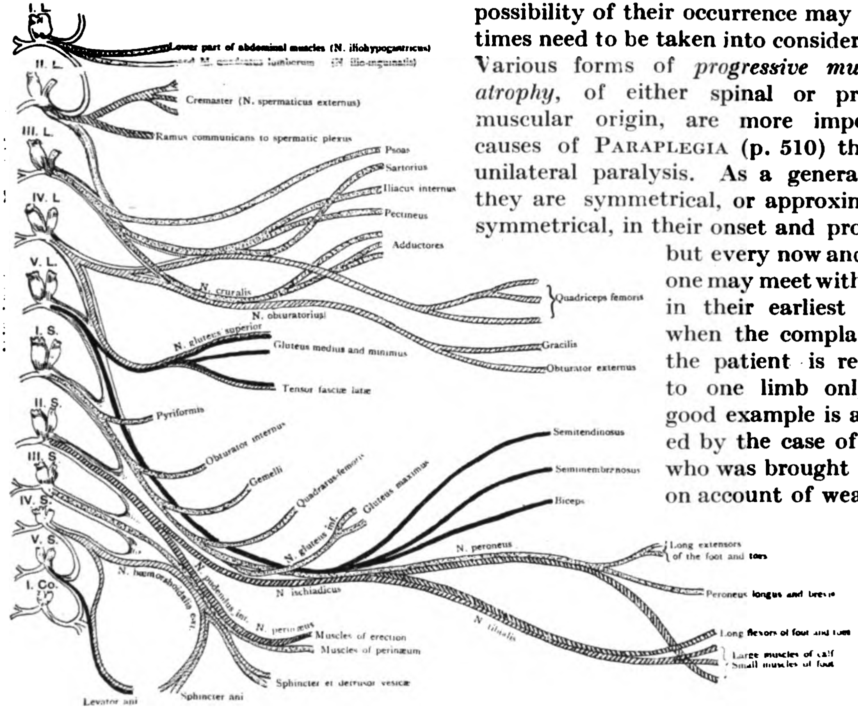


Fig. 205.—Diagram to illustrate the lumbo-sacral plexus and its branches (*after Kocher*).

PARALYSIS OF ONE EXTREMITY (UPPER).—The word 'paralysis' has come, by general use, to include partial as well as complete palsies, and to embrace all varieties of impaired voluntary movement. It is in this sense that the word is used for the purpose of this article. No other interpretation would be of value in discussing diagnosis, because the latter depends, not upon the degree of paralysis, but upon its nature, distribution, and associated phenomena. Accurate diagnosis is often most difficult, although perhaps more important from the point of view of successful treatment, when the limitation of voluntary movement is only slight. Before discussing the various forms of paralysis met with in the arm, some reference must be made to a few practical points which are important in the proper investigation of cases complaining of inability to use an arm.

The medical man must not be satisfied with the patient's statement that he has lost power or that he is weak in his limbs. Tests must be employed to ascertain whether this is really the case. The movements at each joint, of flexion, extension, pronation, supination, must be investigated, and if necessary their power measured against the observer's resistance. It may be found that the grasp is powerful in a patient who is unable to use his hand on account of loss of control over the finger movements. In such a case there is not paralysis, but inco-ordination (see ATAXY, p. 55). Similarly, there is certain to be difficulty in carrying out delicate movements if there is loss of cutaneous sensibility. Without tactile sense it is impossible to handle a pen in a proper manner. Sometimes a patient will complain of loss of power, when investigation shows that the ability to execute movements is inhibited by the pain in a muscle or joint evoked by the attempt. In other instances, mechanical limitation of movement by arthritic changes, without pain, may lead the patient to believe that there is loss of power. He finds he cannot lift his arm, and ascribes the disability to paralysis instead of to ankylosis of the shoulder-joint. On the other hand, it must be remembered that pain and loss of power may be associated in some forms of neuritis. If the patient says, " My arm is so painful that I cannot lift it," examination must be directed to ascertain whether the inability is due only to painful inhibition or to real paralysis in addition.

Stress must be laid upon the necessity for obtaining a careful history, and especially an accurate account of the duration of the trouble, whether its onset was sudden, rapid, or slow and progressive, and whether the loss of power was accompanied or preceded by pain, numbness, or tingling. The family and previous history must not be neglected. In examining the paralyzed arm, care should be taken that the whole of *both* upper limbs, as well as the neck, upper part of thorax, and shoulders are stripped, so as to be inspected easily and the two sides compared. It will also be necessary, in the large majority of instances, to investigate the functions of the cranial nerves and the reflexes, etc., of the trunk and lower extremities. This is often imperative even when no complaint is made of loss of power or other symptoms in any part of the body except one upper limb. The importance of this full examination is perhaps obvious, but it may be illustrated by reference to two points. A lesion of one internal capsule may give rise to paralysis of the opposite arm, but it will be likely to cause, in addition, some alteration in the abdominal and leg reflexes of the corresponding side. Similarly, a lesion of the 8th cervical or 1st dorsal spinal segments, or of their corresponding spinal roots, will also affect the fibres leaving the cord at that level and passing, *via* the cervical sympathetic, to the eye of the same side. In this way atrophic paralysis of the muscles of one hand may be associated with a small pupil and a small palpebral fissure on the same side, a coincidence which at once points to the cord or roots as the site of the lesion, and acquits the peripheral nerves of being concerned in the production of the palsy. In such a case the further investigation of the abdominal reflexes, the knee-jerks, and plantar responses, will help to decide whether the lesion is intramedullary or extramedullary ; in the former event the abdominal reflex on the same side would be absent, the knee-jerk would be increased, and the plantar response would be of the extensor type, while in the latter, unless the lesion exerted considerable pressure on the cord, the reflexes below the arm would be normal.

Any attempt to enumerate, let alone discuss, all the possible lesions which can give rise to paralysis in the upper extremity, is out of the question, and we must be content to consider the broad principles of diagnosis in connection with the more familiar instances of brachial palsy. For this purpose a classification based chiefly on the presence or absence of muscular atrophy will be adopted. This will be of practical use because the mere inspection of a paralyzed limb generally enables the observer to detect whether a case belongs to the one category or the other.

PARALYSIS WITHOUT MUSCULAR ATROPHY.

This heading embraces cases in which there may be general impairment of nutrition, and perhaps muscular wasting, due to disuse, but in which there is no localized muscular atrophy and no alteration in the response of the muscles to electrical stimulation. The cases may be divided into two groups : (1) Those in which there is some affection of the upper motor neuronic system (pyramidal lesions), and (2) Cases without lesion of the pyramidal tract.

Paralysis due to Pyramidal Tract Lesions.—The most familiar example of this group is afforded by cases of brachial monoplegia due to a *vascular lesion* (*thrombosis, hæmorrhage*, or *embolism*) *in the internal capsule* or other part of the pyramidal tract in its course through the brain. In the diagnosis of this condition the points of importance are : The presence of some cardiovascular condition capable of producing the lesion, such as disease of the heart, kidneys, or arteries ; the sudden or rapid onset of the symptoms, with or without loss of consciousness or other cerebral disturbance. The arm retains its natural contours, and the muscles are not atrophied, although they may appear, after some time has elapsed, to be smaller than those of the other arm. The paralysis may affect the whole limb and include inability to shrug the shoulder; or the movements of the hand and fingers may be more impaired than those of the elbow and shoulder. There is a tendency for the arm to exhibit more and more resistance to passive movement, that is to say, to develop spasticity. At the same time, if left to itself, the limb will adopt a fixed position, which includes adduction of the upper arm to the trunk, flexion and pronation of the forearm, and flexion of the wrist and fingers. If any movements are possible, they will be those of flexion rather than of extension at the various joints. The muscle tone is increased, and the tendon-jerks, such as the ulnar and radial wrist-jerks, the triceps, biceps and supinator jerks, are exaggerated when compared with those of the opposite limb. Eventually contractures may develop, and it will be found impossible to extend the upper arm, forearm, hand, and fingers into one straight line.

Such is the clinical picture afforded by spastic paralysis of the arm, and one case will differ from another only in the degree of spasm and the degree of paralysis; but the amount of spasticity and the paresis do not always correspond. In one patient the rigidity forms the chief obstacle to voluntary movement; in another the arm, though powerless, shows comparatively little increase in tone.

The fact that the pyramidal fibres destined for the face, trunk, and leg run in close proximity to those for the arm, is sufficient reason for suspecting that, even if no other paralysis is complained of, there may be signs of disturbed function in other parts. The side of the face corresponding to the paralyzed arm may not move so quickly or so powerfully as the other side in a voluntary effort to show the teeth, although no difference may be detected when the patient smiles. The corresponding abdominal reflexes may be found wanting. The knee-jerk may be increased; ankle-clonus and an extensor plantar response may be elicited; all on the same side.

This spastic arm, in all degrees of severity, may result not only from a vascular lesion in the brain, but also from a *cerebral abscess*, a *cerebral tumour*, or *cerebral inflammation* (encephalitis). The arm will present identical features, so that the diagnosis must be made from a consideration of other data. Thus a *cerebral abscess* only becomes likely when there is some infective process either in the bones of the skull (mastoid or frontal sinus disease) or in a distant part such as the heart or lungs (ulcerative endocarditis or bronchiectasis). Headache, vomiting, and optic neuritis, with a slow pulse, slow respiration, and subnormal temperature, may help in the diagnosis. In cases of *cerebral tumour* the development of the brachial palsy is nearly always slow and progressive, spreading from one part of the limb to another, and again there may be headache, vomiting, and optic neuritis. It should be remembered, however, that these signs of increased intracranial pressure are not always present, and that the presence of a tumour is always to be suspected when a spastic paralysis of one limb comes on in a slow and progressive manner. Some tumours grow at the expense of neighbouring tissues in such a way that pressure is raised but little or not at all. *Encephalitis* will need to be considered when there is a history of acute constitutional disturbance with fever, vomiting, headache, and perhaps convulsions preceding or attending the onset of the paralysis. The latter, however, is not progressive. It reaches its maximum within a few hours, and shows a general tendency to improve after the acute symptoms have passed off.

Disseminated sclerosis is another disease in which a spastic monoplegia is not uncommon. The diagnosis is easy if it occurs late in the disease, when nystagmus, optic atrophy, spastic paraplegia, and sphincter trouble are already present, or if there is a history of previous transient palsies affecting other limbs. When, however, paralysis of one arm is the first symptom, as it may be, the diagnosis may be difficult. The rapid onset of the palsy in a healthy young adult without constitutional disturbance, severe headache, or vomiting, and perhaps the discovery of absent abdominal reflexes and an extensor plantar response, should direct suspicion to the possibility of a patch of disseminated sclerosis being responsible for the trouble.

Diseases of the *pons, medulla, and that part of the spinal cord which lies above the cervical enlargement*, whether vascular, inflammatory, or neoplastic, may cause spastic palsy of the upper limb, but it is rarely a monoplegia. The arm and leg on one side, or both arms and both legs (double hemiplegia), are much more likely to be involved simultaneously, and the site of the lesion is inferred from the knowledge that the two pyramidal tracts are in close proximity in those regions.

Paralysis without Lesions of the Pyramidal Tract.—It is not uncommon for a patient in the earliest stage of *paralysis agitans* to complain of loss of power in one arm. This sometimes leads to a wrong diagnosis, the trouble being described vaguely as due to neuritis, or even hysteria. This mistake will be avoided if notice is taken of the fact that the limb is not only weaker than its fellow, but that it is somewhat stiff and conspicuously slow in carrying out movements. A lack of expression in the face, or tendency to carry the arm in a flexed position across the trunk, and perhaps some hesitancy in the gait, should guide the observer to a correct diagnosis even if tremor is absent, as it often is at this stage of the malady. This form of paralysis is unattended by changes in the reflexes.

Children suffering from *chorea*, and especially hemichorea, are often brought to a doctor with the complaint that he or she has lost the use of an arm. Examination will show that there is really some weakness of the affected limb, which is demonstrated, not so much by the poorness of the grasp, as by the fact that the child is unable to maintain a steady pressure. He will grasp the observer's fingers, but quickly release the pressure, although urged to continue the squeeze. In the same way, when asked to put out his tongue he will do so, but withdraw it at once. When required to extend his arm in front of him with the palm of the hand facing downwards, it will generally be noticed that the wrist is slightly flexed although the fingers are extended. These are points which may be useful in coming to a right conclusion when choreic movements are not conspicuous; but attention must also be paid to the condition of the heart and to any history of rheumatism. No information of value can be obtained from the reflexes unless perhaps the choreic form of knee-jerk is present (p. 359).

Hysterical brachial palsy may resemble one due to a pyramidal lesion in presenting a marked amount of rigidity, or, on the other hand, the whole limb may be flaccid and limp. Some general wasting of the muscles may be present, but there is no alteration in their electrical reactions. Organic pyramidal lesions must be excluded by an examination of the reflexes. The supinator, biceps, and triceps jerks may be tried, but they will not be appreciably more brisk than those of the opposite limb. The abdominal and leg reflexes will be natural. If the limb is rigid the observer will probably be able to overcome the rigidity by steady pressure, and to extend the arm, forearm, hand, and fingers into one straight line. When the patient is asked to perform a certain movement, the observer can often see that in the effort to carry it out the antagonistic muscles are put into action rather than, or as well as, those which are necessary for its execution. Thus the triceps will contract as well as the biceps when the patient is requested to flex the elbow, with the result that the forearm is moved very little or not at all. This may also be demonstrated when the observer resists the movement of flexion by grasping the wrist and then unexpectedly relaxes his resistance; in an organic palsy this will be followed by further uncontrolled flexion at the elbow, whereas in a hysterical patient the contraction of the triceps maintains the forearm in its former position. Another important point in distinguishing a palsy of cerebral origin from one which is hysterical is that in the organic case, even when no voluntary movement whatever can be carried out by the fingers, the latter may move involuntarily in association with energetic movements in the opposite limb. Thus, when the patient is asked to grasp some object as tightly as he can with the sound hand, flexion of the fingers may be detected in the paralyzed side. The same phenomenon is seen in connection with involuntary movements, such as yawning. The writer remembers being requested to see a case in which there was paralysis of one arm, and in which the diagnosis between organic and functional disease was in doubt. The first question he asked the patient was whether he could open his hand and extend his fingers; the patient replied in the negative, but immediately volunteered the statement that the fingers became extended whenever he yawned. This settled the point in dispute at once, because such associated movements do not occur in hysterical palsies. In many, if not most, cases of hysterical palsy of an arm, the limb is also anæsthetic, and this anæsthesia can generally be recognized as hysterical on account of its complete character. In a cerebral palsy there may be some loss of sensibility to light touches and some impairment of pain sense, but the hysterical patient is usually insensitive to all forms of stimulation, even pinching or a strong faradic current. Morover, the distribution of the anæsthesia does not correspond to any form seen in organic disease, and is frequently of a glove or sleeve type with a very sharp line of demarcation.

PARALYSIS WITH MUSCULAR ATROPHY (OR ATROPHIC PALSY).

In this category are included all cases of brachial palsy in which there is true muscular atrophy associated with some alteration in electrical reactions, either the typical reaction of degeneration or quantitative diminution of excitability to galvanic and faradic currents. In all such cases there is some organic lesion affecting some part of the lower motor neurons ; there must be some disease involving (1) the spinal segments from the 5th cervical to the 1st dorsal, (2) the corresponding anterior spinal roots, (3) the brachial plexus, (4) the peripheral nerves of the arm, or (5) the muscles themselves.

In addition to atrophy and alteration in electrical response, each paralyzed muscle tends to lose its tendon-jerk. For instance, the tendon-jerk of an atrophied biceps cannot be obtained, and in all probability direct percussion of the muscle itself will also fail to elicit a contraction, or will give rise only to an abnormally slow contraction. Muscles which are undergoing atrophy may also exhibit fine fibrillary contractions of a spontaneous kind, but these are seen only when the disease affects the nerve fibres, and not when the muscles themselves are affected primarily. These fibrillations are very rarely seen in the group of muscular atrophies to which the name of ‘ myopathy ’ is given.

When making a diagnosis of the site of the lesion in cases of atrophic brachial paralysis, it is essential to analyze carefully the distribution of the atrophied muscles. This must be done in order to answer the questions : Are all the atrophied muscles supplied by one peripheral nerve, or are they innervated by one or more spinal segments, or by one or more anterior spinal roots ? The diagnosis will be comparatively simple when it is found, for instance, that all the atrophied muscles are supplied by the musculospiral nerve, and that all the muscles supplied by that nerve are atrophied and paralyzed. A lesion of that nerve can then be diagnosed and its nature inferred from other data, such as the use of a crutch or the history of a fractured humerus, with the detection of callus involving the nerve at the site of the fracture.

Let us now consider briefly some of the various conditions giving rise to atrophic palsy of the upper extremity, and the features which are most characteristic for the purpose of their diagnosis.

In cases of *neuritis* there may be paralysis of the muscles supplied by one nerve only, or of muscles supplied by several nerves (multiple neuritis). In the former case the correct diagnosis of the lesion depends on a knowledge of the muscles innervated by each of the chief brachial nerves, and this may be gleaned from the following list :—

Nerve.	*Muscles.*	
Posterior scapular (C. 5)	Levator anguli scapulæ Rhomboideus minor	Rhomboideus major
Long thoracic (C. 5, 6, 7)	Serratus magnus	
Suprascapular (C. 5, 6)	Supraspinatus	Infraspinatus
Anterior thoracic (C. 5, 6, 7, 8, D. 1)	Pectoralis major	Pectoralis minor
Musculocutaneous (C. 5, 6)	Biceps Brachialis anticus	Coraco-brachialis
Median (C. 6, 7, 8, D. 1) forearm	Pronator radii teres Flexor carpi radialis Palmaris longus Flexor sublimis digitorum	Flexor longus pollicis Pronator quadratus Flexor profundus digitorum (outer half)
hand	Abductor pollicis Opponens pollicis	Flexor brevis pollicis (superficial head) Two outer lumbricals
Ulnar (C. 8, D. 1) forearm	Flexor carpi ulnaris	Flexor profundus digitorum (inner half)
hand	Palmaris brevis Flexor brevis minimi digiti Abductor minimi digiti Opponens minimi digiti Interossei	Two inner lumbricals Adductor obliquus pollicis Adductor transversus pollicis Flexor brevis pollicis (deep)

Nerve.	*Muscles.*	
Circumflex (C. 5, 6)	Deltoid	Teres minor
Musculospiral (C. 6, 7, 8) upper arm	Triceps Anconeus	Supinator longus Extensor carpi radialis longior
forearm (posterior interosseus branch)	Extensor carpi radialis brevior Supinator brevis Extensor communis digitorum Extensor minimi digiti Extensor carpi ulnaris	Extensor ossis metacarpi pollicis Extensor longus pollicis Extensor brevis pollicis Extensor indicis
Subscapular (C. 5, 6, 7, 8)	Subscapularis Teres major	Latissimus dorsi

When several nerves are involved in neuritis, the condition is one of *multiple neuritis* and, being generally due to some toxic cause, tends to be bilateral and symmetrical. Multiple neuritis is further characterized by the facts that the peripheral muscles are more affected than the proximal, that the extensors of the wrist and fingers suffer out of proportion to the flexors, and that there are often pain and tenderness in the paralyzed muscles. These features are present in *alcoholic neuritis*, the most common form (p. 66). In *lead palsy* the extensors of the wrists and fingers are particularly susceptible, although other muscles supplied by the musculospiral nerve—such as the supinator longus and triceps—may escape altogether. The association of dropped wrist with a blue line on the gums, and other signs of plumbism (p. 34), is diagnostic of this form of brachial palsy. In some cases of multiple neuritis it is impossible to identify the causative toxin, but glycosuria, mercury, and arsenic, in addition to alcohol and lead, must be remembered in this connection. Leprosy may produce a precisely similar condition (*Fig.* 206), but it is rarely met with in Great Britain. (See also Atrophy, Muscular, p. 59.)

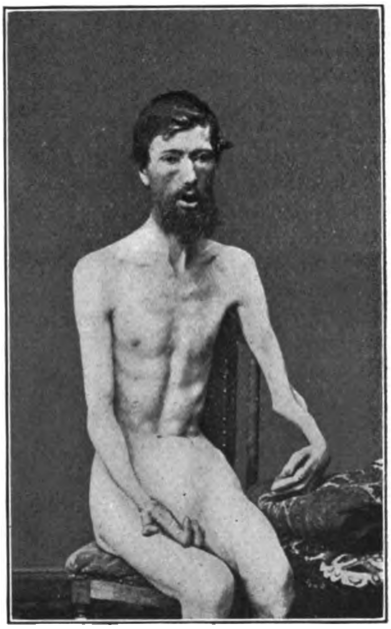


Fig. 206.—Paresis of the arms due to peripheral neuritis in an arrested case of lepra maculo-anaesthetica in a Norwegian. (*From a photograph by the late Dr. Hansen, Inspector-General of Leprosy in Norway.*)

In most cases of single nerve palsy the diagnosis, based on the distribution of the muscular atrophy and paralysis with altered electrical reactions, can be confirmed by the detection of sensory loss in the cutaneous area supplied by the same nerve. In other cases the sensory fibres appear to offer more resistance than the motor to the exciting cause of the neuritis, and little or no disturbance of sensibility can be found. (For areas supplied by the peripheral nerves, see *Plate XXV*, p. 608, and Sensation, some Abnormalities of, p. 604).

Reference may be made to one or two of the single nerve palsies which present special points in relation to diagnosis.

In *paralysis of the serratus magnus* (*Fig.* 207) due to injury or neuritis of the long thoracic nerve, the patient may complain of general weakness of the arm, and particularly of inability to raise it above the horizontal position. The trouble arises from the fact that the scapula is no longer held against the thoracic wall, and cannot be rotated with the movements of the limb. The origin of this disability may be overlooked if the arm only is examined. If, however, the position of the scapula is observed when the arm is moved in different directions, the correct diagnosis can be arrived at. When the arm hangs at rest by the side, the scapula is seen to be slightly raised and displaced outwards, with the inferior angle inclining towards the vertebral column and perhaps somewhat separated from the chest wall (*Fig.* 207). When the arm is raised forward to the horizontal position and pressed against some resistance, the inner border of the scapula projects backwards and presents a 'winged' appearance. This deformity ought always to suggest paralysis of the serratus magnus.

The movements of the upper limb are also hampered somewhat in cases of neuritis or injury to the *suprascapular nerve*, and the patient may complain of difficulty or fatigue in writing. Examination will show flattening of the infraspinous fossa and weakness in rotating the humerus outwards against resistance. The infraspinatus muscle may show the reaction of degeneration, but the supraspinatus is not accessible, being covered by the trapezius.

Adhesions within the shoulder-joint, with secondary wasting of the deltoid, may simulate paralysis in the distribution of the *circumflex nerve*, owing to the difficulty in abducting the arm ; but a little care in examination and electrical testing of the deltoid muscle will suffice to make a diagnosis. In circumflex palsy, moreover, some sensory loss may be found in the skin over the upper and outer aspect of the arm.

In cases of *musculospiral paralysis* it must be remembered that the injury to the nerve may be above or below the points where branches leave to supply the triceps and supinator longus muscles, and that these muscles may therefore escape. Sensory symptoms are often absent, but some anæsthesia is sometimes found on the radial border of the hand.

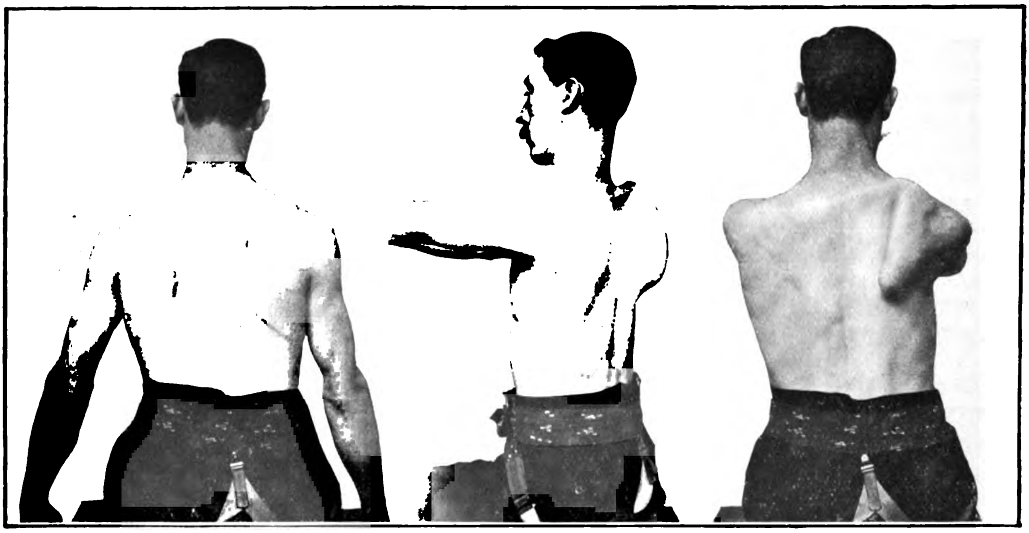


Fig. 207.—Photographs illustrating the deformities produced by palsy of the right serratus magnus. It is often associated with weakness of the lower trapezius fibres, and is found most commonly in carpenters and sawyers.

In connection with traumatic affections of the *median nerve*, the distribution of the paralysis depends on the site of the wound ; the branch which supplies the muscles of the hand may leave the main nerve in the forearm, and thus escape injury when the wound is at the wrist.

Ischæmic paralysis of the hand (Volkmann's contracture, *Fig.* 58, p. 141) must not be forgotten in cases of injury to the forearm when there is a history of the patient wearing a splint, and the condition must not be mistaken for ulnar or median paralysis. The diagnosis depends partly upon the history, but chiefly upon the rigid contracture of *all* the flexor tendons of the wrist and fingers with wasting of the muscles and other trophic changes. The electrical excitability of the flexor muscles is sometimes impaired.

Paralysis of one arm due to a *lesion of the brachial plexus* is a common event, the most frequent cause being some injury. When the whole plexus is damaged, complete brachial palsy, with atrophy of the muscles and extensive sensory loss, results. The diagnosis of such a lesion is simple, because it would be impossible for the spinal cord to be damaged sufficiently to bring about such a paralysis without giving rise to symptoms of atrophic palsy in the opposite arm, and spastic paralysis, with disturbances of sensibility, in the trunk and legs.

In addition to a lesion of the brachial plexus as a whole, two forms of partial palsy are not uncommon, and have received special names. The first is known as *Erb's palsy*, and is due to a lesion of the upper trunk of the brachial plexus, composed of fibres from the 5th and 6th cervical roots. The paralyzed muscles include the spinati, deltoid, biceps, supinator longus, and to a less extent the extensors of the wrist and fingers. The arm hangs by the side, and the forearm remains pronated owing to weakness of the supinator muscles, and especially of the biceps. There is sometimes, but not always, anæsthesia over the outer aspect of the forearm and hand. This form of palsy is usually produced by a fall on the shoulder of such a kind as to separate the latter forcibly from the head, and so to exert sudden and severe traction on the upper part of the brachial plexus. A similar lesion is often seen in infants as a result of injury during birth, and has been called *Duchenne's palsy*, after the observer who first described it. In both Erb's and Duchenne's palsy, the grouping of the paralyzed muscles resembles that which may follow an injury to the 5th and 6th cervical segments of the spinal cord, but in the latter case bilateral symptoms are practically certain to be present, as well as more extensive disturbances of sensation of the trunk and limb, probably of the dissociative type. (See SENSATION, SOME ABNORMALITIES OF, p. 604.) In spinal cord lesions, moreover, we may see an atrophic paralysis of the muscles supplied by the 5th and 6th cervical segments, together with a spastic paralysis of the remaining muscles in the arm—that is to say, of the muscles innervated from the 7th and 8th cervical and 1st dorsal segments. This mixture of atrophic and spastic paralysis in the upper limb can only be brought about by some injury or disease of the spinal cord.

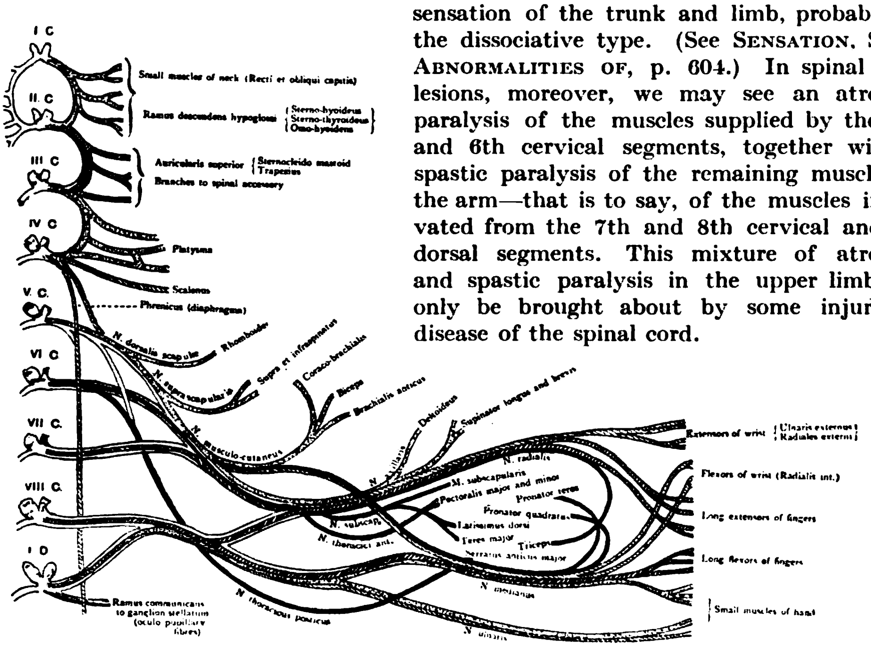


Fig. 208.—Diagram to illustrate the cervico-brachial plexus and its branches (*after Kocher*).

The other form of partial brachial plexus palsy, *Klumpke's palsy*, depends on a lesion of the trunk formed by the 8th cervical and 1st dorsal roots. The flexors of the wrist and fingers, and the intrinsic muscles of the hand, undergo atrophy, and although the patient can carry out all movements at the shoulder and elbow, he is unable to use his fingers. The area of anæsthesia in this form involves the ulnar border of the forearm and hand from the elbow downwards. This condition may be distinguished from a spinal-cord lesion affecting the 8th cervical and 1st dorsal segments, not only by its limitation to one upper extremity, but also by the absence of the oculo-pupillary symptoms which are nearly always present in spinal lesions of that level ; a lesion of the spinal segments in that region, or of the corresponding spinal roots in their intravertebral course, produces a diminution in the size of the pupil and a narrowing of the palpebral aperture on the same side. Such a pupil does not dilate to shade, nor when the skin of the neck is pinched, nor when a cocaine solution is dropped into the eye. Although the presence or absence of oculo-pupillary symptoms affords a point of differentiation between lesions of the 8th cervical and 1st dorsal segments of the spinal cord, or of their corresponding roots on the one hand, and a lesion of the lower trunk of the brachial plexus on the other, in the majority of cases, in very severe injuries to the neck the lower roots of the plexus may be actually torn away from their connection with the spinal cord, in which case the oculo-pupillary symptoms mentioned above will be present.

Amyotrophic lateral sclerosis is a condition dependent on a gradual degeneration and disappearance of the anterior cornual cells of the spinal cord, associated with sclerosis of the upper motor neuron tracts. The muscular atrophy begins insidiously and progresses gradually. It often begins in the intrinsic hand muscles (Aran-Duchenne type), less commonly in the shoulder and upper arm muscles. The loss of power is in proportion to the amount of atrophy. The alteration in electrical reactions is usually more a quantitative diminution of excitability to both currents than a true reaction of degeneration. Fibrillary contractions are common. The atrophy usually begins in one limb before the other, but soon becomes more or less symmetrical. The tendon-jerks of all muscles which are not atrophied are exaggerated. The abdominal reflexes may be absent, and the plantar reflexes may be of the extensor type. There are no pains, no sensory loss, and no oculo-pupillary phenomena.

In *syringomyelia* the spinal changes frequently begin in the cervical enlargement of the cord, with the result that atrophic paralysis is first noticed in the upper extremity, generally in the intrinsic muscles of the hand and the flexor muscles on the ulnar aspect of the forearm. The onset is insidious and the progress gradual. One limb is generally affected many months, perhaps years, before the other. Manual deformities are common. (See CLAW-HAND, *Fig.* 45, p. 110.) The electrical reactions vary like those of amyotrophic lateral sclerosis. The knee- and ankle-jerks are increased as a rule, and spastic paralysis of the lower extremities usually develops in the later stages, with extensor plantar responses. Pains shooting down the arms from the neck occur, but are not common. Trophic changes develop in the skin, subcutaneous tissues, and joints. There is sensory loss of a dissociative type, i.e., loss of sensibility to pain, heat, and cold, with preservation of tactile sensibility. Oculo-pupillary phenomena and nystagmus are common. Spinal curvature, in the form of a dorsal scoliosis, is another frequent physical sign. It is not necessary that all these signs and symptoms should be present for making a diagnosis. The combination of muscular atrophy, dissociative anæsthesia, and trophic changes in the skin is usually sufficient.

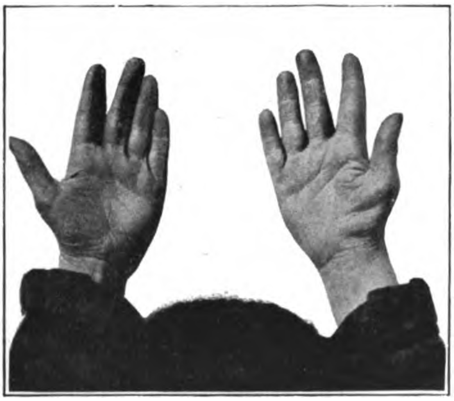


Fig. 209.—Atrophic palsy of right hand associated with long 7th cervical rib of that side. Note the marked wasting of the abductor pollicis as compared with the other muscles of the thenar eminence. According to S. A. K. Wilson this is a common feature of these cases, but it is not pathognomonic.

(*Photo by Dr. P. W. Saunders.*)

A *cervical rib* may be responsible for atrophic palsy in the upper extremity. Paralysis is usually preceded by pain, chiefly referred to the inner aspect of the arm, and sometimes shooting into the little and ring fingers. The pain is worse after exertion, and often relieved by placing the hand behind the head. Muscular atrophy begins in the hand muscles (*Fig.* 209), the interossei, thenar, and hypothenar eminences, and often involves the flexors of the wrist and fingers. It develops gradually and does not spread beyond the distribution just described. There is often sensory loss in regions corresponding to the cutaneous areas supplied by the 1st dorsal and 8th cervical spinal roots. The loss is usually less marked to touch than to painful and thermal stimuli. The atrophied muscles show reaction of degeneration. There are no oculo-pupillary phenomena and no signs of disease in other parts of the body. The condition is usually one-sided, occasionally bilateral, and the chief point in diagnosis is the discovery of the ribs by means of skiagraphy (*Fig.* 187, p. 443). At the same time it must not be concluded that the absence of a rib shadow in an x-ray photograph is in all cases a contra-indication to the diagnosis. A ligamentous band, undetected by the skiagraphic examination, is sometimes found to occupy the position of a supernumerary rib and to be responsible for similar results in the way of pain and atrophic palsy. (See also PAIN IN THE EXTREMITY, UPPER, p. 442 ; and CLAW-HAND, p. 109.)

Another disease in which atrophic palsy of the intrinsic hand muscles is a prominent feature is *peroneal atrophy.* As the name suggests, the muscular atrophy and paralysis generally begin in the lower, before they affect the upper, extremities, and further details concerning the condition may be found under Paralysis of One Extremity, Lower, p. 498 ; and Atrophy, Muscular (*Figs.* 20 and 21, p. 60).

The diagnosis of muscular atrophy in the arm dependent on a preceding *acute poliomyelitis* (*Fig.* 19, p. 59) is not difficult if an accurate history can be obtained. The onset was acute, with constitutional disturbance, varying from a transient and perhaps overlooked malaise to a pyrexia up to 104° F., with generalized pains all over the body, vomiting, and convulsions. It is almost invariable that the initial paralysis was more extensive than that which remained permanent. Attention to the distribution of the paralysis shows that it is irregular and different in every case, but there is more liability on the part of the shoulder and upper arm muscles to suffer than those of the forearm and hand. If both arms are affected there is little likelihood of any great degree of symmetry in the distribution of the atrophy. With regard to the electrical reactions, much will depend on the stage at which the case comes under observation. Some muscles may show the reaction of degeneration, others may respond fairly well, and others show no response whatever to either faradism or galvanism. Vasomotor changes, general defects in the growth of the limb, deformities, and contractures are common, but no sensory changes and no oculo-pupillary phenomena are to be observed. Only those reflexes are altered or lost which are concerned with atrophied muscles.

Hæmorrhage into the spinal cord, or *hæmatomyelia,* due to injury, occurs more often at the level of the 8th cervical and 1st dorsal segments than at any other. The resulting paralysis has much the same distribution as that described in Klumpke's palsy (p. 507), but the diagnosis may be made from the fact that the former also produces spastic paralysis of the trunk and legs, and frequently gives rise to areas of dissociated anæsthesia. Oculo-pupillary phenomena are usual as well in cases of hæmatomyelia at this level. Injuries to the cord result in bilateral symptoms, whereas Klumpke's palsy is confined to one arm.

Various forms of root palsies may be caused by *tuberculous* or *malignant disease of the vertebræ,* and also by *pachymeningitis,* frequently syphilitic. The diagnosis of the nature of such lesions depends on examination of the vertebral column and of the cerebrospinal fluid. The symptoms are more often bilateral than unilateral, and may be complicated by the results of pressure on the spinal cord, leading to spastic paralysis of parts below the level of the disease.

Tumours originating in the meninges or in the spinal cord at the level of the cervical enlargement are rare, but they may produce atrophic paralysis of the arm muscles, with spastic paralysis of the trunk and lower extremities. These phenomena may be more marked on one side at first, but they tend to become bilateral with the gradual growth of the tumour.

In the group of diseases to which the name *myopathy* or *muscular dystrophy* is applied, the arm is often more or less completely paralyzed. The diagnosis of this condition depends on a consideration of various factors. The gradual onset and the bilateral symmetry of the affection, the marked involvement of the shoulder and upper arm as compared with the forearm and hand, are important characteristics. The absence of fibrillary contraction and of the reaction of degeneration are also to be noted, while the history of a similar affection in other members of the family, and the presence of muscular atrophy or muscular pseudo-hypertrophy in other parts of the body, serve to confirm the diagnosis.

Table Showing the Muscular Distribution of the Various Nerve Roots of the Brachial Plexus.*

C. 5.	*Deltoid. Spinati. Teres minor. Rhomboids.* Diaphragm. Biceps. Supinator longus. Serratus magnus. Pectoralis major. Brachialis anticus. Coraco-brachialis.
C. 6.	*Biceps. Coraco-brachialis. Brachialis Anticus. Supinator longus.* Deltoid. Spinati. Teres major. Serratus magnus. Pectoralis major. Subscapularis. Pronators of forearm. Extensors of wrist.
C. 7.	*Triceps. Extensors of wrist and fingers.* Pronators of forearm. Pectoralis major. Subscapularis. Latissimus dorsi. Teres major.
C. 8.	*Flexors of wrist and long flexors of fingers.* Interossei and lumbricales. Muscles of thenar and hypothenar eminences.
D. 1.	*Muscles of the thenar and hypothenar eminences. Interossei and lumbricales.* Flexor carpi ulnaris. Oculo-pupillary fibres.

* Those muscles which are the most useful 'landmarks' for individual segments are printed in italics.

A glance at this list shows that nearly all muscles derive innervation from more than one spinal segment, generally from two or three. The table does not purport to give a complete anatomical list of all the muscles of the arm, but provides a guide to clinicians in their endeavours to localize spinal or root lesions from the distribution of atrophic muscular paralysis. *E. Farquhar Buzzard.*

PARAPLEGIA implies partial or complete paralysis of both legs, with or without part of the trunk. It does not, however, include inability to walk owing to mechanical defects, such as old fractures, joint disease. and so forth ; it is due, as a rule, to changes either in the brain, the spinal cord, the peripheral nerves, or in the muscles themselves, though sometimes it is caused by errors of function without any structural change in the neuro-muscular system. For clinical purposes, although naturally a paraplegia that has arisen in childhood may persist into adult life and thus cause overlapping of the classification, paraplegia in children may be discussed separately from paraplegia in adults. Let us suppose that the patient is a child, and that the chief complaint is weakness or paralysis of both legs. The following table indicates some of the causes that may produce this condition :—

I. THE CAUSES OF PARAPLEGIA IN CHILDREN.

(*A*). *Conditions in which there is no definite local disease, though there may be some general pathological condition* :—

(1) Simple delayed walking ; (2) Rickets ; (3) Cretinism ; (4) Idiocy.

(*B*). *Paraplegia associated with a definite upper neuron nerve lesion* :—

1. Infantile diplegia due to : (*a*) Congenital defect of the cortex : porencephalus ; (*b*) Infantile encephalitis ; (*c*) Injury, for example by forceps at delivery ; (*d*) Superior longitudinal sinus thrombosis ; (*e*) Meningitis ; (*f*) Congenital or acquired hydrocephalus.
2. Congenital malformation, such as meningocele, spina bifida, or spina bifida occulta.
3. Spinal caries, with compression of the spinal cord.
4. Friedreich's ataxy.

(*C*). *Paraplegia due to a lesion of the lower neuron type* :—

1. Acute anterior poliomyelitis, leading to infantile paralysis.
2. Tooth's peroneal type of progressive muscular atrophy.
3. Peripheral neuritis.

(*D*). *Paraplegia of the primary muscular type* :—

1. Pseudo-hypertrophic muscular paralysis.
2. The infantile type of primary muscular dystrophy.
3. The juvenile type of primary muscular dystrophy.

In arriving at the diagnosis, the first point to pay attention to is the history ; the case will belong to one or other of two main groups, the first containing those that have never been able to use the legs properly, the second those that have lost the use of the legs after having been at one time able to walk, or otherwise use them efficiently.

To the first group belong all cases of congenital malformation, such as *hydrocephalus* or *meningocele*, and most cases of *infantile diplegia*. Before diagnosing any of these, however, it is necessary to exclude *rickets*, *cretinism*, *idiocy*, and *simple delayed walking*, as causes of an apparent rather than real paraplegia. These cannot be the sole diagnosis if there is absolute flaccidity on the one hand, or if there is spasticity upon the other. It is important to remember how deceptive the reflexes may be ; almost any illness in an infant or young child—bronchopneumonia for example, or simple diarrhœa—may so depress the knee-jerk that it is often unobtainable until the patient's general health is restored ; the plantar reflex is normally more often extensor than flexor in infants ; ankle-clonus, however, does not occur except when there is degeneration of the lateral columns. If there is neither absolute flaccidity nor intermittent spasticity, and if the limbs are moved spontaneously, the mere fact that the child is late in walking by no means necessarily indicates nerve disease ; the delay may commonly extend to the second year, and occasionally even to the third or fourth. The main factor in making a diagnosis in such a case is time, for, until with the lapse of time the little patient begins to walk, it may sometimes be difficult to exclude organic paraplegia. If there are definite signs of rickets, or if the patient is a cretin or an idiot, the diagnosis is more obvious. The good effects of giving thyroid extract over a prolonged period may be the only conclusive means of distinguishing cretinism from idiocy, and this remedy should be employed in all such cases, however hopelessly idiotic the infant may seem to be. If there is congenital optic nerve blindness, the case is one of idiocy and not cretinism.

Having excluded the above, the next thing to consider is whether there is any congenital malformation of the brain or cord. Cases of *meningocele*, *myelocele*, or *spina bifida* will generally be obvious enough ; even *spina bifida occulta* will often suggest itself from the presence of a pigmented or hairy mole over the lower part of the lumbar spinal region, and the diagnosis may be confirmed by careful palpation there. Congenital *hydrocephalus* makes itself evident from the characteristic enlargement of the head, which in extreme cases can be mistaken for nothing else, and which in lesser degrees can be distinguished from the enlargement due to rickets or to congenital syphilis by the fact that it is more uniform, and that the bones are fragile and thin, and separated at the sutures. The only doubt that arises in a case of infantile hydrocephalus is as to whether it is truly congenital or was caused by an early but post-natal posterior basal meningitis. There is increasing evidence to show that the majority of these cases are not really congenital, that the head is not large at birth, and that the enlargement follows some febrile illness, with or without convulsions—really a meningococcal meningitis.

Congenital defect of the cortex would suggest itself as the cause of infantile diplegia in a case in which delivery had taken place without difficulty and without the use of forceps, the head and spine not being hydrocephalic or deformed, and yet the limbs being paralyzed from birth ; they may be flaccid or they may be spastic, and there is no constant type of reflex, though there is a tendency to increased knee-jerk, ankle-clonus, and extensor plantar reflex. Intelligence will very likely be defective at the same time. In rare cases the kidneys may be so large and cystic that they can be palpated, and the brain may then be cystic also—*porencephalus*.

Paraplegia due to *injury at birth*, either by the use of forceps or by excessive compression in a contracted maternal pelvis, is a diagnosis that can only be made when there has been an unusual amount of difficulty at birth, for it is remarkable to what extreme degrees the child's head may be squeezed and altered in shape without apparent detriment. Seeing that it is the lateral aspects, especially the arm areas of the Rolandic cortex, that will be most affected by forceps, these instruments are more likely to cause bilateral arm paralysis than ordinary paraplegia. Just the converse of this is true of *superior longitudinal sinus thrombosis*, for the leg areas of the brain lie close up against either side of this sinus, whilst the arm areas, being more distant from it, are likely to be less affected. The symptoms of superior longitudinal sinus thrombosis, of acute encephalitis, and of meningitis, may be so similar—pyrexia, general head symptoms, vomiting, and convulsions—that it is almost a matter of guess-work to choose between them when they are actually in progress ; if death occurs in a few days, suppurative meningitis is likely ; if in a few weeks, tuberculous meningitis ; if the patient lingers for many weeks before dying, or if recovery occurs with hydrocephalus, posterior basal or cerebrospinal meningitis ; if recovery occurs without hydrocephalus, it may be almost impossible to decide between meningococcal meningitis, acute encephalitis, and superior longitudinal sinus thrombosis ; nor is the distinction material, except in so far that it is important to remember always that a favourable issue may occur even in a case that seems to be hopelessly comatose and dying—a diagnosis of meningitis may have been made erroneously when the condition was really one of acute encephalitis only. If it seems to be of great importance to arrive at the accurate diagnosis in the acute stages, it may be justifiable to perform lumbar puncture. A cytological examination of the cerebrospinal fluid may show many polymorphonuclear cells in a suppurative case, or many lymphocytes in a tuberculous case. Of more value than the cytological examination, however, is the bacteriological test, which may succeed in isolating the causal organism.

If one is able to exclude the infantile diplegias, and the conditions which simulate them, the probability is that the patient will have shown obvious signs of being able to use the legs, or may even have been able to walk before the paraplegia set in. In that case, if the paralysis is of the upper neuron type, with spasticity, no wasting except such as may be due to disuse and non-development, no reaction of degeneration, increased knee-jerks, extensor plantar reflexes, ankle-clonus, and probably bladder and rectal trouble—especially if there is anæsthesia in the legs at the same time—by far the commonest cause for the condition is *spinal caries* with compression of the cord.

If, on the other hand, the patient develops a lower neuron type of paralysis, with wasting of the affected muscles and reaction of degeneration, the chances will be greatly in favour of *acute anterior poliomyelitis* followed by infantile paralysis, particularly if diphtheria can be excluded, and if a clear history can be obtained that the child was perfectly well until he developed an obscure febrile complaint, which may at first have been regarded as of gastric origin, but which, in a day or two, led to one or more limbs, possibly all four, becoming limp and paralyzed, with rapid subsidence of the fever and great improvement in the paralysis during the next few weeks. It is possible for a child to have had absolute paralysis of all four limbs from acute anterior poliomyelitis, and yet for complete recovery to occur ; more often, however, one or another group of muscles remains weak ; in a typical case, the extensors of the toes and ankle are affected permanently, the consequent contraction of the unparalyzed calf muscles leading to talipes equinus or equino-varus. Weakness of other groups of calf muscles leads in a similar way to other forms of club-foot, such as T. calcaneus, T. valgus, and so on. In other cases, the muscles below the knee recover completely, but some other group is involved—the quadriceps extensor femoris for instance, or the adductors of the thigh. It is of course possible for the legs to recover completely, whilst paresis of some group of muscles in the shoulder, arm, or forearm persists. The infantile paralysis which follows acute anterior poliomyelitis is nearly always asymmetrical, but it is by no means necessarily so, and it may cause persistent partial paraplegia. It is important to remember that the knee-jerk is deficient or absent only when the quadriceps extensor femoris muscle is affected ; and also that reaction of degeneration is no longer obtainable in the muscles when the disease is of sufficiently long standing for all the degenerate fibres to have become fibrous, by which time the only muscle and nerve fibres that remain are normal, though they are fewer in number than they should be.

Peripheral neuritis in a child is decidedly uncommon, except as the result of *diphtheria* ; it should not be diagnosed lightly. Being an affection of the lower neuron type, with wasting of the muscles, flaccidity, reaction of degeneration, and deficiency in the tendon reflexes, it may be difficult to distinguish between it and acute anterior poliomyelitis. It might be urged that the occurrence of pain or other sensory symptoms is in favour of peripheral neuritis and against poliomyelitis, but this is not really the case ; the inflammation in poliomyelitis is by no means necessarily restricted to the grey matter of the anterior cornua, and the acute stage of the disease is often accompanied by severe pains referred to the peripheral parts. There may, however, be bacteriological or other evidence of the patient's having had diphtheria during the preceding few weeks, in which case peripheral neuritis would be diagnosed ; if there is paresis of the soft palate, as evidenced by the regurgitation of fluids through the nose when the patient tries to swallow them, or by the nasal character of the voice, diphtheritic neuritis would be very probable.

There remains for discussion the following causes of paraplegia in children : Friedreich's ataxy, Tooth's peroneal type of progressive muscular atrophy, and the primary muscular dystrophies, particularly pseudo-hypertrophic muscular paralysis. There are two points common to all these, namely, that they all are insidious in onset, slowly progressive for years before the end comes as the result of an intercurrent malady ; and that they are familial diseases, the family history having an important bearing upon their diagnosis.

Friedreich's ataxy is characterized by paraplegia, often associated with deformity, such as talipes and scoliosis, owing to persistent error of posture, without wasting except such as may be due to disuse or non-development ; the knee-jerks are absent, but the pupils react normally ; there is no sphincter trouble unless quite late ; ankle-clonus is absent, but there is generally a remarkable condition of hallux erectus, which amounts to a sort of permanent extensor plantar reflex ; there are no sensory disturbances ; the arms may not be affected at all, or they may present some degree of ataxy, with or without intention tremors—that is to say, tremors which are increased when the patient tries to perform voluntary movements—sometimes even choreiform movements are present; speech is monotonous, nystagmus is sometimes present, and occasionally there is optic atrophy. If progressive paraplegia develops at about 8 or 9 years of age in a child with a family history of similar trouble; if the knee-jerks are absent, whilst the big toes are permanently erect, and if there is neither atrophy nor pseudo-hypertrophy of the muscles, the diagnosis is in all probability Friedreich's ataxy. The patient may survive to puberty or even longer, but is liable to death from phthisis, pneumonia, or other intercurrent malady—the same applying to all the familial diseases now under discussion.

Tooth's peroneal type of progressive muscular atrophy is apt to develop after some simple specific fever, such as whooping-cough or measles. The first point the mother notices is that the child—hitherto normal—is unable to bend the big toes upwards; a condition of permanent plantar flexion of the big toes ensues; inability to extend the other toes follows; and presently the patient cannot dorsiflex the ankles. It is chiefly the muscles supplied by the external popliteal nerve, formerly called the *peroneal* nerve, that are affected; hence the name of the disease. Talipes may result. The lesion is not primarily in the muscles, but in the anterior cornual cells of the lumbo-sacral part of the cord, so that reaction of degeneration is obtainable in the wasted muscles. The knee-jerks remain normal so long as the quadriceps extensor femoris is unaffected, there is no ankle-clonus, and the big toe may not move at all when the sole is stimulated. A brother or a sister is very likely to have suffered from the same complaint (see *Figs.* 20, 21, p. 60).

In the *primary muscular dystrophies* the nerves are normal, so that there is no reaction of degeneration; if a muscle has become entirely atrophied, there will be no reaction in it at all; but as long as any reaction is obtainable it is of the normal type. The same applies to the reflexes. The most easily recognized of all the primary muscular dystrophies is pseudo-hypertrophic muscular paralysis, the only difficulty being when no family history is obtainable, and when the case is still in too early a stage to be typical. Boys are affected more often than girls, but it is generally inherited from the mother's side. It is possible for some members to have presented atrophic myopathy, whilst others suffer from the pseudo-hypertrophic form. When fully developed, the most striking feature of the case is the marked weakness of the legs, notwithstanding the apparent firmness and great size of the calves. The muscles are really atrophied, their apparent enlargement being due to extensive deposition of intramuscular interstitial fat. Ultimately, if the patient survives, all the muscles in the body become wasted and fibrous; but whereas some of them atrophy from the first, others exhibit marked pseudo-hypertrophy before they atrophy—particularly the gastrocnemii, the solei, the glutei, the deltoids, the supra- and infra-spinati, and portions of the triceps. The muscles of the hands and feet are generally unaffected. The muscles most frequently atrophied are the lower half of the pectoralis major, the latissimus dorsi, the serratus magnus, the biceps, and the flexors of the knee. There are no sensory or sphincter troubles. When the case is well advanced, the way in which the patient gets up from a lying posture is very characteristic; it is generally described as 'climbing up himself.' He first rolls over and rests on his hands and knees; then puts his head between his arms and raises the knees from the ground, so that he is now supported on his hands and feet; he next brings one hand nearer to his toes, and then, swinging his body over first to one side, places his opposite hand on the corresponding knee, straightens that leg, and repeats the performance on the other side, so that he now stands with his legs widely separated and with a hand resting on each knee; he then works each hand alternately higher up his thighs, until finally, by a sudden backward movement of his shoulders, he attains the erect attitude. Another feature of the case is, that if one tries to lift the boy up by putting one's hands under his armpits, his shoulders rise right up to his ears, and he very easily slips through one's hands. He is also unable to stand on tip-toe, and the gait is waddling.

The two other types of muscular dystrophy mentioned above—the infantile and the juvenile—are but different varieties of the same malady; both are characterized by progressive wasting of the muscles without pseudo-hypertrophy; in the infantile form the muscles have been atrophic from the first, whereas in the juvenile form the muscles develop in what seems to be a normal way up to a certain point, and then gradually waste away. The disease is distinguished from peripheral neuritis, (1) by the absence of reaction of degeneration; (2) by the persistence of the reflexes as long as any muscle tissue is left to respond; (3) by the family history as mentioned above; (4) by the absence of sensory changes; and (5) by the absence of improvement with time. An attempt is sometimes made to classify the primary muscular dystrophies into different kinds, according to the groups of muscles first affected. In the Landouzy-Dejerine type, for instance, the face muscles are first attacked, the trouble slowly spreading to the shoulder and upper arm. It is probable, however, that whatever groups of muscles may be the first affected, the differences are those of degree and type rather than of kind, and that the muscular wasting, wherever it may begin, ultimately becomes widespread, and finally involves all the muscles.

II. THE CAUSES OF PARAPLEGIA IN ADULTS.

We may now pass on to a discussion of the differential diagnosis of paraplegia in adults. It is clear that a paraplegia that has arisen during infancy or childhood may persist into adult life, in which case the diagnosis will be made upon the lines indicated above. It is also possible for some of the causes of paraplegia that usually affect young patients not to do so until they have grown up. The chief causes, however, for paraplegia arising for the first time in adult life are as follows:—

(*A*). *Causes of the lower neuron type of paraplegia:—*

1. Peripheral neuritis, which may be due to various different causes (p. 63)
2. Anterior poliomyelitis
3. A pelvic tumour interfering with the lumbo-sacral plexus
4. A tumour affecting the cauda equina
5. Compression of the lumbar enlargement of the cord.

(*B*). *Causes of the upper neuron type of paraplegia:—*

1. Transverse myelitis.
    (*a*). Primary
    (*b*). Due to compression by: (i) Spinal caries; (ii) New growth in the vertebræ or meninges; (iii) Injury; (iv) Aortic aneurysm
2. Disseminated sclerosis
3. Amyotrophic lateral sclerosis
4. Primary lateral sclerosis
5. Ataxic paraplegia
6. Combined scleroses of the cord
7. Syringomyelia
8. Meningitis
9. Hæmorrhage into the cord
10. Cerebellar tumour or abscess
11. Bilateral cerebral softening or hæmorrhage.

(*C*). *Causes not conforming either to the lower or to the upper neuron type:—*

1. Tabes dorsalis
2. General paralysis of the insane
3. Landry's paralysis
4. Functional paraplegia
5. Malingering.

The first points which call for attention in making a diagnosis are the history and progress of the case. In only a few of the above conditions is the onset sudden; these are certain cases of acute anterior poliomyelitis, transverse myelitis, meningeal hæmorrhage, Landry's paralysis, functional paraplegia, and malingering. If the paraplegia is of sudden onset, of the upper neuron type, and not the result of injury, it is almost certainly due to some form of transverse myelitis. The great majority of cases of paraplegia, however, have an onset that is not absolutely acute, and generally it is quite gradual.

There are certain conditions that can, as a rule, be either diagnosed or excluded at once. If the patient has Argyll Robertson pupils and no knee-jerks, *tabes dorsalis* can be diagnosed at once. It is necessary to remember, however, that the pupil may react neither to light nor to accommodation in some cases of peripheral neuritis, so that, if care be not exercised, the reaction may be mistaken for the Argyll Robertson type of tabes, the latter being diagnosed when peripheral neuritis is the lesion really present. The converse mistake is also possible, especially if the actual strength of the leg muscles be not tested; in both conditions there may be patches of impaired sensation, but in peripheral neuritis with absent knee-jerks, there are absolute wasting, loss of power, and reaction of degeneration, whilst in tabes there is no trophic wasting, and often no great loss of power in individual muscles, though there is apparent weakness owing to the action of opposing muscles being inco-ordinate, and there is no reaction of degeneration.

Another difficulty in connection with tabes dorsalis arises in anomalous cases in which either the pupil reaction has not yet become typical, or else the knee-jerks are not yet gone. If the reaction of the pupil is of the Argyll Robertson type, tabes may sometimes be diagnosed even in the presence of knee-jerks, if there is an obvious history of other concomitants of the disease, such as lightning pains, gastric crises, or any of the rarer crises—laryngeal, rectal, urethral, vesical, renal, general abdominal, or sweating—marked ataxy, history of syphilis, perforating ulcer of the foot, a Charcot's joint, or bladder or rectal trouble, particularly if the patient be a male who has had much brain wear. The tendo Achillis jerks may disappear before the knee-jerks do, and they should be carefully tested. There is also in many cases a remarkable deficiency or even complete absence of deep tenderness in such organs as the testis, tongue, larynx, or mamma. In certain cases the knee-jerk may still be obtainable on one side after it has been lost upon the other, so that both should always be tested and not one only.

If the paraplegia is obviously of the lower neuron type, with deficiency or absence of the superficial and deep reflexes, atrophy of the muscles, and reaction of degeneration, with or without paræsthesia, the probabilities are that it is due to one of the many different causes of *peripheral neuritis* that are discussed on page 63. If the onset has been sudden, however, and if the paralysis began to clear up again rapidly, except possibly in one group of muscles in one leg, there would necessarily be a suspicion of acute *anterior poliomyelitis* which occurs occasionally in adults.

It is important in all cases of suspected peripheral neuritis to make a rectal examination, lest there should be some pelvic mass, malignant or otherwise, interfering with the lumbo-sacral plexus. Peripheral neuritis may also be simulated closely by either a tumour or a gumma interfering with the cauda equina, an uncommon condition that suggests itself if there is severe pain referred to the lower part of the spinal column behind, or if the paraplegia comes on in such a way as to affect one leg before the other, the pelvis being found free from growth. It is also important to remember that transverse myelitis due to lesions which, if they are situated a little higher up in the cord, cause a paraplegia of the upper neuron type, produces wasting, reaction of degeneration, and loss of reflexes when they affect the cord at the level of the lumbar enlargement.

When the paraplegia is definitely of the upper neuron type, with spasticity of the legs without wasting, with increased knee-jerks, extensor plantar reflexes, ankle-clonus, and perhaps retention of urine with overflow, and incontinence of fæces, the first step in arriving at the diagnosis is to determine if there is any sensory disturbance at the same time. The only diseases mentioned under heading *B*, that produce obvious sensory disorders, are transverse myelitis, syringomyelia, hæmorrhage into the cord, and very rarely meningitis or bilateral cerebral softening. The latter can only be diagnosed when there has been an apoplectic seizure associated with hemiplegia, followed after an interval by another cerebral seizure which, by producing hemiplegia of the opposite side to the one first involved, results in paraplegia, or rather diplegia. The arms and face are likely to be affected as well as the legs, and there will be either a history of syphilis to account for endarteritis and thrombosis in a young male, or a bruit, a history of acute rheumatism, or other evidence of a heart lesion, to account for embolism ; or senile changes, with or without albuminuria, a high blood-pressure, retinitis, and other signs of renal and arterial degeneration, to account for hæmorrhage.

Hæmorrhage into the cord is hardly ever spontaneous ; it may follow an injury, such as a bullet wound or a stab in the back, and then the history will indicate the diagnosis. *Acute meningitis*, whether tuberculous, suppurative, posterior basal, or cerebrospinal, seldom causes complete paraplegia until a late stage of the illness is reached, by which time the nature of the malady will generally be indicated by the cerebral symptoms, particularly headache, vomiting, convulsions, strabismus, and ophthalmoscopic changes such as optic neuritis or choroidal tubercles. Bacteriological investigations of the fluid obtained by lumbar puncture may assist the diagnosis materially. There is a chronic form of meningitis, however, of which the diagnosis is not so easy, and that is the *chronic hypertrophic hæmorrhagic pachymeningitis* which affects chiefly the vertex and the cervical portion of the cord. The condition is generally caused by chronic alcoholism in syphilitic subjects, especially if there has also been some injury; the diagnosis is difficult, but it may be suggested by the history, and by the degree of pain referred to the nerves that are involved in the meningeal thickening—the chief difficulty being to exclude spinal caries in cases involving the cord. Wassermann's reaction may be positive if no antisyphilitic remedies have been employed. In *syringomyelia*—a very slowly progressive disease that is by no means always associated with paraplegia, the nature of the symptoms depends upon the degree to which the central canal of the cord and the gelatinous substance around it are affected, and also upon the level in the cord at which the changes occur. The diagnostic symptom is that, in some region or another, the skin will be found to have lost its power of distinguishing heat from cold and pain from touch, though it still retains ordinary cutaneous sensibility. It is apt to give rise to skin lesions in the paræsthetic parts (Morvan's disease), and also to acute painless swelling of the joints, with deformity from destruction of the ends of the bones—Charcot's joints—precisely similar to those that may occur in tabes dorsalis.

If the patient has marked impairment of all kinds of sensation in both legs, with paraplegia of the upper neuron type, and no paralysis of the arms, the lesion is almost certainly *transverse myelitis* of some kind. The absence of sensory disturbance, however, does not exclude transverse myelitis, for when the conductivity of the spinal cord is interfered with, without being entirely inhibited, the sensory columns are able to transmit impulses longer than the pyramidal tracts, so that paralysis appears before anæsthesia. The same applies to a transverse myelitis that is getting better, the patient recovering sensation in his legs before he is able to move them. The chief difficulty will be to determine the nature of the transverse myelitis. There are two main types: (1) That due to causes outside the cord compressing it—especially *spinal caries*, secondary *growth*, the effects of such *injuries* as fractures of the spine, bullet wounds and stabs, or more rarely erosion of the bones by an *aortic aneurysm*; and (2) That due to softening from thrombosis of a spinal artery, the result of *syphilis*, or a fever such as *enteric* or *scarlet*. One of the first points to attend to is the presence or absence of pain. Lesions, such as thrombosis, which affect the cord but not its posterior nerve roots, are painless, whereas swellings which compress the cord from without almost always produce pain, sometimes a typical girdle pain, on account of their irritating the posterior nerve roots. If, therefore, there is or has been any pain in the back other than what may be due to a known injury, it is probable that transverse myelitis is not primary but due to compression. If the spine presents an obvious Pott's curvature, or if the patient has other evidence of peripheral tuberculosis, such as enlarged or caseating glands in the neck, hip, knee, or other joint disease, a psoas abscess, lupus vulgaris, and so on, especially in a young person who has been in the habit of drinking much milk, compression by spinal caries is fairly certain. The main difficulty arises when the cord becomes compressed without deformity of the spinal column, and with no other tuberculous lesion apparent. Local tenderness over one or more vertebral spines will help to suggest the diagnosis, especially if local pain is complained of in the same region, and if the pain is increased by any jarring of the spine. Growth is fortunately much rarer, and it is to be excluded by a routine examination of all the viscera, most cases of spinal new growth being secondary to a neoplasm elsewhere, especially of the breast; primary growths of the spine are so rare that they are generally taken for caries at first, and the correct diagnosis is not always arrived at before post-mortem microscopical examination has been made. Aortic aneurysm is a still rarer cause of compression myelitis; if there is a distinct pulsatile tumour along the course of the aorta, the nature of the case may be obvious; more often, however, an aneurysm which erodes the vertebræ sufficiently to bulge into the spinal canal, does not at the same time enlarge forward to produce a tumour that can be recognized easily by palpation. The patient will generally be a man in the prime of life who has had syphilis, who is not a life abstainer, and who has worked hard; Wassermann's reaction may be positive. Apart from a pulsatile tumour the symptoms will be very like those of paraplegia from spinal caries.

The relationship of injury to transverse myelitis is not always quite straightforward. If, for example, a patient who has syphilitic endarteritis of his spinal vessels receives a kick in the back from a horse, he may find that, by next day, he is unable to move his legs; it may at first seem obvious that the kick has been the sole cause of the paraplegia, when the real cause is syphilis—the kick having been the final factor which led to thrombosis in a diseased spinal artery. Transverse myelitis due to syphilis is exactly comparable in its mode of origin to the hemiplegia which results from endarteritis obliterans in a middle cerebral artery. There is no pain and no deformity of the spine, but in other respects the paraplegia presents the same features as does that which is due to compression of the cord. Syphilis is by far the most important cause of this primary transverse softening, but there are a considerable number of other maladies in which a similar result ensues occasionally; almost any infective disease may lead to it; one may perhaps mention typhoid fever, scarlet fever, and influenza in particular. In infective endocarditis there may be an additional factor, namely embolism of the cord, though this is decidedly rare.

If it is found that the arms are affected as well as the legs, it is unlikely that the lesion is transverse myelitis, unless in rare and anomalous cases such as those mentioned on page 62. If the onset has been slow, the course progressive, and wasting is present, with reaction of degeneration in the muscles of the hands or arms, with increased knee-jerks, ankle-clonus, and extensor plantar reflexes, but no anæsthesia, the malady is almost certainly *amyotrophic lateral sclerosis.*

If there are increased knee-jerks, extensor plantar reflexes, ankle-clonus, ataxy, intention tremors in the hands, nystagmus, and a hesitancy in the voice, which may even be of the type described as 'scanning,' the disease is either *cerebellar abscess or tumour,* or *disseminated sclerosis.* If headache and vomiting have been severe, the former is the more probable, and the diagnosis may be clinched by finding double optic neuritis. Abscess will be more likely than tumour if there is otorrhœa or pyrexia. It is not uncommon to find optic atrophy, with either concentric diminution in the fields of vision or else a central scotoma, in disseminated sclerosis, but optic neuritis is uncommon. The difficulty in diagnosing disseminated sclerosis arises mainly when the complaint is in its early stages; the patches of sclerosis may be anywhere in the cord, and before the affected fibres atrophy there is a period when they are sometimes able to conduct impulses, sometimes not; when they are not able to conduct, there are numerous symptoms, and in a day or two, when conducting power recovers, these symptoms are gone again; this variation from day to day nearly always leads to a diagnosis of neurosis for months or years before the true nature of the malady becomes obvious. In some patients a central scotoma may develop early, leading to peculiar symptoms, such as the inability to distinguish a sovereign from a shilling if the light is not good, or the liability to run into people without seeing them when cycling. If ataxy is marked, the staggering gait may lead to a suspicion of alcoholism; the patient staggers alternately to either side in disseminated sclerosis, whereas in tumours of one cerebellar hemisphere the tendency is to stagger constantly to the same side. Bladder and rectal troubles are not common in either case, and yet they may be prominent. Paræsthesia may also develop in disseminated sclerosis, although as a rule there is no sensory disturbance at all.

If a patient has the symptoms of spastic paraplegia and ataxy, without anæsthesia, nystagmus, or changes in the voice, a diagnosis of *ataxic paraplegia* will usually be made. There is really no difference between this and what has been called *combined scleroses of the cord*; in both conditions there is degeneration of the posterior columns, the crossed pyramidal, and the cerebellar tracts. Some observers use the term combined scleroses only for syphilitic cases, reserving ataxic paraplegia for similar non-syphilitic cases.

Primary lateral sclerosis was a relatively common diagnosis until it was found that the more careful the examination the greater was the likelihood that more than simple degeneration of the crossed pyramidal tracts would be found. Partial compression of the cord produces spastic paraplegia without anæsthesia, and thus simulates primary lateral sclerosis as described above. Disseminated sclerosis may do so likewise, and so on. Primary lateral sclerosis should never be diagnosed, therefore, till all the other affections in which the lateral columns may be affected have been excluded. There is such a disease as primary lateral sclerosis, however; it is generally syphilitic in origin, and it leads to typical spastic paresis of the legs, with increased knee-jerks, ankle-clonus, extensor plantar reflexes, no wasting, no R. D., no sensory disturbances, and in the later stages, retention of urine with overflow, and incontinence of fæces; the disease is generally progressive, but after reaching a certain point it may remain stationary for years, or even improve to a slight extent for a time. When lateral sclerosis is yet in an early stage, a valuable sign of it is the disappearance of the abdominal reflexes; the diagrams (*Figs.* 210, 211) may be of assistance in locating the level of the cord at which a lesion may be present.

The causes of paraplegia that remain for discussion are Landry's paralysis, general paralysis of the insane, functional paraplegia, and malingering.

Landry's paralysis is probably not a distinct entity, but rather a very acute type of perhaps more than one variety of paraplegia. It is rare. It affects young adults, who, hitherto strong and well, become rapidly affected by paralysis which starts in the legs and quickly ascends to the trunk and arms, and may even involve the neck and cranial nerves. It either gets well quite rapidly, or else kills the patients in a few hours or days by affecting the intercostal muscles and diaphragm, with consequent asphyxia. There may be slight pains in the affected parts shortly before paralysis sets in, but sensory symptoms are generally slight, or absent. The nature of the malady is obscure, but if one were to regard it as a very acute and widespread anterior poliomyelitis, one could account both for its main symptoms, its rapid fatality in some cases, and its equally rapid recovery in others. Moreover, seeing that the patient either dies or recovers so quickly, it is not surprising that there is no time for the development of obvious muscular wasting or reaction of degeneration.

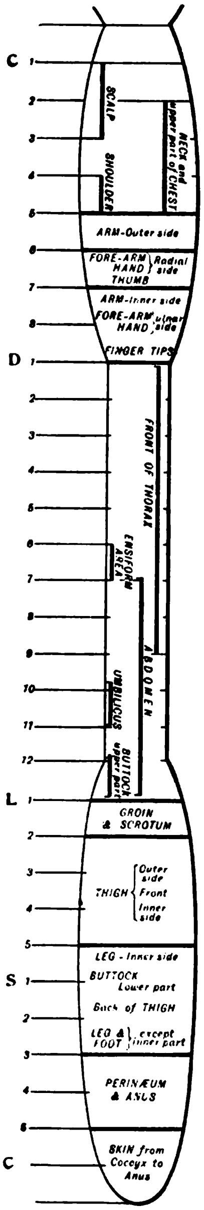


Fig. 210.—Diagram of sensory localization in the spinal cord.

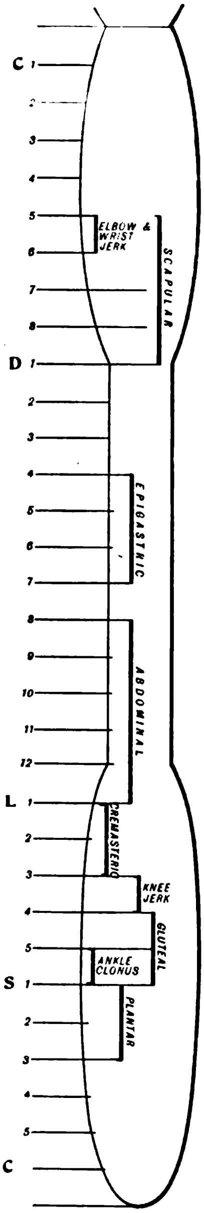


Fig. 211.—Diagram of localization of reflex centres in the spinal cord.

Paraplegia in cases of ***general paralysis of the insane*** does not arise until the third stage of that malady is reached; by that time the diagnosis is generally obvious; the paraplegia is part of a general and extreme weakness, and the patient is bedridden.

Functional paraplegia and ***malingering*** should never be diagnosed until all organic causes—particularly disseminated sclerosis and spinal caries—have been excluded. Malingering may be suggested by the particular circumstances of the case—the patient may be a nervous, self-conscious girl who desires to attract sympathy, or an out-of-work who wants to get a night's shelter in a hospital; careful observation generally leads to the detection of the fraud. Functional paraplegia is less easy to be sure of, and in many patients that which may at first be regarded as functional ultimately turns out to be organic; this is especially true in the case of disseminated sclerosis. The paraplegia is never of the primary muscular, or the lower neuron type, there being no wasting and no R.D. The muscles remain of good bulk as they do in the upper neuron type of paraplegia, but although the knee-jerks may be unduly brisk, the plantar reflexes remain flexor, and there is no maintained ankle-clonus. If there is anæsthesia, the distribution of the latter is sometimes obviously functional; it may, for instance, start sharply at the knee and cease suddenly at the ankle, or in some other way indicate that it corresponds neither to the segments of the spinal cord nor to the distribution of the peripheral nerves. It is by anomalies of this kind, which make it impossible to fit in the case with any organic lesion, that functional paraplegia is diagnosed by a process of exclusion. *Herbert French.*

PARASITES, INTESTINAL.—*Tape-worms.*—The commonest symptom of the existence of a tape-worm is the passage of the detached terminal segments per rectum in longer or shorter tape-like strips. The only condition for which these might be mistaken is muco-membranous colitis, in which long, narrow, white mucous casts of the bowel may be passed with the motions (*Fig.* 172, p. 398). It is easy to distinguish these, however, if the suspected material is floated in water, for in the case of a cast of the bowel a central lumen will be found which is not present in the tape-worm. There is, moreover, no regular segmentation in the case of muco-membranous colitis, whereas tape-worms are obviously segmented. If any doubt remains, examination with a lens will show the glandular structure of the uterus in the tape-worm segments, and no such structure in the strips of mucus in muco-membranous colitis. It is sometimes stated that picking of the nose and a voracious appetite are symptoms of the presence of some kind of intestinal parasite; but this is hardly ever the case; if constitutional symptoms develop at all, they take the form of deficiency of appetite, with more or less anæmia, which may become profound; there is often considerable Eosinophilia (p. 218). The three forms of tape-worm that occur in the human intestine are *Tœnia solium*, *T. mediocanellata*, and *Bothriocephalus latus*, the commonest in Great Britain being the *T. mediocanellata*, the cystic stage of which is spent in cattle. *T. solium* is derived chiefly from pig-meat, whilst *Bothriocephalus latus* occurs mainly in those who live much on fresh-water fish. It may be possible to make the diagnosis of *T. mediocanellata* by holding the segments up against a bright light and seeing a median streak or water-channel, in addition to one down either edge of each strip, this middle water-channel giving the name to the parasite. The ultimate proof of the nature of the tape-worm, however, is afforded by the characters of the head, that of *T. solium* having four sucking discs, with a rostrellum surrounded by thirty-four hooklets (*Figs.* 212, 213); that of *T. mediocanellata* four circular sucking discs and no hooklets (*Fig.* 214); whilst that of the *Bothriocephalus latus* has a more or less conical head, with two elongated lateral sucking discs and no hooklets (*Fig.* 215). The degree of anæmia, chlorotic in type, is usually greatest with *Bothriocephalus latus*, least with *T. mediocanellata*, and the same also applies to the degree of eosinophilia. The eggs of the tape-worm are unmistakable (*Fig.* 216); they are spherical, with a dark-brown central portion, and a lighter striated broad capsule.

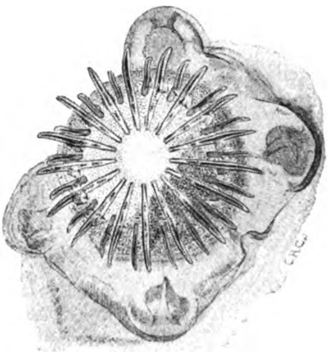


Fig. 212.—Head of *Tænia solium*: showing four sucking discs and thirty-four hooklets, alternately long and short. (Medium power.) (*From a specimen in the possession of Mr. Pellischer, New Bond Street, W.*)

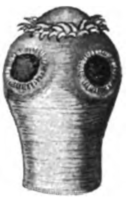


Fig. 213.—Head of *Tænia solium*, semi-diagrammatic. (Low power.) (From French's *Medical Laboratory Methods*.)

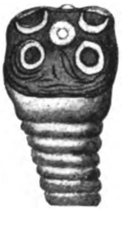


Fig. 214.—Head of *Tænia mediocanellata*. (Low power.) (From French's *Medical Laboratory Methods*.)

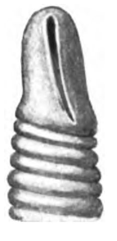


Fig. 215.—Lateral view of head of *Bothriocephalus latus*, showing longitudinal sucking disc. (Low power.)

Microscopical Examination of Fæces.—One of the best ways of preparing fæces for microscopical examination for the ova of parasites or for other solid particles, is to put about as much as would cover a shilling into a test-tube, filling the latter two-thirds full of normal saline solution (1¼ dr. of salt to a pint of water), corking the tube, and shaking it vigorously in order to break up the fæces as much as possible; on allowing to stand for twenty minutes, the upper part of the fluid remains opaque with fine débris, whilst the heavier particles, including the ova of parasites, have sunk to the bottom; the supernatant opalescent or opaque fluid may now be poured off, and the more definite residue again shaken up with normal saline and allowed to stand for another twenty minutes; this process is repeated until the supernatant fluid becomes clear after it has stood for the twenty minutes, and then, when as much of the fluid as possible has been poured away, a drop of the sediment is taken up in a pipette, transferred to a microscope slide, covered, the excess of fluid removed with filter paper, and the specimen examined either with the ⅔ in. or ⅙ in. objective, preferably with the mechanical stage. Such a specimen exhibits all sorts of vegetable cells, keratin particles, and so forth, which may at first be regarded as ova, but when the actual ovum of an intestinal parasite is seen, there is seldom any doubt about it.

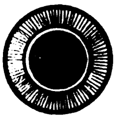


Fig. 216.—Ovum of *Tænia solium*, semi-diagrammatic. (High power.) (From French's *Medical Laboratory Methods.*)

Round-worms.—The only round-worm that occurs in Great Britain is the *Ascaris lumbricoides.* This parasite may or may not give rise to symptoms; if it does so, they take the form of slight and obscure nervous and gastro-intestinal disorders. More often the diagnosis is quite unsuspected until one of the worms is found in the bed, having crawled out per anum, especially when the patient, generally a child, falls ill of some febrile malady. If round-worms have been found previously, and if the existence of others is suspected, the diagnosis may be confirmed by discovering the typical ova (*Fig.* 217) in the fæces; their chief characters are their relatively large size, oval shape, and irregular membranous envelope outside the chitinous shell. This worm does not produce eosinophilia as a rule, but in exceptional cases it may do so.

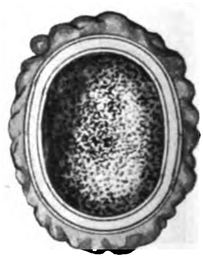


Fig. 217.—Ovum of *Ascaris lumbricoides.* (High power.) (From French's *Medical Laboratory Methods.*)

Thread-worms.—*Oxyuris vermicularis*, if present at all, usually occurs in hundreds, and can be detected immediately by examination of the fæces with the naked eye. Each parasite is rather more than ¼ in. in length, without any colour; its extremities project from the fæcal mass, and move about slowly, like threads waving in the air. These parasites produce no eosinophilia. The patients are nearly always children, and there may be no symptoms at all; but more often there is considerable irritation around the anus, and in young girls about the vulva. Gonorrhœa has before now been suspected when the vulvar infection was really due to the *Oxyuris vermicularis.*

The *Whip-worm* (*Trichocephalus dispar*) is in itself an entirely unimportant parasite occurring in the cæcum and large intestine (*Fig.* 218), and producing no symptoms whatever. The worm with its tail is about 1½ in. in length, and it is often coiled up watch-spring-wise. Its appearances are unmistakable; its ovum (*Fig.* 219) looks more or less like a running-cork, and, with its deep brown central parts and clear ends, it is quite characteristic. Whip-worms are present to the extent of nearly 10 per cent of all the inhabitants of some cities. They produce no eosinophilia, blood-changes, or symptoms.

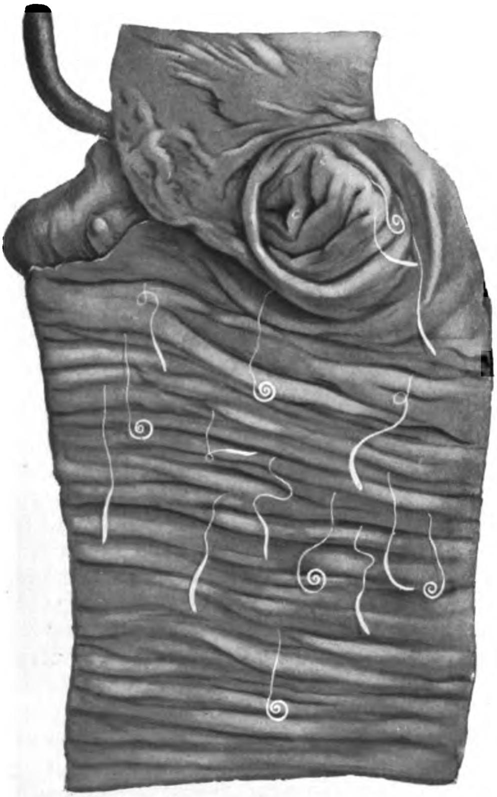


Fig. 218.—Whip-worms (*Trichocephalus dispar*) in the cæcum and ascending colon. (*From a specimen in the Gordon Museum, Guy's Hospital.*)

The *Hook-worm* (*Ankylostomum duodenale*) (*Fig.* 220).—This is not a general parasite in Great Britain, but has affected many persons in certain districts as the result of introduction from abroad, particularly amongst lead-miners in Cornwall. Outbreaks also occurred in the workers in the St. Gothard tunnel, and the disease is prevalent in many parts abroad, especially in India, Egypt, Brazil, and Jamaica. The infection is carried from fæces to soil, from the soil to the hands, thence to the mouth, and so to the alimentary canal. The symptoms are for the most part those of progressive anæmia and asthenia, inability to continue with work, œdema of the lower extremities, anasarca, shortness of breath, and the occurrence of boil-like skin eruptions, described popularly as the 'flowers' of the disease. The appearance of the patient may suggest pernicious anæmia, and the blood-count may sometimes seem to confirm this diagnosis at first; for whereas a great many of the patients have a severe chlorotic type of anæmia, some have a marked reduction of the red corpuscles and a slightly less reduction of the hæmoglobin, so that there is a high colour-index such as is characteristic of pernicious anæmia. There is generally no leucocytosis, but the differential leucocyte-count may suggest the diagnosis at once, for nearly all the patients present a considerable degree of eosinophilia. The administration of anthelmintics such as thymol may lead to the evacuation of the mature worms, which may be recognized in the fæces (*Fig.* 27, p. 79), each being from ⅓ to ⅔ in. in length. The ova (*Figs.* 28 and 29, p. 80) are oval, with a clear transparent shell and coiled-up embryo parasite. Melæna is another symptom which may be prominent in some of these cases.

Fig. 219.—Ovum of *Trichocephalus dispar.* (High power.) (From French's *Medical Laboratory Methods.*)

The two intestinal parasitic affections which produce the most serious anæmias and other toxic effects in man are *Ankylostomum duodenale* and *Bothriocephalus latus.* *Herbert French*

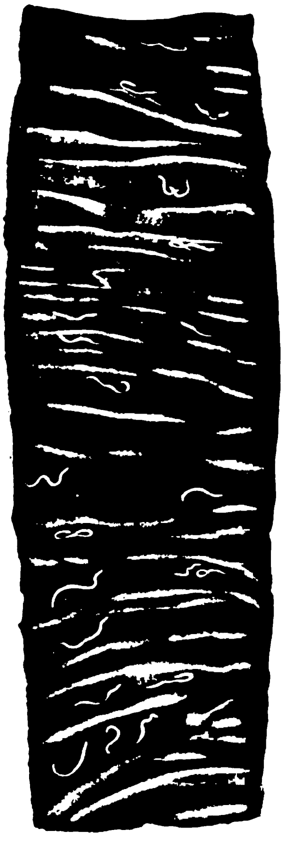


Fig. 220.—Ankylostomiasis of the duodenum. (*From a specimen in the Gordon Museum, Guy's Hospital.*)

PARESIS.—(See Paralysis, p. 491, et seq.)

PENIS, DISCHARGE FROM.—(See Discharge, Urethral, p. 181.)

PERISTALSIS, VISIBLE.—The importance of visible peristalsis lies in the fact that it is always pathological except in a few cases in which its unimportant nature is immediately obvious. The two chief conditions which render the normal movements of the bowels visible are *divarication of the abdominal recti muscles*, and *ventral herniation* of a laparotomy scar. The latter is obvious at once; the former is best detected when the recumbent patient, who is generally a multiparous woman with a soft flabby abdomen, tries to raise her head and thorax from the couch without the use of her arms; the contracting recti come together then, and close over the gap in the middle line in which, under the stretched and unsupported skin, the bowel movements had been seen. In almost all other circumstances visible peristalsis is pathological; it may then be divided into two types—gastric, and intestinal.

Gastric Peristalsis takes the form of a comparatively large swelling in the upper part of the abdomen, coming and going, generally appearing from under the region of the left ribs, progressing slowly downwards and to the right, where it fades away and disappears; it corresponds more or less with the greater curvature of the stomach. It is often stated that a return wave, passing along the lesser curvature from right to left, can also be made out, but this is exceptional. Sometimes, instead of progressing, it comes and goes almost in the same spot, varying in shape but scarcely in position. The exact site of the wave must depend mainly upon the size and position of the stomach. It indicates *pyloric* or *duodenal obstruction*, and its presence serves to exclude atonic gastrectasis. There may or may not be other signs of dilated stomach, particularly a widely distributed succussion splash, vomiting of large volumes of fermenting fluid at relatively long intervals, and a greatly increased bismuth *x*-ray shadow. Whether the pyloric stenosis is simple or malignant has to be decided upon other grounds.

Visible Intestinal Peristalsis is, with the limitations discussed above, one of the surest signs of grave intestinal obstruction. There are almost certain to be abdominal distention, vomiting, and constipation along with it, and the discussion of the differential diagnosis of the different causes of these symptoms will be found elsewhere. The great importance of visible peristalsis is seen in those doubtful or obscure cases in which the patient seems hardly ill enough to be suffering from intestinal obstruction. It may be thought that colic, the result of some indigestible article of diet, is a more likely diagnosis, and that a dose of castor oil will cure the malady. Rather than wait for increasing severity of the symptoms to clinch the diagnosis in these cases, it is most important to arrive at a diagnosis of the necessity for laparotomy at the earliest possible moment if life is to be saved. If the small intestine alone is involved, the waves are multiple, and they run more or less transversely across the abdomen—the ladder-rung type: when the colon is obstructed, vertical waves, especially in one or both flanks, are the chief form the peristalsis takes. Definite and visible intestinal peristalsis is, so far as any single sign can be relied on, an almost infallible indication of the need for laparotomy in any case in which the other symptoms and the history point to a possibility of intestinal obstruction.

Herbert French.

PERSPIRATION, ABNORMALITIES OF.—(See SWEATING, p. 654.)

PHOSPHATURIA.—This is a term the precise significance of which is by no means clear; the meaning it conveys to one observer is not always that which it implies to another. Some restrict it to conditions in which the total quantity of phosphates in each day's urine is greater than the average maximum. Others use the term when there is a spontaneous deposit of phosphates in the specimen glass. Others would include cases in which, on applying the boiling-test for albumin, a cloud of phosphates comes down. So loose is the application of the word phosphaturia that it is generally used whenever anything arises to remind the observer ocularly of the fact that the urine contains any phosphates at all.

What is really required is a series of *different* terms to express the following conditions:—

1. Circumstances in which a greater quantity of phosphates is habitually passed in the urine than is the average maximum in health.
2. The spontaneous deposition of phosphates in a urine that has stood in a specimen glass until cold.
3. The spontaneous deposition of phosphates in the bladder, so that the urine is thick and milk-like when it is being passed.
4. The deposition of phosphates as a white cloud when the urine is heated.

Absolute Phosphaturia.—The phosphoric acid in the urine is chiefly exogenous, i.e., derived from phosphates in the food. It is chiefly in inorganic combination as salts of the alkalies and alkaline earths. There is a certain small percentage of urinary phosphorus derived from the katabolism of nuclein and lecithin, but the amount derived from these in healthy persons is very slight as compared with that which comes direct from the food, so that the phosphates almost disappear from the urine during starvation. There are wide variations in the amounts excreted by normal persons; the average is 3·5 grams per diem, but the healthy limits are as far apart as 1 gram and 8 grams.

It has been asserted that persons whose business entails great wear and tear of the nervous system excrete more than the average amount of phosphates, and the same has generally been held to be true of sufferers from certain nervous disorders of the hysterical or neurasthenic type, particularly when sexual matters are in question. There is very little evidence, however, to show that there is any real increase in the urinary phosphates in these cases. There is often a very abundant deposit of phosphates on applying the heat test to the urine, and this may give the impression that the total quantity of phosphates present must be above the normal; but the impression has not been confirmed by exact analysis. There is only one well-defined condition in which there is absolutely and persistently more phosphate in the urine than healthy limits would allow, and that is *phosphatic diabetes*—a very rare condition, of which the main features are thirst, emaciation, aching in the loins and back, and polyuria without sugar but with an absolute excess of phosphates in the urine.

Physiology of Phosphatic Deposits.—In nearly every case the deposition of phosphates is a purely physiological process. A molecule of phosphoric acid, H_3PO_4, contains three hydrogen atoms. Each of these can be replaced separately by an atom of any monobasic metal, such as sodium. Three types of salts are formed, according as one, two, or three of the hydrogen atoms have been replaced, as in the following examples :—

NaH_2PO_4 - -	Sodium dihydric phosphate
Na_2HPO_4 - -	Sodium monohydric phosphate
Na_3PO_4 - -	Sodium phosphate.

These salts may all be present in the same urine, the proportions of each varying with the amount of phosphoric acid present, on the one hand, and the total amount of bases (i.e., sodium, potassium, etc.), and the total quantities of other acids present in the form of chlorides, sulphates, and so forth, on the other. The greater the quantity of chlorides and sulphates, the greater will be the amount of the metallic bases required to form them, and consequently the less will be the amount of bases left to combine with phosphoric acid ; the result must then be a relative excess of NaH_2PO_4. Conversely, the scantier the chlorides and sulphates, and the more abundant the bases, the greater will be the proportion of Na_2HPO_4 and Na_3PO_4.

Now the three sodium salts differ from one another in at least two physical respects—their action upon litmus, and their solubility in water. Sodium dihydrogen phosphate (NaH_2PO_4) turns blue litmus red—in other words, it is an acid phosphate. The acidity of ordinary urine is mainly due to it. Sodium monohydrogen phosphate (Na_2HPO_4) is also an acid salt technically speaking, and there are some colour tests which exhibit the acid reaction with it ; litmus, however, is not one of these, for Na_2HPO_4 turns red litmus blue. When a given urine contains more Na_2HPO_4 than NaH_2PO_4, the reaction of that urine to litmus is alkaline ; that is to say, it turns red litmus blue and does not turn blue litmus red. Some urines have what is known as an amphoteric reaction—they turn red litmus bluish and blue litmus reddish—a different thing from neutrality of reaction, in which neither red litmus nor blue is turned in colour at all. The cause of the amphoteric reaction of a urine is the even balance in that urine of the Na_2HPO_4 on the one hand and of the NaH_2PO_4 on the other.

Now the dihydrogen phosphate is much more soluble in water than is the monohydrogen phosphate, whilst the tribasic phosphates are as a rule far less soluble still. When it is stated, therefore, that phosphates are more soluble in acids than they are in alkalies, it must be remembered that it is not a question of a difference of solubilities of the *same* salt of phosphoric acid, but of an acid urine containing the bulk of its phosphates in a salt *different* from the one present in an alkaline urine. The very fact of a urine being alkaline means that there is relatively little of the more soluble NaH_2PO_4 present, and relatively much of the less soluble Na_2HPO_4 and Na_3PO_4. Conversely, the fact that a urine is acid implies that the phosphates are relatively more abundant in the soluble NaH_2PO_4 form than they are either as Na_2HPO_4 or Na_3PO_4. As a matter of fact, the three degrees of phosphates of sodium, potassium, and ammonium are all so soluble that they practically never become precipitated spontaneously, nor do they take part in forming calculi. It is the phosphates of calcium and magnesium that form precipitates, but what has been said above of sodium phosphate applies equally to calcium and magnesium phosphates. The less acid a urine is, the more will the less soluble varieties of calcium and magnesium phosphate preponderate, and it is on this account that phosphates come down in alkaline or neutral rather than in acid urines.

Again, it is often stated that phosphates are less soluble in hot urine than they are in cold, and this is given as the reason for the cloud of precipitated phosphates that so often forms when a urine that is not already very acid is boiled. This, however, does not express the real reason for the cloud ; the heat does not precipitate the same phosphate as the cold urine contained, but leads to the formation of a different, and less soluble, phosphate. The calcium monohydrogen phosphate dissociates into calcium dihydrogen phosphate and normal calcium phosphate ; it is the latter which is so insoluble that it comes down :

$$\underset{\text{Medium solubility.}}{4CaHPO_4} = \underset{\substack{\text{Relatively insoluble, and so}\\ \text{coming down as a cloud.}}}{Ca_3(PO_4)_2} + \underset{\substack{\text{Relatively the most}\\ \text{soluble of the three.}}}{Ca(H_2PO_4)_2}$$

Milky Urine.—The urine of many healthy people, especially children, and eaters of large public dinners, is sometimes milk-like when it is passed soon after a full meal. Many a person has become alarmed at the sight, and has feared some grave disorder of the sexual organs or functions, especially either gonorrhœa or spermatorrhœa. The condition is physiological. It results from increased quantities of hydrochloric acid being required in the stomach at the time. The result of this is that the urine temporarily contains such an abundance of bases in proportion to acids that the less soluble monohydrogen phosphates exceed the more soluble dihydrogen phosphates, and they may become precipitated even in the urine that is still within the bladder. The commonest salt to come down is calcium monohydrogen phosphate, $CaHPO_4$, which is either amorphous, or else assumes the form familiar as 'stellar phosphate.' $MgHPO_4$ may come down with it in the form of amorphous particles, or as needles.

The alternation between oxaluria and phosphaturia exhibited by some individuals is discussed on p. 424.

Ammonio-magnesium Phosphate.—This, generally known as triple phosphate, $MgNH_4PO_4$, is comparatively insoluble, and when precipitated it nearly always assumes the form of prisms—the familiar 'knife-rester' or 'coffin-lid' crystals (*Fig.* 221). It is clear that these will come down only when the urine contains ammonia. The latter may of course have been produced by ammoniacal decomposition of urea after the urine was passed. If urinary decomposition after passage can be excluded, however, it is usually stated that the presence of ammonio-magnesic phosphate crystals indicates a purulent lesion in the urinary tracts, especially in the bladder. It is quite true that ammoniacal urines from cases of cystitis often abound in crystals of triple phosphate. The diagnosis is given by the pus cells and so forth, however, and not by the triple phosphate crystals. It is important to remember, moreover, that each day's urine normally contains enough ammonia for ammonio-magnesic phosphate crystals to occur in an absolutely healthy urine, even apart from decomposition on standing. This fact detracts very greatly from the value formerly attributed to the detection of triple phosphate crystals in the urine. Indeed, the importance of phosphates in the urine lies almost entirely in the fact that errors of interpretation may arise unless their physiological behaviour is clearly understood. Microscopically they are often amorphous, but the three well-defined forms of crystals shown in *Fig.* 221 may be recognized microscopically. The chief chemical test is the addition of dilute acetic acid, which causes a precipitate of phosphates to clear up. The main importance of recognizing them correctly is to avoid mistaking phosphates for pus in the case of a spontaneous deposit, for spermatozoa, or gonorrhœa, when the urine comes milky from the urethra, and for albumin in the case of the boiling-test for the latter.

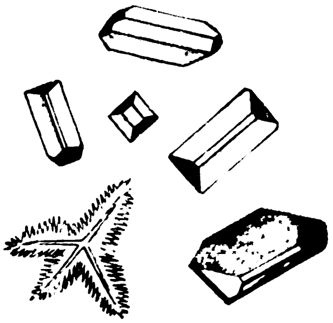


Fig. 221.—Triple phosphate crystals.

Herbert French.

PHOTOPHOBIA, or intolerance of light, may be due to three main groups of causes namely :—

1. **Causes in the Eye itself:**—

Foreign body
Injury
Conjunctivitis
Ophthalmia
Keratitis
Ulceration of the cornea

Iritis
Cyclitis
Glaucoma
Retinitis
Retrobulbar neuritis

Eye-strain from close work in those suffering from uncorrected errors of refraction, especially astigmatism and hypermetropia.

2. **Certain Occupations:**

Involving work under eye-straining conditions, such as affect furnace stokers, electric steel welders, workers in strong sunlight, those who are exposed to the dazzle of snow in strong sunshine, those who come out into strong daylight after prolonged work in the dark, as in coal pits ; those who work much with x-rays. Some persons are unable to stand any strong light, whether of the sun, gas, or electric.

3. Causes not primarily in the Eye itself :—

(*a*). *Some fevers, especially :—*

Measles
Influenza
Typhus.

(*b*). *Intracranial lesions :—*

Tuberculous meningitis
Suppurative meningitis
Epidemic cerebrospinal meningitis
Acute encephalitis
Syphilitic pachymeningitis
Cerebral tumour.

(*c*). *After the administration of some drugs :—*

Arsenic
Potassium iodide
Potassium bromide.

(*d*). *Functional conditions :—*

Migraine
Tic douloureux
Hysteria
Sick headache.

(*e*). *Supra-orbital herpes zoster.*
(*f*). *Severe anœmia* (see ANÆMIA, p. 20).
(*g*). *Secondary to dyspepsia or constipation.*
(*h*). *Malingering.*

Some idea of the cause will generally present itself as soon as the patient gives an account of how the trouble began and how it has progressed. By itself photophobia is seldom a symptom of diagnostic significance, and nearly always there will be other symptoms to assist one. The eyes should be examined carefully in the first place, to find or exclude a local cause, especially glaucoma, which is the most serious of the local lesions ; it is associated in acute cases not only with severe photophobia, but also with acute pain in the eye (see EYE, ACUTE INFLAMMATION OF, p. 231). Retinal changes will be discovered by ophthalmoscopic examination (see OPHTHALMOSCOPIC APPEARANCES, p. 415). *Retrobulbar neuritis* may not cause any visible changes in the optic disc, at any rate not until several days have elapsed, by which time optic neuritis may be visible ; but it may be suggested by the rapid onset of impaired vision without increased intra-ocular tension as in glaucoma, going on perhaps to temporary complete blindness (p. 760).

Eye-strain from errors of refraction requires special ophthalmic knowledge for its exact determination, though it may be suggested by the circumstances of the case.

Occupation photophobia will be likely if the patient works under peculiar conditions of light, though it will be necessary to exclude organic lesions in the eyes by a thorough ophthalmic examination. When local eye conditions have been excluded definitely, the cause of photophobia will generally be either pretty clear at once or soon, or else open to so much doubt that the actual cause may be one of conjecture only.

It will not be the photophobia, but other symptoms, such as the rash, the fever, and the course of the disease, which will give the diagnosis in cases of *measles*, *influenza*, or *typhus fever* ; headache, vomiting, optic neuritis, and the results of lumbar puncture or the Wassermann test in the case of *meningitis*, *encephalitis*, *cerebral tumour*, or *syphilitic pachymeningitis*; knowledge of the drug given and cessation of the photophobia when it is stopped in the case of *arsenic*, *potassium iodide* or *bromide*, or *quinine*.

Migraine and *tic douloureux* are so characteristic in their attacks that they are both diagnosable as a rule from the patient's story ; photophobia may be severe in either, or even extreme in tic douloureux, in which, during the excruciating paroxysms of facial pain, the patient will often desire to remain for days in an absolutely darkened room ; exposure to any light may bring on a paroxysm.

Supra-orbital herpes is generally obvious from the eruption of groups of vesicles on a reddened base along the course of one supra-orbital nerve ; and the photophobia is generally unilateral ; it may persist however for weeks or months after the eruption has subsided, and then its origin may be overlooked.

Malingering can be diagnosed only by catching the patient unawares and discovering that when he is not watched he is able to read in an ordinary light notwithstanding the photophobia he complains of. *Dyspepsia*, *constipation*, and *severe anœmia* are somewhat problematical as causes of photophobia, but might be regarded as its origin in a patient who, suffering from any of the three, also had photophobia without any other discoverable cause, especially if the photophobia disappears when the patient is cured of the constipation, the dyspepsia, or the anæmia, respectively.

Sick headache is a periodic malady of many women and some men. Headache of great severity is the chief symptom, lasting a whole day or even longer ; gastric functions are in abeyance during the attack, so that food in the stomach remains undigested and medicines unabsorbed ; the patient may feel ill enough to have to stay in bed ; towards the end of the attack vomiting occurs once or several times, and by the next day the patient feels perfectly well again. Dislike of any but a dim light is common during the attack. The diagnosis is generally based upon the patient's familiarity with similar symptoms on many previous occasions. The malady is often hereditary ; possibly it is related to migraine.

Biliousness is a very common ailment, due to many different causes, especially injudicious eating or drinking, or to deficient fresh air or exercise. Some cases of recurrent biliousness are closely allied to sick headache and to migraine. During an attack the tongue is coated and the bowels inactive. The precise pathology of the condition is little understood, but the diagnosis is not difficult, although it is important to make a routine examination to exclude more serious lesions. Photophobia, though common in a minor degree in bilious attacks, is seldom very marked.

Hysterical photophobia is met with only occasionally, but when it occurs it may be extreme ; that is to say, the patient may mimic—not intentionally, as in the case of a malingerer, but without at all wishing to do so—so extreme a degree of intolerance to light that she may cover her eyes with deep-tinted glasses, or even cover her whole head with an impervious dark robe ; and may perhaps require to be led about when she walks, like a person totally blind. It is this over-doing of the part that may give the clue to the diagnosis. Naturally a thorough examination, particularly of the eyes, will be required to exclude organic disease, but if functional photophobia appears to be the diagnosis in a particular case, the suspicion may be confirmed by the way the symptom can be made to disappear by sufficiently bold suggestion. *Herbert French.*

PIGMENTATION IN THE MOUTH generally consists of flecks, streaks, or spots of pale brown or pale sepia discoloration of the mucosa, especially upon the inner aspect of the cheeks along a line roughly corresponding to the level of the closed teeth ; with or without similar pigment spots, streaks, or patches upon the mucous surface of the lips, seen best when the latter are everted in a good light ; upon the roof of the mouth, generally upon the soft palate or upon the posterior part of the hard palate rather than more anteriorly ; upon the gums occasionally ; and sometimes upon the sides of the tongue (*Plate XXI*). Such pigmentation of the buccal mucosa immediately suggests *Addison's disease*, especially if there is generalized pigmentation of the skin at the same time, extreme asthenia, a low blood-pressure, a tendency to vomiting or to fainting attacks on any exertion, with inability to maintain any effort, mental or physical. Unfortunately, however, although such buccal pigmentation is highly suggestive of Addison's disease, it is not pathognomonic, for it has been noted in a variety of other conditions also.

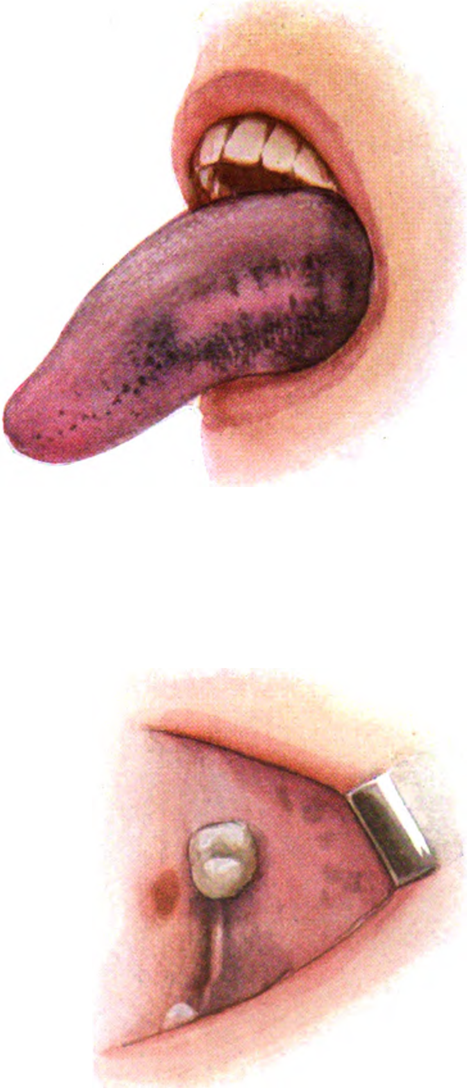
Thus, it is an almost constant feature in persons who have *negro blood* in their ancestry, even though this be from one great grandparent only. This source of difficulty in interpretation is commoner, perhaps, in Africa, the West Indies and America, than it is in Britain, but even here it makes it uncertain sometimes whether one is to diagnose Addison's disease or not.

Then in *pernicious anæmia* buccal pigmentation precisely similar to that of Addison's disease is met with occasionally (*Plate XXII*, p. 528), and unless the blood-count is very definite (p. 24) it may be difficult, even up to the time of post-mortem examination, to say which of the two conditions the patient is suffering from. Probably the right course to follow then would be to treat the case with salvarsan and arsenic, and watch the effect ; pernicious anæmia rallies, temporarily, to these remedies much more certainly than Addison's disease does.

Arsenic itself may cause pigmentation, not only of the skin, but also within the mouth, as was shown by some of the cases in the Manchester epidemic of arsenic in beer poisoning. Some have supposed that the buccal pigment in pernicious anæmia is due to the arsenic employed in treatment ; that this is not so, however, is shown by the fact that some cases

PLATE XXI

PIGMENTATION OF TONGUE AND MOUTH IN ADDISON'S DISEASE



Pigmentation of the tongue and buccal mucosa in Addison's disease.

of pernicious anæmia exhibit pigmented spots inside the mouth even before any arsenic has been given; the patient from whom *Plate XXII* was taken was an instance in point.

One has also seen pigmentation of the buccal mucosa, suggesting Addison's disease, in *chronic cachectic conditions* in which the suprarenal capsules have proved to be healthy at autopsy. In a case of *phthisis* recently, for example, so pigmented was the mouth that it seemed reasonable to diagnose that the tuberculous process was affecting the suprarenals as well as the lungs; yet after death the suprarenals were normal, and one can only suppose that the buccal pigment resulted from the phthisical cachexia which had caused a general tendency to pigmentary degeneration everywhere. In another case Addison's disease was diagnosed during life for the same reason, but at autopsy a *carcinoma* of the splenic flexure of the colon was found, with secondary deposits in the liver; there had been asthenia and general cachexia, and apparently it was the latter which was responsible for the pigment changes. In a third case the patient was both anæmic and cachectic, without any other definite symptoms, and there was extensive pigmentation in the mouth. The blood-count showed extreme anæmia with a colour index that was approximately 1. The doubts during life lay between Addison's disease and pernicious anæmia; both suggestions proved wrong, for at autopsy syphilitic gummata of the liver were found, together with *tertiary syphilitic amyloid disease*.

Fortunately, buccal pigmentation from cachectic states such as phthisis, cancer, and syphilis is rare, though the possibilities have to be kept in mind. In the great majority of cases, however, if pernicious anæmia, arsenic, and negro blood can be excluded, Addison's disease will be diagnosed correctly if the patient is clearly asthenic and ill without any very definite physical signs, but with marked pigmentation within the mouth. *Herbert French.*

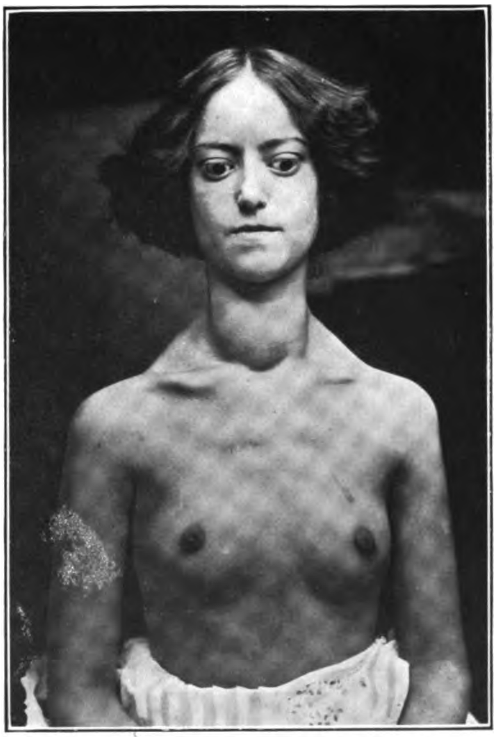


Fig. 222. —A case of typical exophthalmic goitre, to illustrate general pigmentation of the skin in this disease.

PIGMENTATION OF THE SKIN.—Anomalies of the natural pigmentation of the skin, on the side either of excess or deficiency, may be due to irritation of the abdominal sympathetic, and particularly the solar plexus, leading to *general* pigmentation, or to the exudation or extravasation of the colouring matter of the blood, producing *local* pigmentation. Local pigmentation may be brought about by the action of irritants, may result from a condition of hyperæmia, or may be a sequela of skin eruptions. The most familiar errors of pigmentation are covered by the term *chloasma*. This may be either idiopathic or symptomatic. *Idiopathic* chloasma is usually caused by counter-irritants, such as vesicants, or some other form of external irritation, especially scratching, as in vagabond's disease—*phtheiriasis*; but in some cases it is impossible to trace the cause. *Symptomatic* chloasma is a sequela or an accompaniment of cutaneous eruptions, or is the result of abnormal conditions of the uterus or of other abdominal viscera, or of cachexia. It is most often met with as *chloasma uterinum*, which may occur not only in connection with pregnancy, but also in association with any form of uterine irritation. The smooth yellowish-brown patches are seen most commonly on the forehead, but almost the entire face may be involved and also the trunk and limbs. Somewhat similar irregularities of pigmentation occur in *rheumatoid arthritis*, *pernicious anæmia*, *Hodgkin's disease*, *Graves's disease*

(*Fig.* 222), *abdominal tuberculosis*, *constipation*, *chronic intestinal stasis*, and other disorders of the abdominal viscera, and in cases in which *arsenic* has been given over long periods. In *Addison's disease* there is a general bronzing of the skin, together with pigment deposits in the mucous membranes of the mouth (*Plate XXI*), anus, vulva, and urethra ; buccal pigment, however, does not by itself prove that Addison's disease is present, for precisely similar pigmentation in the mouth is observed in some cases of pernicious anæmia (*Plate XXII*), of phthisis without suprarenal disease, and of malignant disease ; whilst negro blood in the ancestry often causes buccal pigmentation in perfectly healthy persons.

Pigmentary abnormalities of the skin occur also in *cachexia* associated with malaria, cancer, nodular leprosy, and secondary syphilis—in malaria, a yellowish-brown to black ; in cancer, a sallow tint ; in nodular leprosy, a fawn colour early in the disease, and a general bronzing at a later stage ; in secondary syphilis, an earthy tint affecting the face. In the rare condition known as *ochronosis*, the skin, cartilages, and sclerotics are blackened, as the result in some cases of alkaptonuria, in others of the prolonged absorption of carbolic acid. In *hæmochromatosis*, another rare condition, apparently due to diseases of the alimentary tract and liver, the patient may be pigmented from head to foot, the prevailing colour being a deep blue-grey slate tint. The diagnosis of *urticaria pigmentosa seu nigricans* is generally clear. Pigmentary deposits in the skin form only part of the skin changes characteristic of *Kaposi's disease*. The pigmentation of *bronzed diabetes* can scarcely be misinterpreted if, when the urine is examined, glycosuria be found ; most cases of this form of diabetes have cirrhosis of the liver as well, so that there is a non-teetotal history.

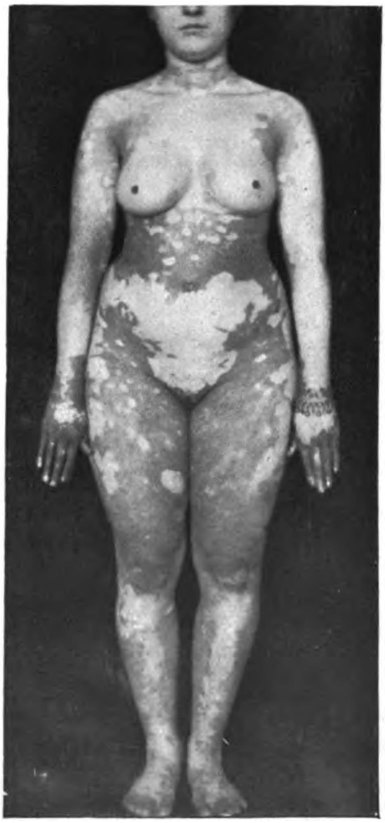


Fig. 223.—Extensive syphilitic leucomelanodermia.

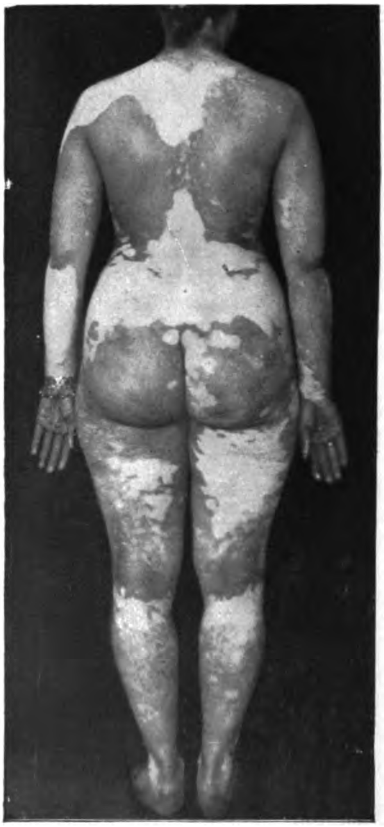
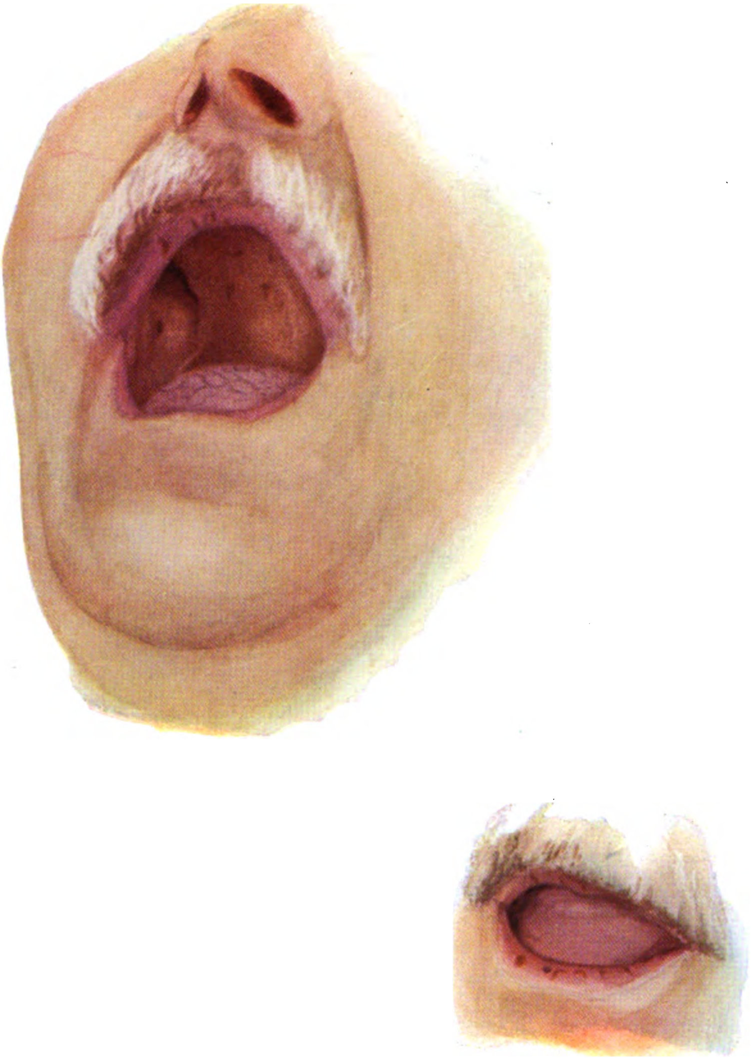


Fig. 224.—The same case as *Fig.* 223, seen from behind.

The diagnosis of the various forms of chloasma is usually easy, though the particular cause of the pigmentation can only be deduced, of course, from the general symptoms.

PLATE XXII

PIGMENTATION OF MOUTH IN PERNICIOUS ANÆMIA



Pigmentation of the mucous membrane of the lips, cheek, and palate, in a case of pernicious anæmia in which no arsenic had yet been given. Addison's disease was simulated, but the diagnosis of pernicious anæmia was confirmed by autopsy.

Chloasma can be differentiated from *chromidrosis*, by observing that in the latter condition the colour, which is derived from the exuded secretions, disappears if washed with ether or chloroform. In *tinea versicolor*, and some other fungous diseases which resemble chloasma, the patches are not smooth but scaly, and the discoloration can be scraped off. The *pigmentary syphilide*, which may take the form of a diffused brownish hue, brownish spots, or dappled patches, is seldom met with except on the neck, but sometimes it may take the form of widespread leucomelanodermia (*Figs.* 223 and 224). Question may arise between chloasma and *leucodermia* (vitiligo) when in the latter condition the white areas have spread over the greater part of the body, and are taken for the normal colour ; but in leucodermia the border of the area is concave, whereas in chloasma it is convex. Moreover, in leucodermia the history is that of the formation of white patches, surrounded by a pigmented border, which may spread until large areas, and even the whole surface of the body, are affected.

Leucodermia has in its turn to be distinguished from sclerodermia, morphœa, macular leprosy, pigmentary syphilide, and partial albinism. The skin is not stiff and thickened as it is in *sclerodermia*. The edges are not streaked with small dilated vessels, making a pink or violet border, as in *morphœa*, nor is there any intermingling of atrophic striæ. The patches are not destitute of sensation as in *nerve-leprosy*, nor, though it has been styled 'white leprosy,' has leucodermia any other resemblance to that affection save the colour of the patches. Doubt as between leucodermia and a congenital condition like *partial albinism* could only arise by disregarding the history. Of albinism itself, whether partial or universal, nothing more need be said here ; for though it is an abnormality of pigmentation, its true character can never be in question. Nor need I speak of jaundice, for that condition forms the subject of a separate article.

Discoloration of the skin may be due to the prolonged administration of drugs. Thus picric acid may turn the skin and the conjunctivæ yellow, arsenic may cause a peculiar greyish, brownish or freckle-like pigmentation (*Fig.* 225, and *Plate VII*, p. 64), nitrate of silver may set up the condition known as *argyria*, in which the integument and the mucous membrane, particularly in situations exposed to light, take on a bluish-grey or greyish-black colour, especially on the face and the flexor aspects of the limbs. This condition may closely resemble hæmochromatosis and similar abnormalities, but the history of protracted use of the drug will make the diagnosis clear. Since, however, arsenic may be derived from some unsuspected source, as was the case in the peripheral neuritis epidemic in and around Manchester due to beer containing it as an impurity, chemical analysis of the hair should be made, for arsenic becomes stored up in the hair that grows whilst this is being taken.

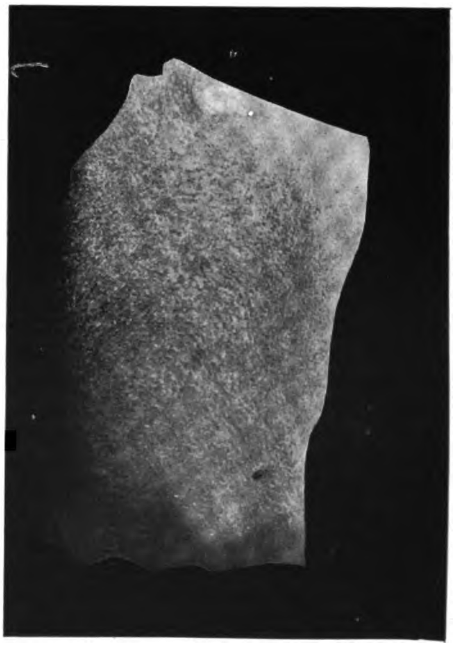


Fig. 225. —Pigmentation of skin from arsenic in a case of pernicious anæmia.

Malcolm Morris.

PLANTAR REFLEX, EXTENSOR.—(See BABINSKI'S SIGN, p. 68.)

PLEURAL EFFUSION.—(See CHEST, p. 102.)

PNEUMATURIA—or the passage of gas per urethram, either along with or independently of urine—is a rare symptom, but when it does occur it is very striking, particularly in males.

It may be due to one or other of two entirely distinct groups of causes, namely :

1. Communication between the rectum, cæcum, vermiform appendix, or other part of the alimentary canal and the bladder, ureter, or renal pelvis; either directly, or *via* an intermediate gas-containing abscess cavity.

2. Infection of the bladder or other part of the urinary tract by micro-organisms that produce gas, without there necessarily being any breach of surface of the mucosa.

When the cause lies in the first group, the patient is very liable to pass fæcal material at the same time as the gas, and the differential diagnosis between the various possible lesions is discussed under FÆCES PASSED PER URETHRAM (p. 238). It should be added, however, that the passage of gas without fæces per urethram by no means excludes there being a fistulous communication between some part of the alimentary canal and the urinary tract; the fistula may be tortuous, so that gas gets along it, but not fæces. It may happen, moreover, that a lesion such as appendicitis has led to the formation of a local abscess which, owing to its infection by the *Bacillus coli communis*, contains gas; this abscess may open into the bladder and cause the discharge of pus and gas, but no fæces, per urethram. The same applies to other abscesses which, though not arising primarily in connection with the bowel, nevertheless occasionally contain gas from infection by the *B. coli communis*—a suppurating hydatid or ovarian dermoid cyst, for instance, or a pyosalpinx.

Sometimes there may be serious doubts as to whether the gas is finding its way into the urinary passages from some external source, as above, or whether it is being produced *in situ*. In the absence of any rectal or other pelvic or abdominal evidence of disease outside the bladder, it will be remembered that certain organisms produce gas when they grow in urine; notably the *Bacillus coli communis*, and in glycosuric cases, various yeasts. The urine will be examined for sugar, and if it be present, a catheter specimen will be obtained to see if saccharomyces are present in the bladder-urine; if so, and if there is no pus or evidence of infection by other micro-organisms, the nature of the pneumaturia will be clear; as a rule, in these cases the patient voids urine that is bubbly rather than distinct and separate from the gas. If, on the other hand, no sugar is present, a catheter specimen will be cultivated to find out whether the *B. coli communis* is present, and if so, in what quantity. If it is, and if no sign of any fistulous communication between any part of the bowel, or a gas-containing abscess cavity, and the urinary tracts, can be made out, the presumption will be that the pneumaturia is due to coli bacilluria, although the latter is far commoner without than with pneumaturia. The urine in these cases may contain very little obvious pus and only a trace of albumin; it may be acid, and not foul smelling or ammoniacal; on the other hand, it may sometimes be so foul and fæculent as to cause serious suspicions of a communication between the colon and the bladder, even when there is none. A cystoscopic examination will serve to exclude a fistulous opening into the bladder, but it may be much more difficult to exclude a similar communication with the higher parts of the urinary tract, especially the renal pelvis. The latter condition is so rare, however, that it is wiser to diagnose coli bacilluria only unless there is direct evidence of a cause for communication between the bowel and the renal pelvis, such as a carcinoma coli.

Herbert French.

PNEUMOTHORAX, or gas in the pleural cavity, may exist with or without clear fluid, pus, or blood in the lower part of the pleura at the same time. If there is any kind of fluid in the cavity along with the air, the fact is generally made obvious at once, when the patient's thorax is auscultated whilst it is being actively or passively shaken, so as to produce the typical succussion splash, often followed by the ringing sounds made by drops of fluid falling from the compressed lung into the pool of fluid beneath. The nature of the fluid—hydro-pneumothorax, pyo-pneumothorax, or hæmo-pneumothorax, as the case may be—can seldom be diagnosed except by means of an exploring needle and syringe. Whether the pneumothorax is or is not associated with any of these fluids, the diagnosis is generally easy on account of the deficiency in movement of the affected side of the chest, the displacement of the heart in the opposite direction, and hyper-resonance to percussion, together with remarkable deficiency or complete absence of the vesicular murmur and voice sounds. The coin-tap sound, obtained by placing one silver coin on the chest wall, tapping it with another silver coin, and listening through the stethoscope for the ringing echo produced, when the sign is positive, may serve to confirm the diagnosis, but it is not essential. Partial pneumothorax, in which complete collapse of the lung is prevented by adhesions, is proportionately more difficult to diagnose, but the same type of physical signs, including the coin-tap sound or *bruit d'airain*, will generally be found in these cases, though in less degree than when the pneumothorax is complete. The *x*-rays show an abnormal clearness corresponding to the air in the pleural cavity (*Fig.* 226.) It is not sufficient, however, merely to diagnose pneumothorax; its cause has to be determined from amongst the following :—

Phthisis : (*a*) early, (*b*) late.

Rupture of an emphysematous bleb.

Gangrene of the lung with necrosis of the pleura.

Empyema ruptured through the lung.

Instrumental : e.g., after tapping a pleural effusion.

Stabs, or gunshot wounds of the chest wall.

Epithelioma of the œsophagus ulcerating into the pleura.

Gastric ulcer or carcinoma ventriculi, leaking so as to produce a gas-containing subdiaphragmatic abscess, which in its turn may perforate the diaphragm and cause a pneumothorax.

Infection of the pleural cavity by gas-producing organisms, such as the *Bacillus coli communis.*

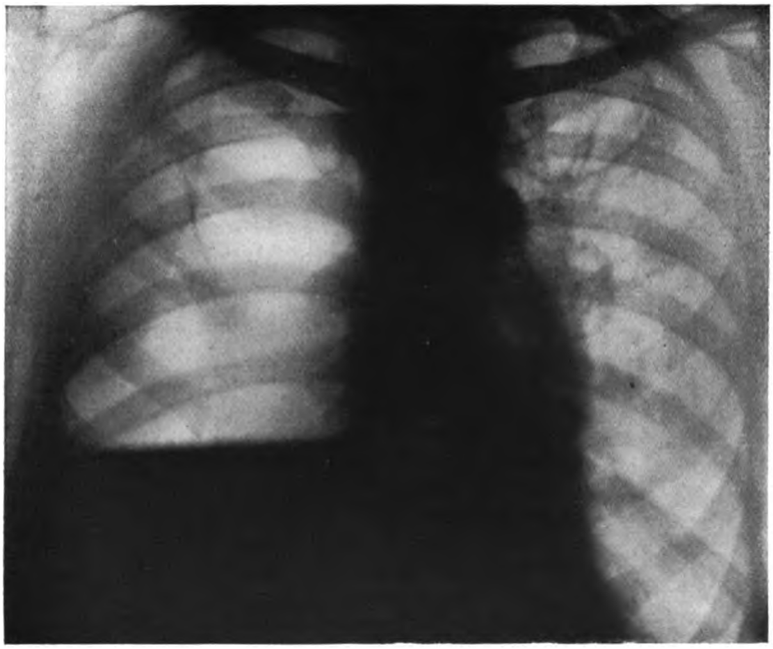


Fig. 226. —Skiagram of a right-sided pyopneumothorax (left side of the skiagram). Note the horizontal upper level of the pus (black) and the clearness of the pneumothorax above it. (*Skiagram by Dr. Lindsay Locke.*)

The commonest cause by far is *phthisis* ; and when the occurrence of the pneumothorax does give rise to symptoms, it is generally due to comparatively early phthisis ; indeed, when it comes on acutely with sudden lancinating pain in one side of the chest, associated with rapid shallow breathing, and cyanosis with or without hæmoptysis in a young apparently healthy adult, it is almost certain that the patient has a tuberculous focus at one apex, even though, as frequently happens, there have been no abnormal symptoms previously, such as cough or night sweats, and even though absolutely no abnormal physical signs can be detected at the apex of the other lung. There may be a little sputum, and in this tubercle bacilli may be detected.

When pneumothorax is attributed to *rupture of an emphysematous bleb*, there must always remain in the physician's mind a serious doubt as to whether it is not really due to a bleb in the immediate neighbourhood of an undiagnosable tuberculous deposit, and the case should be treated as one of potential phthisis.

If the tuberculous process in the lung has made considerable advance pneumothorax is far less common, because there will almost certainly have been pleurisy with thickening and adhesions sufficient to prevent pneumothorax occurring ; nevertheless, in some such cases pneumothorax does develop, and the diagnosis of its cause is easy both on account of the abnormal physical signs and of the sputum with the tubercle bacilli in it. In a later stage still, the occurrence of pneumothorax may cause very little additional disturbance, on account of the extent of lung already diseased, and although its cause would be obvious enough, the occurrence of the pneumothorax often escapes detection.

When the patient has had a *pleuritic* or *pleural effusion* tapped, detection of air free in the pleural cavity next day is by no means uncommon ; it does not follow that this air has leaked in through the tapping instrument, for it is quite as commonly derived from the rupture of superficial alveoli which have been re-expanded rather too rapidly in the withdrawal of fluid by the aspirator. The air generally becomes reabsorbed in a few days, and the temporary pneumothorax is of little significance.

Similar escape of air into the pleural cavity, as the result of cuts, stabs, fractured ribs, or gunshot wounds, is remarkably rare ; this rarity depends upon the fact that the two layers of pleura tend to cohere in a way similar to that which makes two thin sheets of Indian paper difficult to separate, so that when an injury from outside penetrates one layer it nearly always perforates both, and air from within the lung escapes into the subcutaneous tissues instead of into the pleural cavity, and produces surgical emphysema instead of pneumothorax. It rarely happens that an injury separates a sufficient area of the two layers of pleura one from the other to cause a pneumothorax.

All the remaining causes of pneumothorax in the list above are uncommon, and none of them will arise without there having been other symptoms to indicate the nature of the malady. It is possible for an *empyema* to rupture into a bronchus, and so lead to the sudden expectoration of much foul pus, without any pneumothorax arising, or at least none of any extent, because for such an empyema to rupture into the lung it must have been shut off all round by firm pleural adhesions. When pneumothorax results from *gangrene of the lung* due to any cause (p. 259), it is but a terminal factor in an already serious disease, and may even pass without recognition on account of the severity of the symptoms already existing in the case. *Gas-containing abscesses beneath the diaphragm*, such as may either perforate directly into the pleural cavity or lead to infection of that cavity by the *Bacillus coli communis* or other gas-forming organism, never arise suddenly, but are preceded by a simple or malignant ulceration of either the stomach, duodenum, or colon, the symptoms of which will generally have existed for days, weeks, or months ; so that if the possibility of gas appearing in the pleural cavity in this way is borne in mind, the diagnosis of its origin need not be difficult. The *x*-rays may serve to show a large gas bubble below the diaphragm as well as gas in the pleural cavity, and that the gas bubble is not intragastric may be demonstrated by filling the stomach with a bismuth meal and finding that the gas bubble does not become blackened. *Herbert French.*

POLYCYTHÆMIA is the term used to denote a material increase of the red corpuscles above their normal number per cubic millimetre of blood. In males they should average 5,000,000 per c.mm. ; in females, 4,500,000. Any considerable increase above these figures, for instance up to 6,000,000 per c.mm., or more, constitutes polycythæmia. Figures as high even as 14,000,000 are reached sometimes. The following are some of the conditions in which polycythæmia occurs :—

1. Congenital heart disease of the type spoken of as morbus cœruleus—generally due to pulmonary stenosis (see *Fig.* 46, p. 111, and *Fig.* 67, p. 157.)
2. Persons who live in high altitudes.
3. Patients afflicted with chronic shortness of breath, with a tendency to periodic cyanosis, particularly cases of (*a*) mitral stenosis, (*b*) fibroid lung with and without bronchiectasis, (*c*) chronic bronchitis and emphysema, (*d*) spasmodic asthma, (*e*) some renal cases.
4. Patients who have recently lost a quantity of fluid from the tissues, the result of such conditions as (*a*) severe vomiting, e.g., the uncontrollable vomiting of pregnancy ;

(*b*) severe diarrhœa, e.g., the summer diarrhœa of infants, cholera, ptomaine poisoning, arsenic; (*c*) inability to obtain fluid to drink, especially if there exists already a disease tending to polyuria, such as diabetes mellitus, diabetes insipidus, or granular kidney.

5. From the prolonged use of certain drugs, especially acetanilide, veronal, and some others of the type that may cause methæmoglobinuria (p. 284).

6. Splenomegalic polycythæmia.

As a rule, the diagnosis of the cause of polycythæmia in a given case is not difficult. When it is due to *congenital heart disease* it is nearly always associated with an extreme degree of cyanosis without proportionate dyspnœa, and with clubbing of the fingers, both these dating from birth or early childhood. The patient is generally young, though some survive into adult life. There is not always a cardiac bruit, and the precise lesion will then be obscure; often, however, percussion shows increased cardiac dullness to the right of the sternum, and upwards towards the second left rib, indicating increased size of the right auricle and ventricle; and in most cases there is either a loud rumbling or blattering systolic bruit of pulmonary stenosis, heard loudest in the second left space close to the sternum, but also audible over the greater part of the precordial region, and often over both sides of the chest in front and behind; or else a very similar universal systolic bruit, differing chiefly in having its maximum intensity either behind the sternum between the two fourth ribs, or else in the fourth left intercostal spaces close to the sternum, indicative of patent septum ventriculorum. These two lesions may both be present in the same patient, and they are the commonest cause in cases of morbus cœruleus that survive infancy. The red corpuscles seldom number less than 6,000,000 per c.mm., and in some cases they have been no fewer than 14,000,000 per c.mm. The percentage of hæmoglobin is also increased greatly, but usually to a less degree than are the red cells, so that the colour index falls below 1. This applies to nearly all causes of polycythæmia. There is no simultaneous increase in the number of leucocytes per c.mm., and the differential leucocyte count falls within the normal limits. It is noteworthy that cases of persistent ductus arteriosus seldom present either cyanosis, clubbed fingers, or polycythæmia.

Residence at *high altitudes* often causes polycythæmia. The increase is seldom extreme, but the red cells not infrequently reach 6,000,000 or more per c.mm. This rule is not by any means universal, however, though upon the whole the higher the altitude the higher the normal average number of red cells per c.mm., particularly in those who have resided long and continuously in the mountains. The individuals are not ill; it is merely that their red cells stand at a higher figure normally than do those of dwellers nearer sea level.

Quite apart from the presence or absence of anasarca, patients suffering from chronic lesions which tend to produce dyspnœa are also very apt to have polycythæmia, particularly when the lesion causes marked redness of the lips. This is well seen in many cases of *mitral stenosis* when there has been a tendency to failure of compensation for some time past. There is no similar polycythæmia in aortic cases unless mitral disease is present as well, and the red cells are much less increased in mitral regurgitation than in mitral stenosis. In the latter they often reach 6,000,000 or even 7,000,000 per c.mm., and it would seem to be an attempt on nature's part to try and compensate for the failing circulation by distributing the hæmoglobin over a larger corpuscular area. The same explanation probably accounts for the similar polycythæmia due to morbus cœruleus, and to high altitudes, and to certain cases of *fibroid lung*, *bronchiectasis*, *emphysema*, *chronic bronchitis*, *renal disease* with chronic dyspnœa, and *spasmodic asthma*, in which some degree of polycythæmia, though not the rule, is sometimes met with, just as it is in mitral stenosis. The colour index is less than 1, for although the hæmoglobin is increased, it is less so than are the red cells. The leucocytes remain unaltered. The polycythæmia will seldom if ever be the most prominent symptom in the case, so that the diagnosis will nearly always have been made upon other grounds—the presystolic bruit at the impulse; the displacement of the heart towards that side where the lung presents an impaired note, with or without crackling râles and bronchial breathing; and so on. The maintenance of the polycythæmia is important, however, and therapeutic measures should be directed to this end, for many cases of mitral stenosis with 5,000,000 red cells per c.mm. are relatively anæmic; they should have 6,000,000 or more.

The effect of *cholera*, *ptomaine poisoning*, *arsenic*, *summer diarrhœa* of infants, *severe thirst* that cannot be assuaged, the *toxæmia of pregnancy*, and so on, in concentrating the blood by withdrawing or withholding fluid from it, and thus producing some degree of polycythæmia, is an acute condition which is to be counteracted by continuous saline infusion or some similar method of restoring fluid to the tissues. The polycythæmia seldom reaches any marked degree except in quite early stages, for instance, in cholera; later, the red cells disintegrate more rapidly than they are replaced. In measuring the concentration of the blood in these conditions, it is of less value to count the red cells than to measure the specific gravity. This is done most readily by the chloroform and benzene method. The specific gravity of chloroform is high, that of benzene is low, and by mixing the two in different proportions it is possible to obtain fluids of every intermediate specific gravity. A mixture of the two of the normal specific gravity of the blood, viz., 1056, is made, and poured into a specimen glass of sufficient depth to allow a urinometer to float in it. For strict accuracy, certain corrections in the readings of the ordinary urinometer are required, but for emergency use the instrument will serve. The lobule of the patient's ear is pricked, a large drop of blood is allowed to fall into a suitable small cup or other receiver containing some of the chloroform-benzene mixture, and thence transferred to the main bulk of the fluid in the specimen glass. If the blood-drop sinks, more chloroform must be added; if it floats, more benzene; ultimately a point is reached at which the blood-drop neither sinks nor floats; the specific gravity of the chloroform-benzene mixture is then the same as that of the blood. In cases of collapse from loss of fluid there is a rise in the specific gravity of the blood even when there is no polycythæmia,—and the greater the rise, the greater the need for infusion.

Splenomegalic polycythæmia is a somewhat rare condition that is also termed *erythræmia*, or *erythrocythæmia*. Its name suggests its main features, which are: Enlargement of the spleen, increase in the red cells up to as many as 10,000,000 per c.mm., or even more, and duskiness or lividity of the face (*Plate XXIX*, p. 634) and of the extremities. The nature of the malady is still obscure, though some regard it as due to disease of the bone-marrow. It affects adults and females rather than children and males, and its course is chronic. It only remains to add, that whereas to be typical the spleen must be enlarged, there are cases, probably of the same affection, in which, without the spleen becoming palpable, the only definite clinical signs are progressive lividity and polycythæmia. *Herbert French.*

POLYDIPSIA.—(See THIRST, EXTREME, p. 719.)

POLYURIA.—The term polyuria signifies the passage of more than the average amount of urine per diem. It may be either (I) *Transient*, or (II) *Continued*. It is important not to mistake frequency of micturition for polyuria, for although the latter almost necessarily causes the former, there are many conditions that lead to frequency of micturition without polyuria—for example, tuberculous ulceration of the bladder, enlargement of the prostate, or urethral stenosis, in all of which urine may be passed frequently, but in small quantities at a time. In case of doubt the total amount of urine passed in each period of twenty-four hours should be measured. The normal limits are very wide, the average being about 50 ounces per diem, more being passed in cold weather than in warm, during rest than after exercise, waking than sleeping, and after drinking than after taking little fluid by the mouth. The point at which polyuria begins is arbitrary; if a patient passes 70 ounces or more per diem it is almost certain to attract attention, and therefore to merit the term polyuria. In degree, the polyuria due to causes in Group I seldom exceeds 100 ounces a day; some of the causes in Group II, especially diabetes mellitus and diabetes insipidus, may cause polyuria to the extent of 200 ounces, or even 300, 400, 500, 600, or more, per diem.

In arriving at the differential diagnosis of the cause in any given case, one of the first points to note is whether the polyuria is persistent; or whether, even if recurrent, it is transient. Any of the causes that usually give persistent polyuria may in some individuals produce the symptom intermittently, but upon the whole one may classify the causes of polyuria as follows:—

I. CAUSES OF TRANSIENT POLYURIA.

1. After drinking abundance of water or other fluid.
2. After drinking fluids containing diuretic principles, such as alcohol (wine, malt liquors, spirits); caffeine (tea, cocoa, coffee); citrates or tartrates (artificial lemonades).

3. As the result of nervousness, or of nervous attacks, such as :—
    - (*a*) Medical examination for life assurance
    - (*b*) Preparation for some physical or mental competition
    - (*c*) Hysteria, especially during recovery from an acute outburst
    - (*d*) Neurasthenia
    - (*e*) After an epileptic attack
    - (*f*) After migraine
    - (*g*) After an asthmatic attack
    - (*h*) After an attack of angina pectoris
    - (*i*) Periodic polyuria, apparently without cause.
4. Hydronephrosis, with periodic emptying of the renal sac.
5. The cold stage of a malarial attack.
6. In some cases of convalescence from a febrile illness, such as enterica.
7. As the result of the clearing up of extreme œdema or serous effusions,—for instance, during recovery from acute nephritis ; mitral stenosis, with heart failure ; cirrhosis of the liver, and so on ; especially if the fluid clears up quickly after giving diuretic remedies, such as blue pill, digitalis, calomel, copaiba resin, potassium salts, diuretin, sodium-theocin-acetate, uva ursi, broom tops, or dwarf elder.

When the cause lies in Group I its nature is generally obvious, though it is essential to examine the urine carefully for sugar, albumin, and renal tube-casts, for purposes of exclusion. Nevertheless, the diagnosis may be in doubt until the course of the symptom has been watched for a while. For instance, polyuria may seem to be due to profuse drinking, when really the kidneys are granular and contracted ; or in a life insurance case, nervousness may seem to be the cause, when there has really been a bout of drinking ; or, again, the drinking may be secondary to the extreme thirst produced by diabetes insipidus.

Excessive Drinking.—When due to drinking water, tea, wine, spirits, or artificial lemonades, the polyuria ceases when the drink in question is limited.

Nervousness.—The history and circumstances of the case, together with the absence of signs of gross disease of heart or kidneys, will be the main factors in deciding whether the polyuria is caused by excitement, nervousness, hysteria, or neurasthenia.

Epilepsy.—The character of the convulsive seizures, their recurrence at intervals, and the influence of bromides upon them, will serve to diagnose epilepsy, for polyuria in association with the latter nearly always follows immediately after an attack of grand mal. It may, however, be associated with petit mal, or even be the chief phenomenon in some cases of epilepsy.

Migraine.—The diagnosis of migraine depends on the history of the case and the absence of optic neuritis and other evidence of gross intracranial disease.

Asthma.—Asthma is sometimes easy to diagnose, sometimes very difficult ; it may be mistaken for recurrent bronchitis, cardiac dyspnœa, renal dyspnœa or uræmic ‘ asthma,’ mediastinal new growth, thoracic aneurysm, thymic ‘ asthma,’ laryngeal papilloma or fibroma, foreign body in the air-passages, syphilitic stenosis of a bronchus, goitre, or hysteria. It so frequently develops into emphysema and bronchitis that one is apt to forget that the essential symptom of asthma is dyspnœa, and not cough. To diagnose a difficult case it may be necessary to examine the chest with the *x*-rays to exclude aneurysm and new growth ; to examine the larynx and vocal cords ; the heart, the retinæ, and the urine to exclude renal and cardiac mischief ; and even then, doubt may remain unless there is a clear and typical account of the nature of the earlier attacks in a patient who has had recurrences for years, and who is relieved by cocaine sprays to the nose, by ethereal tincture of lobelia, by inhalations of stramonium fumes, by injections of small doses of adrenalin, or by other anti-asthmatic remedies ; EOSINOPHILIA (p. 218) is more likely to be found during an attack of asthma than as the result of any of the other conditions that may simulate it. The polyuria occurs in by no means every case ; when it does so, it generally follows immediately after an attack, and this applies also in cases of *angina pectoris*, the diagnosis of which is not difficult when the acute attacks of precordial pain radiate upwards and outwards to the left shoulder and down the left arm, and when there is evidence of an aortic lesion, or of atheroma and arteriosclerosis with high blood-pressure.

Periodic Polyuria, apparently without cause, is a condition which is regarded by some as a clinical entity ; the diagnosis must always be difficult to be sure of, however, and the more carefully a cause is looked for, the fewer will be the cases remaining in this category ; it will be found that some are due to epilepsy ; others to secret drinking ; others to granular kidney ; others to hydronephrosis, and so on.

Hydronephrosis, with periodic emptying of the renal sac, is the chief cause of typically periodic polyuria. The diagnosis is arrived at by having the urine measured carefully each day, and by palpating the loins bimanually for evidence of renal enlargement. When a kidney swelling can be detected, and when this increases in size at the same time that less urine is being passed, whilst it materially decreases on the days when the polyuria occurs, the diagnosis of hydronephrosis or pyonephrosis is clear ; and the distinction between the two depends on whether there is or is not pyuria. The commonest causes for hydronephrosis are movable kidney and renal calculus ; and the *x*-rays often serve to distinguish the latter from the former.

Fevers.—The polyuria that occurs during the cold stage of a malarial attack is replaced speedily by the opposite condition when the hot stage is reached ; the diagnosis is afforded by the circumstances of the case, such as residence in a malarial district and previous attacks of the malady ; by the discovery of malarial parasites in blood-films ; by the absence of leucocytosis, the relative increase in the large lymphocytes in the differential leucocyte count, and by the beneficial effects of quinine upon the disease. Polyuria during convalescence from other fevers, such as enterica or pneumonia, is not uncommon ; it is a phenomenon that may attract some attention at the time, but it seldom gives rise to difficulty in diagnosis.

Œdema and Diuretics.—The considerable polyuria that often results in renal or cardiac cases when œdema is clearing up under treatment is noteworthy, but the diagnosis is not, as a rule, difficult. If the polyuria is due merely to the excretion of accumulated fluid it will cease when there is no longer any œdema ; whilst if it is due to granular kidney, or other underlying malady, it will continue even after the œdema has gone.

II. CAUSES OF CONTINUED POLYURIA.

1. Diabetes mellitus.
2. Red granular contracted kidneys.
3. Arteriosclerosis.
4. Pale granular contracted kidneys.
5. Lardaceous or amyloid kidneys.
6. Cystic kidneys.
7. Diabetes insipidus :
   (*a*) Due to no gross nervous lesion
   (*b*) Due to tumour or injury of the medulla oblongata.
8. Incorrigible drinking of beer or spirits.
9. Phosphatic diabetes.
10. Azotic diabetes.
11. Some cases of acromegaly.
12. Some cases of myxœdema.

Diabetes Mellitus.—A very important step in the diagnosis is to examine the urine carefully. If sugar is present, a diagnosis of diabetes mellitus will be made, especially if diacetic acid and acetone are also present, and the specific gravity is between 1035 and 1045. Some authorities distinguish in kind as well as in degree between what they term true diabetes mellitus on the one hand, and alimentary glycosuria on the other, though others hold that these differ only in degree ; it is chiefly in severe diabetes of young people that polyuria is marked, something between 100 and 600 ounces of urine being passed per diem ; in elderly people with glycosuria the polyuria is often slight ; in these cases the specific gravity need not be above the normal, and diacetic acid and acetone are generally absent. If no sugar is present upon one occasion it may be on another, so that several examinations may be required.

Albuminuria.—If albumin is present, and the polyuria cannot be attributed at once to anything so obvious as the clearing up of œdema or the administration of a diuretic, a careful microscopical examination of the centrifugalized deposit for renal tube-casts should be made; if the latter are absent, and if the patient is a young adult male, who seems to be in good health, whose heart and other organs present no abnormal physical signs, and whose polyuria troubles him chiefly at times of excitement, for instance when he is in for an examination, the diagnosis is very likely to be that of 'functional' or 'physiological' albuminuria, in which case repeated tests will show that the urine is often quite free from albumin, especially the first thing in the morning, and the blood-pressure would not be raised. If, on the other hand, more than an occasional renal tube-cast was found, and the albumin and polyuria were persistent, the diagnosis of red granular contracted kidney, arteriosclerosis, pale granular contracted kidney, lardaceous kidney, or cystic kidney would suggest itself. The differential diagnosis between these is discussed under ALBUMINURIA (p. 6, et seq.).

Diabetes Insipidus.—If neither albumin nor sugar is found, even on repeated testing, and if the polyuria is extreme and persistent, whilst the specific gravity of the urine is constantly low (1004 to 1008), a diagnosis of diabetes insipidus will suggest itself. Before this diagnosis is made finally, however, precautions must be taken to determine that the patient's thirst and polyuria are not due to habits of drinking to excess: it may be difficult to decide this in cases in which alcoholic beverages are consumed; but when the patient is a water-drinker, and yet cannot do with less than 8 or 10 pints a day, the drinking is probably a necessity, and not a habit; and diabetes insipidus is the probable diagnosis. In cases of doubt, the difficulty can be decided by restricting the intake of fluid and determining the specific gravity of the blood. This should be about 1056, and in a case where polyuria is due to drinking habits, restriction of fluids will not alter it materially; in a case of diabetes insipidus with restricted intake of fluids, however, the drain of the latter from the blood still goes on, and the specific gravity rises to 1060, or 1065, unless the patient is allowed fluid by the mouth again.

There are two classes of diabetes insipidus, according as there is, or is not, a gross lesion of the central nervous system. If the malady follows on a fractured base of the skull, or if there are vomiting, headache, optic neuritis, or other symptoms of cerebral tumour, there is probably a gross lesion of the base of the brain in or near the medulla oblongata—thrombosis, softening, hæmorrhage, small aneurysm, gumma, glioma or other neoplasm. In other cases, the complaint arises after a fright or shock, or even without any apparent cause, and there seems to be no gross lesion to account for it.

Phosphatic and Azotic Diabetes.—Another point that needs investigation in a case suspected to be diabetes insipidus, is the amount of solids excreted daily in the urine. In ordinary diabetes insipidus the total solids are normal, the only increase being in the water. There are rare cases in which, in addition to polyuria, there is a great increase in the total solids in the urine also—so-called *baruria*. Rare though these cases are, they have been divided into two types, namely, those in which the inorganic salts are most increased—phosphatic diabetes (p. 522), and those in which the nitrogenous constituents are augmented mainly—azotic diabetes. The diagnosis here depends mainly on quantitative estimation of the various urinary substances.

Acromegaly and **Myxœdema.**—It only remains to add that symptoms not unlike those of diabetes insipidus have sometimes arisen in cases of acromegaly and in myxœdema. There is probably a nervous factor in both cases, coupled in myxœdema with dryness of the skin, and consequent deficiency in perspiration; whilst in acromegaly there is the tumour-like enlargement of the pituitary body which may cause polyuria like any other lesion near the medulla oblongata. The diagnosis of acromegaly may be confirmed by the *x*-rays, which will show the great enlargement of the bones of the hands, feet, and head, or even perhaps of the pituitary fossa itself; whilst in myxœdema, if the general symptoms, the pseudo-œdema of the legs, the acquired dullness of intellect, the increasing weight, and the broadening of the features (*Fig.* 104, p. 234), the fingers, and the hands, do not at once indicate the nature of the complaint, the beneficial effects of treatment by thyroid extract may serve to clinch the diagnosis. *Herbert French.*

PRECORDIAL PAIN.—(See PAIN IN THE CHEST, p. 430.)

PRIAPISM signifies erection of the penis, continual, of troublesome degree, and not necessarily accompanied by sexual desire. Though generally spoken of in connection with the male sex, a precisely similar affection may occur in the female clitoris. The symptom is not often by itself of diagnostic importance, though it may be due to a considerable number of different causes. Most of the latter need be little more than enumerated, for if they are borne in mind they will nearly always lead to a speedy diagnosis. Two in particular merit special mention, however. The first is priapism in elderly men. In some such cases there may be enlargement of the prostate, or local inflammation such as gouty urethritis, but in many the priapism seems to occur, without pathological cause, as a sort of final outburst of sexual energy before the onset of senile impotence.

The other special condition under which priapism may be extreme is after injury to the upper dorsal region of the spinal cord. The damage may be so serious as to have produced a fracture dislocation of the spine with paraplegia, in which case the diagnosis will be obvious; short of this, however, there may have been only a minor degree of injury, with contusion and perhaps multiple small hæmorrhages into the substance of the cord, in association with which priapism may in some instances be very pronounced and last for weeks or months before recovery occurs.

For the rest, the causes of priapism may be summarized briefly according to age periods, the chief being :—

Priapism in infancy :—

- Phimosis
- Oxaluria
- Worms, especially oxyuris vermicularis
- Balanitis
- Posthitis
- Calculus, urethral or vesical
- Certain conditions of mental deficiency
- Circumcision.

Priapism at puberty :—

The changes in the genital organs associated with the onset of puberty.

Priapism in young adult life :—

- Sleeping on the back
- Non-emptying of the bladder when full
- Ill-fitting trousers
- Sexual excitement
- Gonorrhœa
- Epilepsy
- After circumcision
- Masturbation
- Convalescence from an acute disease
- Tetanus
- Hydrophobia
- Leukæmia
- Thrombosis of the pampiniform plexus
- Fracture of the dorsal spine
- Transverse myelitis of the upper dorsal region
- Spinal meningitis
- Certain aphrodisiac drugs :—
    - Cantharides
    - Turpentine
    - Alcohol, especially port wine in some persons, champagne in others
    - Strychnine
    - Cannabis indica
    - Camphor
    - Phosphorus
    - Damiana.

Priapism in older men :—

- The male menopause
- Local irritation as the result of :—
    - Gouty urethritis
    - Enlarged prostate
    - Hæmorrhoids
    - A loaded rectum.
- Hæmorrhage into the middle lobe of the cerebellum
- Lesions of the pons varolii

Very seldom indeed will priapism be the only symptom in the case; the diagnosis will be made from the history and from the other symptoms. *Herbert French.*

PROLAPSE OF THE UTERUS.—As a matter of practical fact, the uterus only descends as a result of a much wider displacement of all the movable structures which go to make up the pelvic floor. This is composed of a movable or pubic portion, and a fixed or sacral portion, and it is descent of the pubic portion which produces the actual lesion known as prolapse of the uterus. In other words, the uterus only descends because it is a part of the pubic portion of the pelvic floor. The uterus, bladder, and anterior vaginal wall are normally kept in position chiefly by the connective-tissue sheaths which accompany the blood-vessels supplying them, and it is injury and stretching of this connective tissue which allows of descent of the organs named. There is no doubt, however, that injuries to the fixed portion of the pelvic floor, the perineal body, and levatores ani muscles and their fasciæ, will contribute something to the facility with which the structures mentioned may descend. In practice, therefore, prolapse of the uterus and descent of the pelvic floor lead to the appearance of a swelling at the vaginal orifice. There are other swellings which come down the vagina and appear at the vulva, and from them, therefore, prolapse of the uterus has to be diagnosed. These swellings are : (1) *Hypertrophic elongation of the cervix uteri* ; (2) *A tumour protruding from the vagina* ; (3) *Inversion of the uterus* ; (4) *Cystocele and rectocele* ; (5) *Extroversion of the bladder.*

Hypertrophy of the Cervix may be of the vaginal, the intermediate, or the supravaginal portion. The first is always congenital, and consists of elongation of the portio vaginalis. It may protrude from the vaginal entrance, but the vaginal fornices will be found unaltered at their usual level, and the sound will pass an increased distance proportionate to the length of the portio. The os uteri forms the apex of the protrusion. The fundus remains at its usual level. In hypertrophy of the intermediate portion the anterior fornix of the vagina is carried downwards with the cervix and may be obliterated, whilst the posterior fornix remains at its usual level, because the elongated portion lies between the insertion levels of the anterior and posterior vaginal walls. The sound passes an increased distance, and the os uteri forms the apex of the protrusion. The fundus remains at its usual level. In hypertrophy of the supravaginal portion *both* fornices are carried down with the cervix, and both may be obliterated. The bladder is displaced downwards, but the rectum does not descend. The fundus uteri will be found on bimanual examination to be at its usual level, whilst in true prolapse the fundus uteri descends as a whole with the rest of the uterus. It is common for some prolapse of the uterus as a whole to accompany elongation of the cervix, and this can be appreciated only by a careful bimanual examination.

A Tumour protruding into or from the vagina is most commonly a *fibromyoma of the uterus.* It may be a pedunculated growth either of the cervix, or protruding through it, in either case hanging free in the vagina. It may grow from the cervix in the connective tissue in front, between the uterus, bladder, and anterior vaginal wall ; or behind, between the uterus, rectum, and posterior vaginal wall ; in either case the vaginal wall is stretched over the growth. The uterus will be felt high up. When the pedunculated growth is protruding from the os the hard ring of the cervix is felt encircling the pedicle. In the case of sessile interstitial growths, the cervix is high up in front or behind the growth, as the case may be, and if the tumour is a large one, may be out of reach altogether. In any case there is no descent of the uterus, and it may even be higher than usual. The growth may be a *fibroid growing from the vaginal wall,* a *mucous polypus* of the cervix, or a *malignant growth.*

Inversion of the Uterus may be chronic, or may occur immediately after labour as an acute condition which could hardly be mistaken for anything else, except perhaps extrusion of a fibroid immediately after delivery. In the latter case the tumour protrudes through the cervix, whilst the whole uterus can be felt above it bimanually, whereas in inversion the uterus turns inside-out, partially or completely, a cup-shaped depression is felt above instead of the rounded fundus, and a finger or the sound will only pass a short way by the side of the mass, or not at all if inversion is complete. Both conditions may be accompanied by hæmorrhage, but that with inversion may be exceedingly severe. Acute inversion is always accompanied by great shock, whilst extrusion of a fibroid is not. Chronic inversion is more likely to be mistaken for prolapse or a polypoid fibromyoma. It is distinguished from prolapse in that the uterus does not necessarily descend as a whole, the cervical ring is felt high up in its usual position, and the sound will only pass a short distance all round the protruding mass, according to the degree to which the uterus is inverted. A cup-shaped depression, instead of the rounded fundus, is felt in the vaginal vault by a hand on the abdominal wall.

Cystocele and **Rectocele** more often accompany prolapse of the uterus, but may occur independently of it. They are essentially bulgings of the anterior or posterior vaginal walls towards or through the vaginal entrance, the bladder or rectum being attached, and following them of necessity. A sound passed into the bladder, or a finger in the rectum, will directly enter the bulging vaginal wall, whilst the uterus will be felt bimanually above in its normal position.

Extroversion of the Bladder can occur either through a congenital defect in its wall, or through an injury to its basal portion; for instance, in removing a growth from the vaginal wall a gap may be left in the bladder through which extroversion may occur. The mucous membrane will be exposed in the vagina, and on it will be seen the two orifices of the ureters, with urine issuing by intermittent jets. The uterus in such a case may have its normal position. *Thos. G. Stevens.*

PROPTOSIS.—(See EXOPHTHALMOS, p. 229.)

PRURITUS.—Itching may occur without visible lesions of the skin, save those due to scratching, or may be associated with various cutaneous eruptions. It is to the former condition that the word 'pruritus' should be restricted. The diseases of which itching is a symptom may be either neuroses, such as hysteria, hypochondriasis, and other affections of the nervous centres, or general nutritive disorders affecting the nervous system secondarily, such as arthritism and diabetes mellitus; or the irritation may be set up by the attacks of parasites, or by definite skin lesions. Itching varies in character: it may be interpreted by the patient as a tingling, or pricking, or as a formication—a feeling as of insects crawling on the skin. It varies also in degree, from a mild sensation which is welcome to the patient from the pleasure he finds in scratching, to an irritation so severe and persistent as to endanger his life from sleeplessness, or his reason from the nervous irritability which it sets up. The affections in which itching is slight are seborrhœa, erythema, pityriasis rubra pilaris, and pemphigus; it is more severe, in varying degrees, in eczema, prurigo, some cases of psoriasis, dermatitis herpetiformis, dermatitis gestationis, application dermatitis, lichen planus, lichenization, lichen urticatus, pityriasis rubra, mycosis fungoides, pityriasis rosea, cheiropompholyx, chilblain, prickly heat, tinea marginata, urticaria, scabies, the various kinds of pediculosis, flea-, mosquito-, and bug-bites, jellyfish and other stings. Even in the affections in which it is usually severe it varies much in degree in different cases. Itching seldom has any distinct diagnostic value, but in cases in which the cutaneous lesions may admit of more than one interpretation, its presence or absence may suffice to turn the balance. Syphilides hardly ever itch.

Pruritus proper may be general or local. Of general pruritus there are four varieties —pruritus universalis, pruritus hiemalis, pruritus senilis, and bath pruritus. The local varieties affect chiefly the anus, the vulva, and the scrotum, but the nares, the palms of the hands, and the soles of the feet may be the seat of the irritation. One of the most curious forms of pruritus is that which is associated with bathing. According to Stelwagon, who has made it a subject of special study, it most commonly affects the legs from the hips downwards; but the forearms also may be involved, and it may have even wider range. It is an affection of adolescence and adult life, and is more frequent in males than in females.

If no lesions of the skin are present save those which can be accounted for, directly or indirectly, by the scratching, the diagnosis of pruritus 'imposes itself.' Care must, however, be taken to exclude all possible sources of parasitic irritation; and it must always be remembered that lice and acari sometimes find harbourage in the most unexpected quarters. If the scratches are on the shoulders, or in the genital region, the presence of lice must be suspected; if on the wrists and between the fingers the burrows of the *Acarus scabiei* must be sought for. Some patients, without developing actual urticaria, suffer from severe itching after the ingestion of certain foods, notably strawberries, or crab. After serum injections pruritus may be extreme, especially about the ninth day, though urticaria generally accompanies it. Pruritus from the irritation of sugar (grocer's itch), or of primula obconica or rhus toxicodendron (gardener's itch); or of satin wood sawdust (carpenter's itch); or of some kinds of soaps; or of the hairs of some caterpillars, may or may not be associated with objective evidence of dermatitis. Only when careful investigation fails to reveal any local source of irritation should the case be diagnosed as one of pruritus pure and simple. *Malcolm Morris.*

PTOSIS is the term applied to drooping of the upper eyelid with inability to raise it to the full extent (*Figs.* 227, 228, 229, 230); it must not be confused with the inequality of the palpebral apertures sometimes observed in people accustomed to screw up one eye.

It is usually caused by *paralysis of the third nerve,* in which case it may also be associated with paralysis of other ocular muscles, either external or internal (*Fig.* 228). Sometimes it is accompanied by paralysis of other motor cranial nerves, the 7th for instance (*Figs.* 229 and 230), in which case the multiple cranial nerve paralyses immediately suggest a syphilitic cause, which may be verified in many cases by means of Wassermann's serum reaction.

Paralysis of the Left Third Nerve.

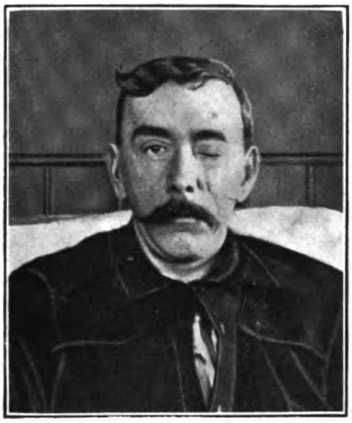


Fig. 227.—The patient's face at rest; there is complete ptosis from paralysis of the left levator palpebræ superioris. Note the scar of the healed gumma on the left cheek near the left angle of the nose.

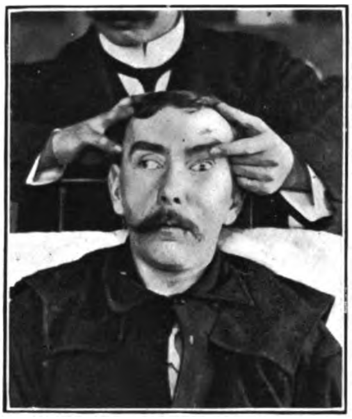


Fig. 228.—The patient is trying to look to his right; the left eyelid is being held up to show that the left eye is unable to look to the right owing to paralysis of the left internal rectus muscle.

Left-sided Nuclear and Infranuclear 7th Nerve Paralysis and Complete Ptosis from Paralysis on the Left 3rd Nerve; the result of Cerebral Syphilis.

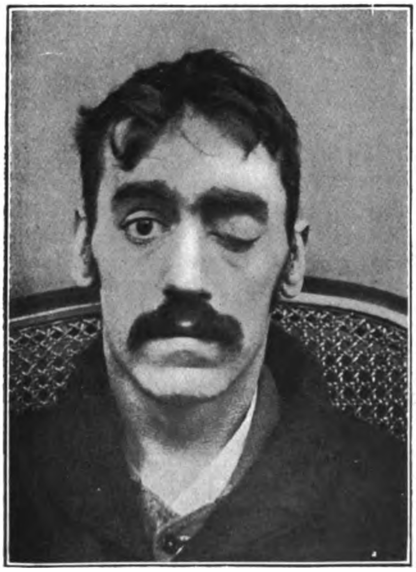


Fig. 229.—At rest.

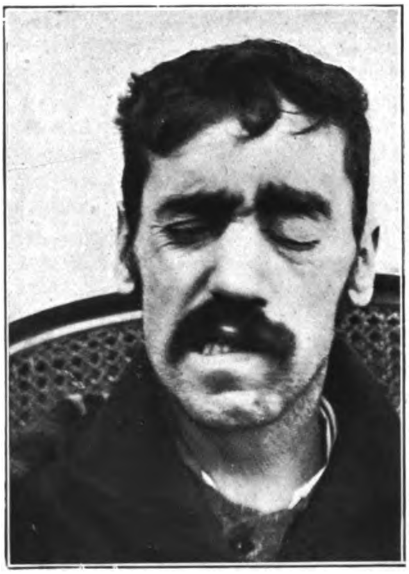


Fig. 230.—When voluntary effort was made to show the teeth and close the eyes.

In *paralysis of the cervical sympathetic*, slight ptosis may be associated with diminution in the size of the pupil on the affected side, and retraction of the eyeball or enophthalmos. It may also occur in *myasthenia gravis* (*Fig.* 111, p, 235).

Ptosis of the lids, associated with much œdema and infiltration of the lids, is also found in all *inflammatory affections* of the conjunctiva, in *angioneurotic œdemas* (*Fig.* 178, p. 412), and is a very constant symptom in *trachoma*.

Congenital ptosis is usually bilateral, and associated with smoothness of the upper lids and absence of all the usual cutaneous folds. The levator palpebræ is absent or ill-developed, and efforts to open the eye are made by the occipito-frontalis muscle.

Herbert L. Eason.

PTYALISM denotes excessive secretion of saliva. It is not easy, however, to determine in every case whether there is really excess, or whether the patient is not merely allowing the normal saliva to dribble from the mouth. It is difficult to draw an absolute distinction, therefore, between dribbling of saliva and ptyalism, though in practice the nature of the case may be obvious enough. One has but to consider the various conditions under which trouble with the saliva may arise to see how in some cases the difficulty is solely one of swallowing the normal secretion, as in bulbar paralysis and in babies ; how in others there is both excess of secretion and difficulty in swallowing it, as in mercurial stomatitis ; and how in others, again, there is too much secretion but no difficulty in swallowing it, as in functional or hysterical ptyalorrhœa. The first step in arriving at the diagnosis of the cause is to inquire carefully as to any *medicine* or *drug* the patient may be taking orally or applying externally, especially :—

- Mercury
- Pilocarpine
- Jaborandi
- Iodide
- Bromide
- Phosphorus
- Arsenic
- Antimony
- Aconite
- Chlorate of potash
- Cantharides
- Copper salts.

Mercury is the most important of these ; its effects are most serious when the mouth is not kept scrupulously clean, particularly when there is also nephritis, as is not uncommon in severe secondary syphilis. The saliva is also apt to have peculiar effects when mercury is being taken; thus, in repairing submarine cables it is customary to use saliva in completing the process of covering in the central core, and it has been found that if the repairer is taking mercury medicinally the repaired part of the cable speedily becomes defective again, in a way which does not result when the repairer is a healthy man taking no medicine.

If the salivation is not due to any drug, it may be the result of one of the many forms of *general stomatitis* :—

- Aphthous
- Dyspeptic
- Septic
- Suppurative
- Ulcerous
- Malignant
- Variolous
- Diphtheritic
- Syphilitic
- Tuberculous
- Due to pyorrhœa alveolaris
- „ necrosis of the jaw
- Due to angina Ludovici
- „ cancrum oris
- „ pernicious anæmia
- „ hæmophilia
- „ sprue
- „ scurvy.

The exact nature of a severe stomatitis will be diagnosed by making a careful local examination, ocular and digital, assisted by the history and, if need be, by bacteriological examination of swabbings from the mouth, by Wassermann's serum reaction for syphilis, or by microscopical examination of a fragment of the affected tissues. Tuberculous stomatitis is one of the rarer forms, but when it occurs it is severe ; it may be primary, but more often is associated with obvious phthisis.

If drugs and general stomatitis can be excluded, local examination may still serve to detect a local cause acting by reflex irritation of the fifth nerve, especially :—

- A jagged carious tooth
- A rough filling
- A stump left beneath a tooth-plate
- A broken or ill-fitting tooth-plate
- A foreign body, such as a fishbone, impacted in the gum
- Neuralgia of the fifth nerve
- A ranula
- A gumboil
- An epulis
- A myeloid sarcoma of the jaw
- A salivary calculus
- An eschar left by some recent irritant or corrosive substance, or injury.

If careful examination serves to exclude all these, the salivation, apparent rather than real, may be found to result from *mechanical difficulties in swallowing*, the effect of such lesions as :—

- Mumps
- Acute tonsillitis
- Quinsy
- Fracture of the jaw
- Dislocation of the jaw
- Fixation of the jaw, as by osteo-arthritis of the temporo-maxillary joint
- Painful affections of the larynx, pharynx, or œsophagus.

In the absence of any obvious structural lesion locally, it may yet be clear that inability to swallow, owing to paralysis of some kind, is the cause of the apparent salivation, for instance in cases of :—

Bulbar paralysis
Pseudo-bulbar paralysis
Bilateral facial paralysis
Myasthenia gravis
Hypoglossal nerve paralysis
Diphtheritic paralysis
Paralysis agitans
Hydrophobia.

The differential diagnosis of these conditions is discussed elsewhere, and of them all it is only in bulbar and pseudo-bulbar paralysis that the dribbling of much saliva is a prominent symptom. The sequence of events summarized by the term labio-glosso-pharyngolaryngeal paralysis is sufficiently characteristic as a rule ; pseudo-bulbar paralysis, being of cortical instead of medullary origin, has not the wasting of the tongue that is prominent in the latter.

The salivation that results from *gastric* or *hepatic reflexes* is almost physiological, though sometimes it reaches a pathological degree in certain cases of :—

Dilatation of the stomach
Gastric ulcer
Duodenal ulcer
Acute dyspepsia
Acute gastritis
Gastric carcinoma
Biliousness
Hepatic disorder
Pancreatitis.

Mere slovenliness and lack of proper cerebral control are responsible for the slobbering and salivation of :—

Idiots
Imbeciles
Dements and other mental cases.

Finally, a remarkable degree of salivation can sometimes be attributed to nothing but functional disorder—*ptyalorrhœa.* It can scarcely be called hysterical, because it may occur in men as well as in women, generally in later life rather than at a time when hysteria is commonest. The condition is a sort of salivary neurosis, which may come on suddenly and without obvious cause, or as the result of some worry, shock, or mental emotion. It may possibly be a functional affection of the 5th nerve analogous to the far more distressing tic douloureux. It is sometimes prominent amongst the neuroses that are apt to accompany pregnancy. It can only be diagnosed when a careful examination has served to exclude any likelihood of organic disease, when the history is suggestive, and when the excessive salivation ceases after a time almost as suddenly as it began. In some male cases, notwithstanding the diagnosis being 'functional,' a high blood-pressure will be found, with other signs of arteriosclerosis, suggesting that errors in the circulation involving the vasomotor and other brain centres are responsible for the symptom.

Herbert French.

PULSATING TUMOURS.—(See SWELLING, PULSATILE, p. 693.)

PULSATION, UNDUE ABDOMINAL AORTIC.—Excessive pulsation of the abdominal aorta may occur in cases of aortic regurgitation, when all the arteries throughout the body may pulsate with undue violence. Apart from aortic regurgitation, however, it is nearly always an entirely functional disorder of the aorta. It occurs much more frequently in women than in men, the patients generally being unmarried or childless, between 20 and 40 years of age. They complain of pain in the abdomen, especially in the epigastrium ; a feeling of discomfort and distress ; a sensation of pulsation and throbbing over the abdominal aorta ; nausea, retching, sickness, and constipation ; they are usually thin, anæmic, extremely nervous, often hysterical, and sometimes decidedly hypochondriacal. There may be nothing else the matter with them at all, or they may be suffering from some other complaint of which much nervousness is a feature, exophthalmic goitre for example. The condition is frequently associated with movable kidney and enteroptosis ; the symptoms may suggest some organic disease, such as gastric ulcer, appendicitis, or ovaritis, without any of these being present.

On palpation of the abdomen the pulsation may be found to be forcible ; but the normal cylindrical outline of the aorta can generally be felt to be quite free from any saccular bulging or fusiform dilatation ; there is no thrill over it ; on applying a stethoscope lightly to the pulsating region no murmur will be audible, but firmer pressure, sufficient to compress the aorta slightly, will bring out a systolic bruit. The heart, lungs, and urine are usually normal. The knee-jerks are apt to be much exaggerated, though the plantar reflexes remain flexor, and there is no ankle-clonus. The chief importance of the condition clinically is that it is apt to be mistaken for an aneurysm of the abdominal aorta. Abdominal aneurysm is so extremely rare in women, however, that it should never be diagnosed unless the pulsation can be made out to be definitely expansile, or unless, in addition to pulsation, a definite swelling of the aorta can be felt. *Herbert French.*

PULSE, IRREGULAR.—No advance in clinical medicine is more striking than the change which has come over our view of the arrhythmic pulse during the past decade. Formerly we felt that our duty was finished when we had recorded the main facts: as to whether the force or the frequency varied, or both, and so forth. Now, however, it is possible to refer almost every type of irregularity to a definite cause, and thus to gain information of the utmost value for prognosis and treatment. To this end it is advisable that each case should be investigated by means of the recording polygraph, and the jugular and arterial curves compared.

The Mackenzie ink polygraph, the best of these instruments, consists of (1) A clockwork machine drawing a band of paper at a uniform rate over a flat surface; (2) Tambours for application to different parts of the body where there is superficial pulsation; (3) Levers connected by tubing with these tambours, carrying pens at their free ends which write upon the travelling paper; (4) A time-marking lever, also driven by clockwork, and marking five times per second. One of the tambours is applied to the radial artery, and the other to the internal jugular vein at the root of the neck as a rule. The result is a synchronous record of (i) time in one-fifth second intervals, (ii) the movements of the radial artery, (iii) the movements of the jugular vein. In the records from this last, each cardiac cycle exhibits three principal waves. By comparison with the radial trace the identity of the ventriculo-systolic wave (*c*) is established. The wave immediately preceding this is the auriculo-systolic (*a*), and the interval between its origin and that of the ventriculo-systolic, normally about ·2 second, is spoken of as the '*a-c* interval.' It constitutes an index of auriculo-ventricular conductivity. The third, or *v* wave, arises at the end of ventricular systole. The chief value of the method lies in the accuracy with which it enables us to study the time relations of auricular and ventricular systole.

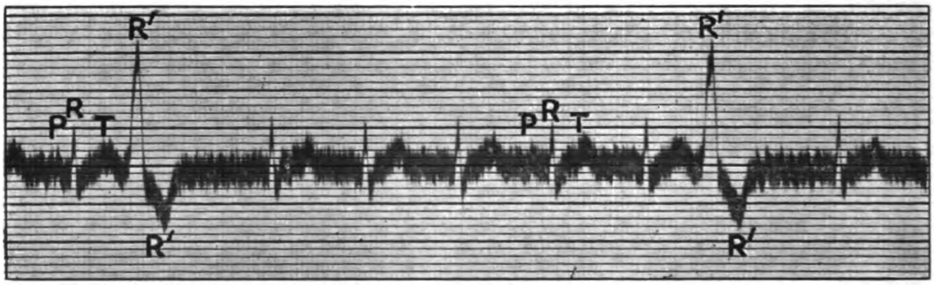


Fig. 231.—An electrocardiograph record showing ventricular extrasystoles. P. Auricular systolic phase. E. Main ventricular systolic phase. T. Terminal systolic phase. R′. Ventricular extrasystolic phase. This movement is premature, and its form (diphasic) indicates that only a part of the ventricle has contracted instead of the whole.

A still newer method, of great value in differential diagnosis, is by means of electrocardiograms. This method depends on the fact that contracting muscle is an electrical battery, the currents in which can be appreciated and their movements recorded by a sufficiently delicate galvanometer. The contracting heart thus generates currents which are led off to an extremely sensitive galvanometer, and the photographic records made from this can be interpreted in terms of cardiac movement (*Fig.* 231). Practically every one of the large medical schools throughout the country has one of these electrocardiographic outfits, so that there are few patients to whom this method cannot be applied if the practitioner feels that the accuracy of the information derived is so valuable as to justify the expenditure of time, trouble, and money involved. Many practitioners, however, are obliged to learn what they can about their patient's heart without these means, and in what follows this has been borne in mind.

First, it must be remembered that the heart generates its own stimuli; that these normally arise at the sino-auricular node, a relic of the primitive cardiac tube lying at the junction of the sinus venosus with the right auricle; that thence they pass through the auricular walls, and are conducted along the auriculo-ventricular bundles, narrow strips of muscle—also relics of the primitive cardiac tube—into the ventricular walls, one branch from the stem of the main bundle running to each ventricle; and that each portion of the cardiac muscle contracts as this stimulus provokes it to do so, the result being a co-ordinated and economical movement of the whole heart.

Pulse irregularities may be (1) *Perversions of the normal rhythm*; (2) *Rhythms originating abnormally.* Under the first heading fall (*a*) Irregularities of the whole heart—'sinus arrhythmias'—due to extracardiac influences acting through the vagus on the sino-auricular node; (*b*) Irregularities due to interference with the conduction of impulses through the heart, and especially with their passage from auricle to ventricle—'heart block'; and (*c*) Irregularities in the response of the musculature to the stimuli reaching it, of which the outstanding example is the 'alternating pulse.' Of abnormally originating rhythms there are three chief examples: (*a*) The simple extrasystole or premature contraction; (*b*) Paroxysmal tachycardia, and (*c*) The totally irregular pulse of auricular fibrillation.

1. Perversions of the Normal Rhythm:—

(*a*). *Sinus irregularities* are specially common in children and nervous subjects, in convalescence and acute illness, and in the presence of increased intracranial pressure. The difference in length between two sequent beats is never great, and sufficiently prolonged observation is nearly always rewarded by the discovery of a 'dominant rhythm'—i.e., a normal rhythm—from which the pulse departs from time to time. This type of irregularity is singularly apt to be exaggerated by excitement, and by bidding the patient swallow or hold his breath. As it does not depend on intracardiac causes, physical examination of the heart detects no sign of disease. It is generally possible to distinguish it from other forms of irregularity without instrumental help; but sometimes it is difficult to be quite sure that the arrhythmia is of this type, and if a tracing be taken it shows that auricular and ventricular systole are following each other in normal sequence.

(*b*). *Heart-block* is of various grades. The mildest type is that in which the interval elapsing between the start of auricular systole and that of ventricular systole is prolonged (*Fig.* 232, xx); this of course can only be detected by graphic records of the arterial and venous pulses. The next grade, in which some of the descending impulses are completely 'blocked' in their passage from auricle to ventricle, reveals itself in ordinary observation of the pulse as a dropping of beats; a gap, equivalent in length to two whole pulse-beats, separates one beat from that which preceded it. Here the auricle has contracted in the ordinary way, but since the stimulus which provoked that contraction has not passed into the ventricle, the latter has failed to contract (*Fig.* 232, x). This proves that there is some disease or disorder of the conducting apparatus. In higher grades of block, every third or every other stimulus, or even two out of three, or three out of four, may fail to pass over from auricle to ventricle (*Fig.* 233); so that the auricle may be beating 72 to the minute while the pulse counted at the wrist comes to 48, 36, 24, or 18 only. In the highest grade of all the auricle is completely dissociated from the ventricle, which assumes a rhythm of its own, usually at 30 to 40 per minute; thus the auricle is

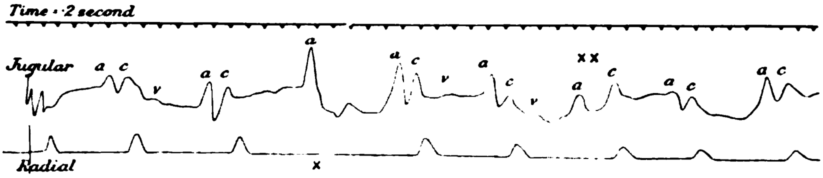


Fig. 232.—Incomplete heart-block. Note at x: (i) The radial pulse misses a beat entirely; (ii) The jugular pulse shows an *a* wave without a sequent *c* wave. This means that auricular systole occurred at the normal interval after the preceding cardiac cycle, but that ventricular systole failed to follow; the reason being that disease implicating the conducting paths hindered the normal transmission of stimuli from auricle to ventricle. Note at xx: The long *a–c* interval (about 0·4 second) indicating considerable though incomplete hindrance to transmisson of the impulse from auricle to ventricle.

beating regularly at one rate and the ventricle at another quite independent of that of the auricle.

To make sure that a dropped beat, or a slow regular pulse, is due to heart-block, it is therefore necessary to prove that the auricle contracted while the ventricle failed to follow suit. The only means of such proof, apart from the use of the polygraph, is furnished by observation of the venous pulse in the neck. If this can be seen to continue regularly, during the radial pauses, as well as immediately before the radial beats, then it is safe to assume that the condition is one of heart-block. It must be confessed, however, that this is very often difficult, and sometimes impossible, and that the observer is on much firmer ground if he obtains solid proof of his suspicion in the form of a graphic record. This will show that in the intervals when the pulse failed at the wrist, the wave which represents auricular systole made its appearance at the proper moment in the jugular curve (see *Fig.* 232). It is particularly desirable to obtain graphic evidence when it is a matter of accounting for occasional failures of the radial beat; for, as will be shown below, this grade of block may be simulated by extrasystoles too feeble to reach the wrist (see *Fig.* 234), and also in extreme exhaustion of contractility.

In the higher grades of block the patient often suffers from severe syncopal and epileptiform attacks, the coincidence of which with heart-block constitutes the Stokes-Adams syndrome. In these attacks the pulse usually becomes slower than ever. It is not quite safe, however, to conclude from the coincidence of such attacks with slow pulse that a lesion of conductivity is present; one or two cases have been described in which tracings have proved that the whole heart—auricle as well as ventricle—was slowed; i.e., that the rhythm was altered at its origin at the sino-auricular node and not in its passage through the heart. The distinction between this condition and that of heart-block is important, for the latter signifies organic disease of the heart, while the former does not; and their distinction cannot be safely founded except on the evidence of graphic records.

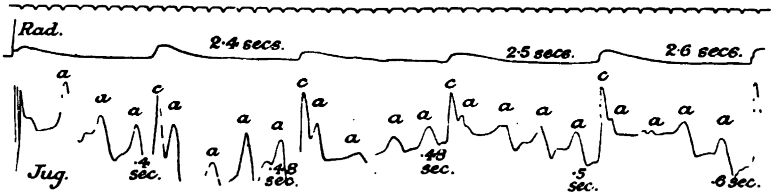


Fig. 233.—Heart-block. The ventricle only responds to every fourth stimulus descending to it from the auricle. The *a–c* interval is more than twice its normal length.

If the existence of heart-block be definitely proved, it points practically always to the existence of organic disease implicating the bundle which connects auricle with ventricle. Heart-block may arise in connection with acute infections such as diphtheria, rheumatism, influenza, pneumonia, or ulcerative endocarditis; or as part of a chronic disease such as gumma or cardiosclerosis. In any given case the cause of the block can only be determined by a general consideration of all its features.

(*c*). The term '*pulsus alternans*' is reserved for cases in which beats, equal in length, alternate in force; that is, for an alternation of a large beat with a small beat, but not of a long beat with a short beat. This phenomenon is an expression of exhaustion of the contractile power of the ventricle, and it is therefore of serious import. In some instances it can be detected clearly with the finger on the pulse; a full wave is followed by a small wave, and this again by a full wave, and so on, each beat being of equal duration. Often, however, the difference in the amplitude of the waves is so minute that it can only be detected by a tracing. Where alternation is suspected, confirmatory evidence may sometimes be afforded by a corresponding alternation in the intensity of the first cardiac sound, particularly if it be accompanied or followed by a bruit.

In connection with this form of irregularity, it is important to note that the smaller beat may sometimes be so feeble as to fail to reach the wrist; consequently the pulse appears to drop a beat.

2. **Rhythms Originating Abnormally**:—

(*a*). Turning to those forms of irregularity which are due to ectopic generation of stimuli, one finds that the simplest type, the *extrasystole*, is very common. It is associated with nervous states and with coronary sclerosis, and though it is of no great import, it is often productive of uncomfortable sensations which distress the patient ; it is therefore necessary to recognize its nature in order that a reassuring statement may be made. The extrasystole is " a premature contraction of auricle and ventricle in response to a stimulus from some abnormal part of the heart, where otherwise the fundamental rhythm of the heart is maintained " (Mackenzie).

The patient sometimes complains of feeling as if his heart had stopped, and then gone on again with a jerk ; or it may be that the jerk alone is felt. The observer, with his finger on the radial pulse, feels a small beat occur before its time, i.e., at a shorter interval after the preceding beat than the usual pause between beats. The beat which follows this premature one is generally more forcible than normal ; in some cases it follows after a compensatory pause, i.e., a pause of such length that it makes up for the brevity of the premature beat, the two together being equal in duration to two normal beats. It is the small premature beat which signifies the occurrence of an extrasystole (*Figs.* 231, 234). Confirmatory evidence of its nature may often be found in the heart-sounds ; simultaneously with the small premature beat at the wrist, a premature feeble pair of heart-sounds (or in some cases the first sound only) is heard.

It might be argued that if the extrasystole can be thus detected by ordinary methods of examination, and that if it has little positive significance in assisting to a full diagnosis of the case, it is needless to apply graphic methods to its elucidation ; and it is certainly true that it is of all forms of irregularity the one in which tracing is least essential. However, there are one or two considerations which may make a tracing advisable and even necessary : (i) The premature contraction may be so small that its beat is not felt at the wrist ; a dropped beat may be thus simulated, especially where the extrasystole is followed by a compensatory pause. In such cases heart-block may be wrongly diagnosed, unless clearer information be sought in a tracing. This will show the fault to be due to a feeble premature beat, and not to a blocked auricular impulse (see *Fig.* 234). (ii) Extrasystoles may occur in complexes, e.g., at every second or third beat. In the former case an alternation will result : a full beat is followed at a short interval by a small beat, and this after a long interval by a full beat, and so on (pulsus bigeminus). The true nature of such an arrhythmia is much easier to appreciate when it is seen mapped out in a tracing. (iii) A polygraphic record enables the observer to discover whether the premature beat arose in the auricle, in the ventricle, or in the junctional tissues. In the first case the jugular curve shows an auricular wave preceding the premature arterial beat at the proper interval ; in the second case there is no such auricular wave ; while in the third case, auricle and ventricle contract simultaneously, and the result is an abnormally large wave in the jugular curve. A knowledge of the source of extrasystoles may occasionally be of material value ; for instance, in mitral stenosis the final breakdown of the auricle is sometimes heralded by a series of extrasystoles of auricular origin. It is true that as a rule auricular extrasystoles are not followed by a compensatory pause, while those of ventricular origin are ; but it is not always easy to appreciate this distinction apart from a tracing, and, moreover, it does not invariably hold good.

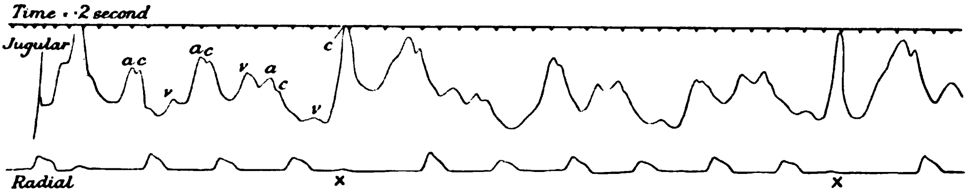


Fig. 234.—Ventricular extrasystole. Note at × a small premature radial beat : that this coincides with a large wave (c') in the jugular curve. (c') is therefore a ventriculo-systolic wave, and as it is not preceded by any auricular movement, it denotes a premature contraction, or 'extrasystole,' arising in the ventricle. Clinically this case simulated 'heart block' until the tracing made the truth plain.

(*b*). In *paroxysmal tachycardia*, as the term is used nowadays, the patient suffers from attacks of fast but regular cardiac action. The duration of these may vary from seconds to days; what is characteristic is the abrupt transition from the normal to the rapid rhythm and back again, at the onset and offset of the attack. The patient is conscious of discomfort, and indeed there is good cause if the speed be above a certain pitch; for in such a case the auricle gets no time for emptying itself, and becomes over-distended, pulmonary stagnation with cyanosis and venous engorgement resulting. Tachycardial paroxysms may appear on a background of organic disease, cardiosclerosis and mitral stenosis in particular; or the patient's heart may be normal. This form of irregularity has especially to be distinguished from that next to be described, the total arrhythmia of auricular fibrillation, which may manifest itself in paroxysms, particularly in its earlier stages. The distinguishing point is that in true paroxysmal tachycardia the pulse is regular, whereas in the other it is quite irregular.

By means of a polygraph tracing it is possible to discover the source of the rapid rhythm, whether it is auricular or ventricular, in most cases. The complexity of the subject of cardiac arrhythmia is, however, illustrated by the fact that there is a form of paroxysmal tachycardia arising in the auricles, which beat at great speed, up to and even exceeding 300 per minute, in which a certain proportion of the beats is blocked; so that only every second, third, or even fourth impulse passes over into the ventricle. That is to say, the auricle may be in a state of paroxysmal tachycardia, and yet the radial pulse is hastened but little or not at all. The difficulty of diagnosis is heightened by the fact that the auricular beats do not always appear in the jugular curve of a polygraph record, though they are manifest in an electrocardiogram. This form of tachycardia goes by the name of 'auricular friction.'

(*c*). The last form of irregularity to be recognized is in many senses the most important —the *total arrhythmia* which is associated with breakdown and fibrillation of the auricles. This is essentially a terminal phase of chronic organic disease, especially of mitral stenosis and cardiosclerosis; it may be prolonged for years, but nevertheless it marks a certain 'last state' of the auricular musculature. The features which characterize it are of two kinds: those which mark the disappearance of auricular systole, and the evidences of absolute irregularity of the pulse. To the observer using ordinary methods of examination without apparatus, this latter is the more obvious and striking side of the picture. If the pulse be counted for one or two minutes, it will be found that there is no 'dominant rhythm'; anarchy is complete. The pulse is usually hasty—over 100 per minute—but not always. The heart's action is similarly irregular, and on comparing it with the arterial pulse, a certain number of beats too feeble to reach the wrist will be discovered. Cessation of effective auricular systole is manifested in two ways if the case be one of mitral stenosis: by disappearance of the presystolic thrill and bruit (the diastolic vibration, however, persisting), and by disappearance of the auriculo-systolic wave from the jugular tracing. Of course, if the case be not one of mitral disease, there are no presystolic vibrations to disappear. However, the absolute disorder of the pulse, coupled with evidences of organic disease of the heart and a gravely embarrassed circulation, combine to form a picture so characteristic that there is little fear of a mistake. Nevertheless, there are cases of sinus irregularity so profound that without a polygraph tracing it is difficult to exclude auricular fibrillation; the same may be said of some few cases of multiple extrasystolic irregularity.

SUMMARY OF VARIATIONS IN RHYTHM OF PULSE.

I.—*The radial pulse may be wholly regular, but abnormally slow or abnormally quick.*

A.—**Regular Slow Pulse** may be due to—

1. *Extracardiac* causes (convalescence from acute illness, sinus irregularity, raised intracranial pressure, jaundice). In such, the whole heart is slowed, auricle and ventricle alike; there are no signs of cardiac disease; and there are signs of extracardiac disease.

2. *Intracardiac* causes. (*a*) *Alternating extrasystoles* may cause an apparent regular slowing of the pulse; if each 'normal' beat is followed by a premature one too small to be felt at the wrist, the radial pulse will appear slow. The real interpretation may be discovered by a polygraph tracing, and by comparing the heart-sounds with the pulse at the wrist; by these means the occurrence of the small premature beat after every normal beat will be observed. (*b*) *Heart-block*, either partial or complete, will render the radial pulse

slow. If the block be partial but regularly recurrent, the pulse at the wrist will be regular and slow ; e.g., if the auricle is beating at 72 per minute, and every other stimulus is blocked in its passage from auricle to ventricle, a regular pulse of 36 per minute will be the result ; while if two out of every three stimuli are blocked, the pulse will beat regularly 24 times per minute. If the block be complete, auricle and ventricle will each have its own regular rhythm ; that of the ventricle is usually at the rate of 30 to 40 per minute, and this will accordingly be the rate of the pulse. The occurrence of epileptiform and syncopal attacks is to a large extent confirmatory of the diagnosis of heart-block ; but this cannot be accepted as proved unless it has been demonstrated that when the auricle contracts, the ventricle sometimes or always fails to follow suit. This evidence is provided (i) by naked-eye observation of jugular, i.e., auricular, movement, (ii) by auscultation detecting auricular sounds during the ventricular pauses, (iii) by polygraph records, which not only afford conclusive evidence, but also facilitate the study of the degree of block present.

B.—**Regular Rapid Pulse** (and see TACHYCARDIA, p. 702) may be due to—

1. *Extracardiac* causes (tuberculosis and other infections, excitement, Graves's disease, etc.). Here the whole heart is persistently hurried, and the cause is usually manifest ; moreover, change to or from a slower rate is gradual and not abrupt.

2. *Intracardiac* causes. The only important intracardiac condition giving rise to a quick but regular pulse is that form of ectopic stimulus production which manifests itself in *paroxysmal tachycardia.* Here there may or may not be other signs of cardiac disease, mitral stenosis and cardiosclerosis being the commonest types ; the tachycardia is temporary, its onset and cessation being abrupt.

II.—*The radial pulse may show a fundamental regularity, occasionally interrupted by premature beats, intermissions, or periods of irregularity.*

A.—**Premature Beats** may be single or multiple ; they may recur at regular or irregular intervals ; the pause following them may or may not be compensatory ; the beat next following the pause is often increased in magnitude. The premature beat bespeaks an extrasystole ; it is a small beat, accompanied by heart-sounds feebler than those coinciding with the normal beats.

B.—**Intermission** of a beat or beats may be due to one of three causes : a premature extrasystolic beat too feeble to reach the wrist ; a blocked auricular stimulus ; or a comparative failure in the contractility of the ventricle.

1. If the intermission be due to a *feeble extrasystole*, the imperfect heart-sounds which accompany the premature beat will be audible. A sphygmographic tracing will sometimes bring to light a wave too feeble to be perceived with the finger. The pause due to the intermission is shorter than the duration of two complete beats, if the gap is due to an unperceived extrasystole which is not followed by a compensatory pause.

2. If it be due to a *blocked auricular impulse,* this can only be proved by demonstrating the occurrence of auricular systole without a corresponding ventricular systole following at the usual interval ; and it is scarcely possible to do this without a polygraph record of jugular and radial pulses. The auricular systole may be audible, and its effect visible in the jugular vein ; but in both cases timing is a difficult matter.

3. If it be due to a *normally timed ventricular systole too feeble to send a wave to the wrist,* other evidences of failing contractile power, among them the alternating pulse, are sure to be detected ; indeed, this type of intermission is in reality an exaggeration of the alternating pulse, without which it will not occur.

C.—**Short Periods of Irregularity** due to a ' sinus disturbance ' are very common, especially in children and nervous subjects. The diagnostic features are absence of physical signs of cardiac disease, marked variation of the rhythm with respiration and swallowing, and implication of the whole heart—auricle as well as ventricle—in the irregularity. This latter fact is of course only discerned by means of graphic records ; but the diagnosis can usually be made without recourse to these.

Longer periods of total arrhythmia (*v. infra*, IV, *A*), sometimes interrupt a normal rhythm.

III.—*The radial pulse is arranged in pairs of beats.*

A.—**The Pulsus Bisferiens** is a single beat with a double summit (*Fig.* 235). This can easily be recognized by the fact that the two summits are very close together, and that one cycle only of heart-sounds corresponds to each pair.

B.—**Alternating Extrasystoles** cause the pulse to be paired (pulsus bigeminus) ; each pair consists of a full beat, followed after an abnormally short pause by a small beat. Each beat, whether normal or abortive, is accompanied by a cycle of heart-sounds ; though the second sound may fail if the accompanying premature extrasystolic beat be too feeble to open the aortic valves. A sphygmographic or polygraphic record will help to make the nature of the irregularity clear.

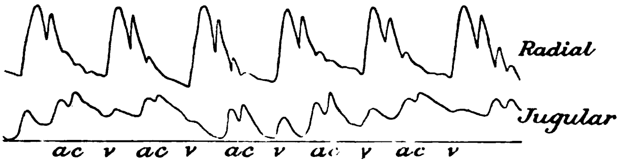


Fig. 235.—Pulsus bisferiens. There is a double peak to each radial beat, simulating a pair of beats.

C.—**The Pulsus Alternans** is associated with other evidences of inadequate contractile power. Each pair consists of a full beat and a small beat, and each beat is equidistant in time from that preceding and that following. A similar alternation in cardiac force may be detected in the heart-sounds. In many cases the alternation can only be recognized certainly by means of a sphygmographic tracing.

D.—**Blocking of every third Auricular Stimulus** results in the ventricular contractions being arranged in pairs. Each beat is of equal force, and the interval separating each pair from that following it is equivalent to the duration of two normal pulse waves.

Beats may fall regularly in groups of three or four if every fourth or fifth beat is either an extrasystole, or missing, owing to auriculo-ventricular block.

IV.—*The pulse is totally irregular,* no fundamental rhythm being discoverable.

A.—**True Total Irregularity** is associated with organic disease, either post-rheumatic or cardiosclerotic. The pulse and heart-sounds are altogether irregular, and the circulation is obviously embarrassed. The picture is therefore characteristic : if any doubt be present, discovery of evidences of auricular asystole (loss of presystolic thrill and murmur, failure of auriculo-systolic wave in jugular curve of polygraph tracing) will remove it.

B.—**Total Irregularity may be Simulated** by extreme sinus irregularity, by depression of conductivity leading to variations in the *a–c* interval, and also by multiple extrasystoles. In any such case all that is needed to establish a diagnosis is a polygraph tracing.

It is clear from this brief summary that most forms of arrhythmia can be recognized without the use of special apparatus ; but that in many cases a polygraphic record is valuable on account of the certainty which it gives to the diagnosis, while in a few it is impossible to be sure of the meaning of the irregularity without such a record. *Carey Coombs.*

PULSE, UNDULY RAPID.—(See Tachycardia, p. 702.)

PULSE, UNDULY SLOW.—(See Bradycardia, p. 82.)

PULSES, UNEQUAL.—Inequality of the pulses may be a perfectly natural phenomenon ; one frequently finds that the radial arteries of the two sides are not of the same calibre, owing to variable degrees of collateral circulation by an enlarged comes nervi mediani. Inequality of the pulses is a much more important sign when known to have developed in a patient whose pulses were formerly normal. In such a case the cause is probably one of the following :—

Thoracic aneurysm
Mediastinal new growth
Accessory cervical rib
Embolism
Atheroma.

Pathological inequality of the pulses, or definite delay of one behind the other, as gauged by simultaneous palpation of the two radial pulses, is distinctly uncommon, even in cases of thoracic aneurysm ; it is true that when very careful simultaneous records are made from the two radial pulses slight differences in size and definite differences in time can be detected, the one being delayed behind the other ; but in clinical medicine such minute methods of investigation are seldom applicable ; if the aneurysm involves the origin of the innominate artery, the right pulse will be smaller than the left; whereas if it affects that part of the arch from which the left subclavian artery is derived, the left radial pulse will be smaller than and delayed behind the right. Similar delay or inequality might be produced by *new growth* compressing either the innominate artery on the right side or the subclavian artery on either side; but this is rare.

An *accessory cervical rib* might stenose the subclavian artery, but the condition is generally bilateral so that it rarely produces inequality of the radial pulses; its symptoms are more likely to be those of interference with the lower part of the brachial plexus, with consequent pain, paræsthesia or paresis corresponding to the nerves distributed upon the ulnar aspect of the arms and hands (p. 443).

Embolism of one or other radial artery will rather obliterate it altogether than cause it to be less in size than that of the other side. It will almost always be due to fungating endocarditis (p. 34).

Atheroma of the brachial or subclavian artery on one side might cause the corresponding radial pulse to be less than that on the other; but this very rarely happens, and in such a case it would be more probable that atheroma of the aorta with an aneurysmal dilatation would be diagnosed than atheroma restricted to the vessels in the upper arm, unless the *x*-rays exhibited no trace of aneurysmal opacity in the thorax. *Herbert French.*

PUPIL, ABNORMALITIES OF THE.—Abnormalities of the pupil may be classified into:—I. *Irregularities in shape*; II. *Irregularities in size.*

I. **Irregularities in Shape.**—The normal pupil is circular or slightly oval. Its outline may become irregular owing to an adhesion between the iris and the lens, the result of *old iritis*. These adhesions are most evident when the pupil is dilated. A similar irregularity sometimes occurs with the *persistence of a pupillary membrane*—a congenital affection. The adhesions due to this cause may be distinguished from inflammatory adhesions by the fact that they arise from the anterior surface of the iris at a slight distance from the pupil and not from the posterior surface and the extreme edge.

The pupil may also become irregular in shape as the result of *injuries*, such as rupture of the sphincter, and tearing of the root of the iris from its ciliary adhesion (iridodialysis); of dislocation of the lens; or of partial adherence to an old perforated corneal ulcer.

II. **Irregularities in Movement and Size.**—Before considering the irregularities in the movements and size of the pupil, it is desirable to remember that its normal size varies during life. In extreme infancy it is small. It becomes larger during young adult and middle life, and ultimately becomes small again in old age. It is also, as a general rule, small in hypermetropic, and large in myopic eyes.

There are also four normal pupillary reflexes; (i) The light reflex; (ii) The reflex to accommodation; (iii) The reflex to sensory stimulation; (iv) Psychic reflexes. The reflexes to light and to accommodation are both constrictive, the constriction in accommodation being more in the nature of an associated muscular action. The sensory and psychic reflexes are both dilatations, the dilatation being caused by either sudden sensory stimuli or some sudden emotion, such as fright or terror.

The pathological variations in the pupil may be classified as follows:—

1. *Loss of the Pupillary Light Reflex*, either with or without constriction of the pupil, but with persistence of the reaction to accommodation, constitutes the Argyll Robertson pupil. It never occurs in healthy individuals, but has been observed most frequently in *tabes dorsalis*, to an extent varying according to different observers from 70 to 90 per cent of all the cases. The condition is usually permanent. It also occurs in *general paralysis of the insane*. The pupil is constricted in nearly all tabetic cases, and the affection is most commonly bilateral.

2. *Loss of Convergent Accommodation Reflex and Retention of the Light Reflex.*—This condition is extremely rare, but has been observed in *syphilis*, basal *meningitis*, *myelitis*, and *tumour of the corpora quadrigemina.*

3. *Loss of the Convergence Pupillary Reflex* may be unilateral or bilateral. It occurs, rarely, in *tabes dorsalis*, and after some cases of *diphtheria* and *alcoholic intoxication*.

4. *Loss of all Reflex Movements of the Pupil.*—In this condition there is paralysis of the sphincter of the pupil and of the ciliary muscle, the extrinsic muscles of the eye being unaffected (ophthalmoplegia interna). The site of the lesion must be in the third nerve

nucleus, and it is most frequently unilateral, though occasionally bilateral. *Syphilis* is the most frequent cause. It may also occur after *diphtheria*, *injury*, or in some intracranial diseases.

5. In the condition in which there is a lesion of the optic nerve on one side, between the chiasma and the globe, there will be, as a result, a loss of direct light reflex in that eye, and of the consensual light reflex in the opposite eye.

6. *Loss of Sensory* or *Psychic Reflex* occurs in lesions of the dilatator pupillary tract, such as *paralysis of the cervical sympathetic*: in which condition it is associated with slight ptosis of the upper lid, enophthalmos, and diminished tension of the globe.

7. *Abnormal Constriction of a Pupil, with Retention of the Light and Convergent Reflexes,* may occur from abnormal stimuli of the sphincter, or paralysis of the dilatator pupillæ as the result of acute *encephalitis*, *intracranial abscess*, or *growth*, in which the lesion irritates but does not destroy the centre for convergence. In all cases of brain disease the constriction is ultimately replaced by dilatation.

8. *Abnormal Dilatation of the Pupil. with Retention of the Light and Convergent Reflexes,* is met with in cases of stimulation of the cervical sympathetic, for instance by an aortic *aneurysm*. It may also be observed in certain mental states, such as *epilepsy*, acute *mania*, or *catalepsy*.

9. *Inequality in the size of the Pupils* is observed frequently, and may have no pathological significance; but pronounced difference in the size of the pupils is nearly always symptomatic of some organic lesion. In cases where the abnormal pupil is the smaller, the condition is usually due to hyperæmia of the iris, such as occurs in *iritis*; *paralysis of the cervical sympathetic*; or the use of a myotic drug such as *physostigmine*. In cases where the abnormal pupil is the larger, the dilatation is usually due to stimulation of the sympathetic, the use of a mydriatic, paralysis of the fibres of the third nerve, or increased ocular tension, such as may occur in *glaucoma*.

In cases of inequality of the pupils one may suspect tabes, general paralysis of the insane, a unilateral lesion of the third nerve or cervical sympathetic, trigeminal neuralgia, carotid or aortic aneurysm, a unilateral intracranial lesion, or glaucoma.

10. Irregularities in the shape of the pupils other than those mentioned above may occur in *tabes* and various cases of *insanity*. There is no marked or sharp irregularity, it only being noticed that the pupil is not circular owing to paralysis of certain fibres of the iris.

11. *Hippus.*—This term is applied to a condition in which, when both eyes are shaded, and then illuminated, the pupils will alternately dilate and contract. It is sometimes associated with nystagmus, and occurs also in *disseminated sclerosis*, and in some cases of *brain tumour*. It is observed most frequently when there is a central scotoma in the field of vision, with some injury to the macular or axial fibres of the optic nerve. It is also common in *alcoholic subjects*.

12. *Paradoxical Pupillary Reflex*: pupils dilating under the stimulus of light. This condition is extremely rare, and has only been observed in patients affected with grave lesions of the central nervous system, usually *tabes dorsalis*.

13. *Hemianopic Pupillary Reflex*: lesions of the brain situated in the optic tract above the corpora quadrigemina may give rise to partial loss of vision, but will not affect the pupil-reflex arc. For example, a lesion in the right occipital cortex may give rise to a left homonymous hemianopsia, but the pupil will react even when a light is thrown on the blind side of the retina (see HEMIANOPSIA, p. 300). In cases, however, where the lesion is situated in the optic tract below the corpora quadrigemina, hemianopsia may also occur, but under these circumstances no pupillary reflex for light can be obtained on stimulus of the blind side of the retina, the pupil reacting to light when the opposite side of the retina is stimulated. This reaction is termed the hemiopic pupillary reflex, and is of great value in the localization of intracranial lesions.

Herbert L. Eason.

PURPURA signifies hæmorrhage into the skin, and, according to the size of the extravasation of blood, the lesions are spoken of as puncta or spots, vibices or lines, petechiæ or small patches, ecchymoses or bruises. The lesions cannot be obliterated by pressure with the finger, which distinguishes the effused blood from mere congestion. The diagnosis of the actual fact of purpura is seldom difficult; the persistence of the discoloration under pressure differentiates it from erythematous lesions, and the colour generally serves to distinguish it from pigmentation of the skin other than that due to hæmorrhage. In a case of doubt, the fact that the lesions presently alter in colour and then disappear serves to distinguish purpura from capillary nævi or from pigmentation of the skin, which persist. It may be more difficult, however, to decide what is the nature of the purpura in any given case; the following is a list of its better-recognized causes:—

CAUSES OF PURPURA.

1.—**Due to Local Injuries:**

- Flea-bite
- Pediculosis
- Leech-bite
- Blows
- Sprains
- Rupture of a muscle
- Rupture of a vein, especially a varicose vein.

2.—**The Effect of Drugs and Poisons:**

- Antipyrin
- Iodoform
- Iodide of Potassium
- Sulphonal
- Copaiba
- Belladonna
- Chloral
- Chloral hydrate
- Butyl-chloral hydrate
- Veronal
- Mercury
- Arsenic
- Quinine
- Ergot
- Salicylic acid
- Potassium chlorate
- Diphtheritic antitoxin
- Ptomaine
- Snake-bite poison.

3.—**In Fevers:**

- Typhus fever
- Cerebrospinal fever
- Small-pox
- Pyæmia
- Septicæmia
- Fungating endocarditis
- General tuberculosis
- Dysentery
- Cholera
- Yellow fever
- Weil's disease, or bilious typhoid
- Plague
- Remittent fever
- Severe malarial fever
- Blackwater fever
- Measles
- Diphtheria
- Typhoid fever
- Scarlet fever.

4.—**In association with Jaundice from whatever cause** (see JAUNDICE, p. 324).

5.—**Bright's Disease.**

6.—**Chronic Alcoholism:**

- Cirrhosis of the liver
- Peripheral neuritis

7.—**The so-called Blood Diseases:**

- Splenomedullary leukæmia
- Lymphatic leukæmia
- Lymphadenoma
- Pernicious anæmia
- Splenic anæmia
- Pseudo-leukæmia infantum
- Scurvy
- Barlow's disease.
- Hæmophilia

8.—**In cases of generalized Malignant Disease,** especially:

- Sarcomatosis
- Chloroma

9.—**Peliosis Rheumatica.**

10.—**Henoch's Purpura.**

11.—**Purpura Simplex.**

- Morbus maculosus of Werlhof
- Purpura hæmorrhagica
- Purpura fulminans.

A number of the above conditions require but little discussion, for if they are only borne in mind their diagnosis will generally be easy.

Flea-bites are by far the commonest cause of purpura in the out-patient department of a hospital, and they may sometimes be so numerous as to raise a misleading suspicion that the patient is suffering from some serious disease. The relatively small hæmorrhagic foci, and their prevalence on the parts covered by the clothes rather than upon the hands, face, or exposed parts of the legs, serve to indicate the diagnosis, even in a severe case.

The commonest variety of *pediculosis* to produce purpura is *P. corporis* or *vestimentorum*; the circumstances of the case and the distribution of the purpura itself and of the marks of scratching, particularly in the regions where collars and other constrictions in the dress occur, would indicate the diagnosis.

The hæmorrhage around a *leech-bite* is so characteristic that, once seen, it cannot be mistaken for anything else.

Blows and *sprains*, if sufficiently severe, produce purpura even in the healthy, in whom the history gives the diagnosis ; it is important to bear in mind, however, that some normal individuals bruise with such ease that there may be no clear evidence of injury unless careful inquiry is made, when some trivial stumble or knock may be recalled to mind by the patient. Such easy bruising may also occur in any of the blood diseases. A case of *epilepsy* may sometimes come under observation for multiple bruises simulating some other kind of purpura, but due to injuries produced during the attacks, which may themselves be unsuspected if they occur during the night. Very extensive purpura on the legs or other parts has sometimes been produced by multiple self-injury in girls suffering from *hysteria* or by *malingerers* ; serious organic disease may be feared unless other factors in the case or the distribution of the purpura suggests an artificial origin ; the hæmorrhagic spots and blotches may be abundant on the fronts and sides of the legs, for instance, and not down the backs of them ; no known organic purpura has so selective a distribution.

Spontaneous *rupture of a muscle* leads to extensive purpuric extravasation of blood, but the diagnosis is not difficult if the history is clear, and one can often feel the place where the muscle has given way unless it is too deep-seated to palpate, as in the case of the plantaris longus, which is apt to rupture during sudden efforts such as may be made in playing tennis or the organ.

The *purpuric discoloration of the skin around varicose veins* in the legs, together with its resultant dark-brown pigmentation, is familiar to all.

As regards *drugs*, the list above indicates that there are many which may sometimes produce purpura ; it may be said at once, however, that none do so at all commonly. Nevertheless the possibility should be borne in mind, and inquiry made as to the remedies the patient may have been taking. *Antidiphtheritic serum* and *ptomaines* merit particular attention. The commonest eruption resulting from antidiphtheritic serum, or from other forms of antitoxic horse-serum administered hypodermically, is urticaria ; purpura is relatively rare ; either form occurs as a rule about nine or ten days after the serum has been given, and is generally associated with lassitude, muscular and joint pains, anorexia, and more or less pyrexia, lasting from a few hours to two or three days. Ptomaine poisoning is often difficult to recognize with certainty unless it occurs in epidemic form. It is due in most cases to the products of Gaertner's bacillus, and seeing that the blood-serum of patients affected by this bacterium develops agglutinating powers against it similar to Widal's reaction for typhoid fever, this serum test should not be omitted ; if it proves positive the diagnosis is clear ; a negative result, however, does not exclude ptomaine poisoning.

In the majority of *acute fevers*, the occurrence of purpura is of prognostic rather than of diagnostic value ; in diphtheria, for example, even a single well-defined purpuric spot is a sign of grave omen, but it does not assist at all in the diagnosis of the disease, which has to be recognized upon other grounds. The same applies to measles, scarlet fever, and so on ; indeed, the only two fevers in which purpura is of essentially diagnostic value are *typhus* and *cerebrospinal fever*. The former is now very rare in Great Britain, but when it was common and typhoid fever began to be differentiated from it, the point upon which greatest stress was laid was that in true typhus or gaol fever there is always more or less purpura, whereas in typhoid fever all the red spots fade upon pressure. It happens occasionally even yet, that typhus fever develops in the poorest parts of cities, and this point is most useful in distinguishing it from typhoid. In the latter, if flea-bites are excluded, purpuric spots are exceedingly rare. *Cerebrospinal fever* presents many characters that are common to it and to other forms of acute meningitis ; but if with these there is a purpuric eruption, it is at once differentiated from the others, though the absence of purpura does not exclude the disease. So characteristic is the purpura in some cases that the malady has earned the title of spotted fever, which used to occur in widespread epidemics, and still does in smaller ones from time to time. The diagnosis may be clinched by bacteriological examination of the cerebrospinal fluid obtained by lumbar puncture.

Small-pox may present cutaneous hæmorrhages of three different kinds ; there may be hæmorrhage *into the pustules* in a late stage, when the diagnosis has already been made and when the prognosis is not thereby made worse ; there may be hæmorrhage *between the pustules*, vesicles, or papules, the diagnosis having already been made, in which case the prognosis is not good ; and there may be a hæmorrhagic eruption either all over the body or in the bathing-drawers region in the *prodromal* stage of the disease, in which case the patient will almost certainly die before the true small-pox eruption develops, so that if there is not an epidemic at the time the diagnosis may be exceedingly difficult.

Almost any condition in which there are pyogenic micro-organisms or their toxins circulating in the blood-stream may be associated with extensive purpura, and this applies to *pyœmia* and *septicœmia* in general. The diagnosis will be confirmed best by obtaining cultivations from the blood, though should blood-cultures prove negative septicæmia will not be excluded necessarily ; there will very likely have been rigors, pyrexia, and other symptoms pointing to the nature of the case. *Fungating endocarditis* is only a variety of pyæmia or septicæmia. Seeing that it is very rare to get purpura in association with chronic valvular disease of the heart if both purpura rheumatica and infective endocarditis can be excluded, the occurrence of purpura in a heart case (*Fig.* 236) may be one of the main symptoms indicating that fungating endocarditis has supervened. So indefinite is the nomenclature in regard to this disease, that the terms malignant, ulcerative, fungating, infected, and infective endocarditis are used indiscriminately by different observers to denote the same condition. The disease may be further indicated by symptoms described on p. 34. *Pyorrhœa alveolaris* is another septic infection which leads occasionally to extensive purpura and other hæmorrhages.

General tuberculosis is not a common cause of purpura, and yet in a few instances extensive purpura has been the first, and for the time being the only, symptom of an obscure illness which has ultimately turned out to be general tuberculosis. The patient has generally been a child, and the diagnosis has only been possible when the course of the case has been watched. The same may be said of *sarcomatosis* in certain cases, though this has been included under a different heading in the above list.

Jaundice should be borne in mind as a cause of purpura, for although the occurrence of the latter does not assist in differentiating one kind of jaundice from another, one might be misled into diagnosing something more serious than is necessary if one did not bear in mind that any kind of jaundice may produce purpura. Moreover, some of these patients may seem to have been grossly ill-treated if one were to judge only by the degree of bruising that may result from ordinary palpation ; the danger of fatal oozing after operation is always to be remembered when surgical measures are thought of in a jaundiced subject. Spontaneous hæmorrhage into the skin is less common here than is hæmorrhage from what otherwise would be trivial causes.

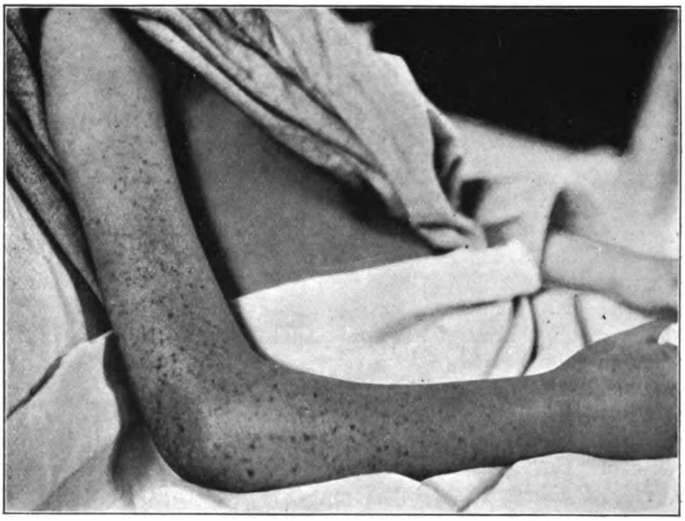


Fig. 236.—From a photograph showing extensive purpura in a case of fungating endocarditis. The limbs were affected more than the trunk. From the same case as *Fig.* 270, p. 640.

Bright's disease, particularly the chronic varieties, may produce hæmorrhage anywhere in the body. Purpura is not a very common form of such hæmorrhage, but when it does occur it may be extensive. The diagnosis is discussed under ALBUMINURIA (p. 6).

Chronic alcoholism, especially if it has already led to either cirrhosis of the liver or to peripheral neuritis, is occasionally a cause of considerable purpura, though the latter is generally confined to the legs, particularly to the parts below the knees. In many instances the diagnosis is easy, even if the history is not given with perfect honesty ; but considerable difficulty sometimes arises in the case of ladies who have contracted the habit of secret drinking, their relatives and friends being entirely unaware of it.

Any of the so-called *blood diseases* may present purpura as a prominent symptom, and in some cases, particularly in *lymphatic leukœmia* in children, extensive purpura may be the first symptom that anything is wrong. More often, however, the disease has already given rise to anæmia or to enlargement of the spleen or lymphatic glands, or to some other prominent hæmorrhage, and the diagnosis has already been made by the time the purpura supervenes. (See ANÆMIA, p. 20; SPLEEN, ENLARGEMENT OF THE, p. 628; and LYMPHATIC GLAND ENLARGEMENT, p. 376.)

Scurvy in an adult is relatively rare, but is sometimes met with in those who have been obliged by poverty to live upon a diet containing no fresh vegetables; a man may try to live for a month or more on plain bread, in which case typical scurvy may develop in him, with the spongy heaping up of the gums both inside and outside the teeth, and with the knotty hæmorrhagic swellings in the muscles of the calves, as well as purpura. Children who are fed upon patent foods without sufficient fresh milk, or vegetable food, or fresh meat, not infrequently develop a milder form of scurvy, with marked tenderness of the periosteum of the long bones, pasty pallor, mouth bleeding from spongy gums, and possibly purpura; this is *infantile scurvy*, or *Barlow's disease*, which should not be confused, as it is apt to be, with rickets.

Hæmophilia is generally indicated at once by the history of persistent oozing from slight cuts and scratches, and also by the fact that other members of the family, especially males, have suffered in a similar way.

Chloroma is a very rare disease, in some ways related to sarcomatosis, and in others to lymphatic leukæmia; it produces swellings in connection with the bones, especially of the head, together with enlargement of the lymphatic, lachrymal and salivary glands; it develops in early life, proves slowly fatal, and the diagnosis is confirmed by the green colour of the new-formed tissue—'green cancer.' The blood changes are negative.

Peliosis rheumatica, or *purpura rheumatica*, or *Schönlein's disease*, was formerly regarded as related to acute rheumatism; but it is exceedingly rare for a patient affected by it to present unmistakable signs of valvular heart disease, although there may be a local systolic bruit at the impulse. The reason why it is thought to be related to acute rheumatism is, that in addition to the extensive purpura, which comes out in successive crops and may affect any part of the body, though it is commoner upon the lower limbs than elsewhere, there is considerable pain, redness, and swelling of many joints, which may become affected successively; the temperature rises during an attack to 103° F. or 104° F., the throat generally being sore at the same time. It is not impossible that the purpura is due to the absorption of microbes or their toxins from the acute tonsillitis; but be this as it may, the diagnosis is not difficult when the purpura, the joint pains, and the pyrexia are present together. The disease is little influenced by sodium salicylate; it may be associated with more or less erythema as well as purpura; the malady affects young persons, especially between the ages of ten and thirty, of either sex.

Henoch's purpura is met with chiefly in children (*Fig.* 157, p. 345), and the same patient may suffer from recurrent attacks, which usually cease at or before puberty. In addition to hæmorrhages beneath the skin, there is generally some tendency to joint pains not unlike those of peliosis rheumatica, but in addition to this the child is seized with more or less severe acute abdominal symptoms, varying from simple vomiting and stomach-ache to severe prostration with agonizing cramp-like attacks of colic, some of which may be followed by the passage of blood and mucus per rectum to such an extent as to simulate acute intussusception; the abdominal attacks are probably the result of submucous intestinal hæmorrhages. There is every degree of the affection, from mild to very severe, but the association of the purpura with the abdominal attacks in childhood suggests the diagnosis at once, especially if there has been a similar attack previously. The chief error to avoid is mistaking for Henoch's purpura that which is really an acute nephritis; the urine should be examined periodically for albumin and renal tube-casts, even if there is no œdema, though the occurrence of blood alone would not be sufficient to indicate acute nephritis, seeing that hæmorrhage from the kidney may be due to Henoch's purpura itself.

It is only when every precaution has been taken to exclude all the above causes of purpura that one can be satisfied with any of the remaining three diagnoses, namely *purpura simplex* (*morbus maculosus* of Werlhof), *purpura hæmorrhagica*, or *purpura fulminans*. These differ from each other only in degree; broadly speaking, purpura simplex signifies hæmorrhage into the skin only; purpura hæmorrhagica has, in addition, hæmorrhages from the mucous membranes, particularly of the mouth, nose, and bowel, less commonly of the urinary passages; whilst purpura fulminans is the term used to denote a condition in which a person may seem perfectly healthy to-day, may be seized with acute purpura and be dead before to-morrow, without developing any other symptoms to indicate the nature of the complaint. These kinds of purpura have sometimes been spoken of as idiopathic, but they must have some underlying cause, if only it can be found. It is better probably to label them cases of purpura of which the exact cause is not yet known, than to be content with such a term as purpura simplex, and it is probable that if bacteriological examinations were made, a bacterial cause would be discovered, particularly in connection with the tonsils, the gums in states of septic gingivitis or pyorrhœa alveolaris, the uterus, the bowel, or the circulating blood itself. *Herbert French.*

PUS IN THE CHEST.—(See Chest, Pus in, p. 103.)

PUS IN THE STOOLS in sufficient amount to be recognizable by the naked eye, indicates the rupture of an abscess into the intestinal tract. The symptom is rare, however, for even when a large appendicular abscess perforates into the cæcum, the pus either becomes indistinguishable when mixed with the fæces, or unrecognizable on account of digestion and decomposition. The less the pus is mixed with other intestinal contents, the nearer to the anus has the site of rupture been; but the diagnosis of the source of the abscess needs to be determined upon other grounds, particularly the history, and the results of general physical examination, including that of the rectum and vagina. Abscesses most apt to cause a discharge of pus with the stools are of the appendicular, cholecystic, perinephric, psoas, pelvic, perigastric, or other local peritoneal types, and pyosalpinx.

Microscopical quantities of pus in the stools may be due to any of the causes already mentioned; they may also be derived, not from lesions outside the intestines, but from affections of the mucous membrane itself: acute or chronic colitis, with or without ulceration; dysentery; cholera; dengue; mucous or mucomembranous colitis; tuberculous, typhoidal, malignant, or venereal ulceration of the bowel. The pus corpuscles may be recognizable as such under the microscope; but it is difficult to determine when the leucocytes derived from the intestinal catarrh are merely leucocytes in excess, and when their numbers become sufficient to merit the term actual pus. Examination with the sigmoidoscope is sometimes invaluable when the diagnosis has not been decided by other methods. *Herbert French.*

PUSTULES.—The pustule, one of the primary cutaneous lesions, is an epidermic elevation, either unilocular or multilocular, containing a purulent liquid, and differing from a vesicle or a bulla only in the character of its contents. Always a product of inflammation, it may originate as a pustule or may develop from a papule, but much more often it is a transformed vesicle; if the metamorphosis is imperfect the lesion is styled a papulo-pustule or a vesico-pustule. Frequently the transformation from a papule or a vesicle is so swift that the true origin of the lesion may escape notice; but in such cases it is usual to find papules or vesicles intermingled with the pustules. The pustular cavity may be situated in the epidermis, in the derma, or in a follicle; a purulent accumulation *beneath* the derma is either an abscess or a gumma. *Epidermic* pustules may be superficial, as in impetigo, or deep, as in the condition known as ecthyma, which I regard as but a severe form of impetigo. *Dermic* pustules, such as the miliary abscesses of new-born children, are seldom met with, while *follicular* pustules, such as those of sycosis, are common. In *colour*, pustules are usually yellowish or greyish, with a red areola; but when the contents are mixed with blood, the yellow may be tinged with red or brown. If a pustule is punctured or ruptured, the liquid is seen to be more or less turbid and yellowish; under the microscope it is found to consist largely of leucocytes and serum as well as cocci. Pustules vary greatly in size: small ones may remain of inconsiderable dimensions, or may become large by excentric extension. The prevalent *shape* is roundish or convex, as in furuncle and acne; but it may be acuminate, as is frequent in sycosis and eczema, or flat and irregular, as in impetigo and syphilis, while in rare instances, as sometimes in scabies, it may be oblong, with a tendency to the linear form. In variola and the varioliform syphilide, the pustules may be flattened or concave, either because the fluid may not fully distend the cleft in which it lies, or from flaccidity of the sac due to commencing absorption. Pustules may develop slowly, as sometimes occurs in impetigo and in the pustular syphiloderm ; but as a rule they run a rapid course, and terminate either by rupture—much more often accidental than spontaneous—or by desiccation. In either case, a yellow, brown, or blackish crust, more or less thick and irregular, is formed ; but if the termination is by desiccation, the crust has a less pronounced coloration, and is friable instead of firm.

A cutaneous affection in which the pustule plays a leading part is *impetigo vulgaris seu contagiosa* ; usually following slight febrile disturbance, small erythematous spots appear, on which form vesicles containing a turbid fluid that quickly becomes purulent ; when the pustules break, as they soon do, they discharge a fluid that dries up quickly into scabs that are at first yellowish, and afterwards green. Dotted about among the scabs are pustules, which may coalesce so as to form, on rupture, crusts of considerable size. The eruption may be limited to a few discrete lesions, or may extend over large areas of the body. In parts where the pustules are exposed to friction, as on the limbs, they are generally ruptured at an early stage, and a flat irregular scab, surrounded by an areola, forms over them—the condition known as ecthyma. Sometimes the distribution is annular (*impetigo circinata* or *gyrata*). In the condition known as *impetigo bullosa* the lesions are much larger, and are not always transformed into true pustules. With impetiginous eruptions, though not with these alone, *cutaneous diphtheria* is sometimes associated, especially in children. The most typical form of this affection has the appearance of an impetiginous eczema, associated with conjunctivitis, and occasionally with otorrhœa and rhinitis. No diphtheritic membrane may be present, and if cutaneous diphtheria is suspected, the Klebs-Löffler bacillus should be sought for bacteriologically. Staphylococci, or streptococci and staphylococci together, are the organisms generally found in simple impetigo, but occasionally other germs produce a precisely similar eruption—the *Bacillus coli communis* for example, or the *Bacillus pyocyaneus.*

The differences between impetigo vulgaris and *follicular impetigo* are well marked. The latter is pustular from the beginning, and always situated around a hair-follicle. It starts as a round pustule, often pierced by a long or coarse hair, and it may be quite small, or as large as a pea ; the pus collects under the horny layer, which it distends and raises. The eruption, usually multiple, has no sites of election, but appears wherever a breach in the horny layer affords entrance to the pyogenic organism. The pustules are more resistant than those of impetigo contagiosa, and are less quick to break. When they rupture, yellow crusts, smaller and thinner than those of impetigo vulgaris, are formed. The pustules of follicular impetigo can hardly be mistaken for those of any other affection. The other form of impetigo, however, has in rare cases to be diagnosed from pemphigus. In the latter the lesions start, not as small vesicles but as bullæ, and the fluid they contain is only sometimes inoculable. Usually, too, there is marked systemic disturbance. But it is with *pustular eczema* that impetigo vulgaris is most likely to be confused, especially when the pustules of the latter condition have run together into a patch. In eczema, however, the pustules are smaller, there are severe itching and burning, there is an inflammatory areola around the crusts, which is seldom the case in impetigo vulgaris, and other definitely eczematous lesions will usually be found if sought for carefully, including infiltration and thickening of the integument.

Like follicular impetigo, *sycosis vulgaris* is a staphylococcic infection. The lesions begin as papules, or as nodules which form round the hairs—usually of the face, and especially of the chin, but sometimes attacking the eyebrows, eyelashes, and the axillary and pubic regions—and presently develop into pustules, each of them pierced by a hair. As the result of suppuration, the hairs are loosened, and if one is pulled out, a drop or two of pus usually exudes. In severe cases the pustules may be packed so closely together as to form infiltrations, which may fungate. The chief diagnostic features of the affection are its inflammatory character, its origin in the hair-follicles, and its limitation to the hairy parts, usually of the face. The differential diagnosis from tinea sycosis has been given under FUNGOUS AFFECTIONS OF THE SKIN (p. 246). Eczema is not limited to the hairy parts, and if the follicles are involved it is only secondarily, nor, as a rule, is the inflammation so severe as in sycosis vulgaris. Of sycosis vulgaris again, intense itching is not a feature. Sometimes, when the sycosis is widely diffused, the crusts may have to be removed to clear up the diagnosis; when this is done, the follicular implication will soon be perceived. Tertiary syphilitic ulceration is not restricted to the follicles, and behind it there lies a history of earlier specific lesions, as well as of the primary infection, unless this should have escaped notice. Wassermann's serum test may be applied.

If there is ever any doubt as between sycosis vulgaris and acne vulgaris, the presence of the latter on non-hairy parts should of itself suffice to decide the question. The pustules of *acne vulgaris* can scarcely, indeed, be confounded with those of any other affection, except with the lesions of small-pox (see below) and those of bromide and iodide eruptions. In these drug eruptions, however, comedones are absent, the lesions occur on any part of the body, and are generally a brighter red, while the fluid they contain is rather thinner. Drug eruptions, again, occur at any time of life, whereas acne vulgaris is essentially a disease of puberty. Pustular syphilides may attack any part of the body, and are generally grouped, which is never the case with the pustules of acne.

A *furuncle* is so characteristic that the only lesion from which it can ever require to be differentiated is a *carbuncle*. The pathological process is the same in both; but while in furuncle there is but one point of suppuration and opening, in carbuncle there are several. The only condition from which a carbuncle has in turn to be diagnosed, except a furuncle and malignant pustule, is diffuse cellulitis, in which there is no circumscribed outline.

In *malignant pustule* (anthrax), following itching and burning at the site of inoculation, a livid red papule appears, on which a bulla or pustule forms quickly and breaks, drying up into a black gangrenous eschar. This is fringed with tiny vesicles or pustules, and surrounded by a broad areola of solid œdematous infiltration, the skin over which is tense and violaceous. There are constitutional symptoms, with septic fever. The diagnosis rests mainly upon the presence of a gangrenous patch surrounded by infiltration in a patient whose occupation exposes him to infection with the anthrax bacillus, especially from cattle, hides, or wool. The organism (*Plate XXVIII*, p. 614) may be detected without difficulty under the microscope. It is a relatively large bacillus which generally forms long chains and is Gram-positive. It is only at the outset that the lesion can be mistaken for a carbuncle. The primary lesion of syphilis can be excluded by its indolence, and by the absence of gangrene and of febrile symptoms.

In *glanders*, the cutaneous lesions begin as red spots, which pass through the papular and vesicular or bullous stage into pustules that give rise to widespread ulceration. The condition, with its severe constitutional disturbance and, except in some chronic cases, the peculiar discharge from the nostrils, is usually easy of recognition; and in exceptional cases in which the diagnosis is in doubt, recourse should be had to the mallein injection test, or the *Bacillus mallei* may be isolated from the lesions.

In *scrofulodermia* (tuberculides), usually an affection of childhood and adolescence, pustular lesions take the form which has been styled by Dühring the large flat pustular, and the small pustular scrofuloderm. The former begins as one or more superficial indurations which, becoming pustular, extend peripherally and form a flat, yellowish, crusted pustule of considerable size, surrounded by a violaceous areola. Neighbouring pustules may coalesce. When the crust is removed, a granular scrofulous ulcer is seen. The small pustular scrofuloderm is usually a papulo-pustule rather than a fully-developed pustule, the pus being frequently limited to the central part of the summit, while the outer part of the lesion remains hard. The crusting is sometimes a slow process, which may occupy several weeks, and when the crust drops off it leaves indelible scars not unlike those of variola. The only diseases with which scrofulodermia generally can be confused are lupus and syphilis. The absence of 'apple-jelly' nodules and of infiltration will distinguish it from lupus, though the two conditions may co-exist. The syphilitic ulcer is met with in adults, and is usually a much more active process than scrofulodermia, nor has the lesion the undermined border which is characteristic of the latter affection. Concomitant syphilitic signs will usually be present, just as in scrofulodermia there will generally be other tubercular symptoms: Wassermann's serum reaction should be tested.

In *syphilis* the pustule is a much less frequent lesion than the papule (p. 490), and is generally found in association with a cachectic state of health. It appears in two different forms, the acuminate and the flat pustular syphilide, and in both the lesion may be either small or large. The *small acuminate* or miliary syphilide, not usually much larger than a pinhead, in most instances begins as a papule, and papules will generally be found intermingled with the pustules. When the crusts into which the pustules dry are detached, there may be some scarring, or the lesions may leave no trace except stains, which presently disappear.

The diagnosis of these small acuminate pustules seldom presents any difficulty ; but it is not so with the *large acuminate* pustules, the acneiform syphilides, which may be mistaken not only for acne, but also for variola and iodide eruptions. Appearing on a base which may at first be pink, and afterwards copper, they may be pustular from the beginning, or may start as vesicles or as papules ; they are more or less generalized, about the size of a pea, disseminated, or grouped irregularly, and while they are predominantly acuminate, some of them may be rounded. Some of the pustules may be dimpled, and occasionally the majority of them display this character. When the crusts fall off, brownish stains are seen, and there may be slight scarring, which, however, is seldom permanent. The grouping which is characteristic of these pustular syphilides, and the drying-up of the pus into scabs, are important points in differentiating them from the lesions of acne, which, further, instead of being generalized, seldom affect parts other than the face, the back of the neck, the chest, and the back between the shoulders. The comedones of acne are another distinguishing feature, the eruption is of a more sluggish and chronic character, and there is no cachexia. The diagnosis as between pustular syphilides and variola is given below. The pustules met with in iodic eruptions are seldom either generalized or profuse.

Small flat pustular syphilides (' impetiginous syphilides ') may begin as such, or may develop from macules or papules. They are discrete ; but in such regions as the face and scalp may run together. The eruption is of a generalized character, with a preference for the genitals, the scalp, and the face. The crusts into which the pustules quickly dry are frequently adherent ; beneath them there is superficial ulceration ; occasionally they are surrounded by an areola of the characteristic raw-ham colour. When the eruption is extensive, the patient is often anæmic and cachectic. The affections from which these syphilides have to be differentiated are pustular eczema and impetigo. The ulceration which underlies the crusts in the syphilides is not found in either of those conditions, nor is itching present as in eczema. In impetigo, the pustules most frequently affect the face and hands, and are superficial ; and the eruption is mild in character and of shorter duration.

The *large flat pustular syphilides* (' ecthymatous syphilides ') differ little from the small ones except in size, and the only lesions with which they are likely to be confused are those of severe impetigo vulgaris. The diagnosis from that condition must rest upon the slow development, the greater number of the pustules, the coppery areola and base, the accompanying cachexia, and the pigmented scars. But it should be remembered—and this applies not to pustular syphilides only, but to syphilis generally—that in most cases a sure diagnosis of syphilis can be made only when all the factors of the case are taken into account : the history, character, course and termination of the lesions, and their reaction to salvarsan, mercury, or arsenic and the iodides. The distinctive characters of secondary lesions generally are their symmetry, their coppery colour, the positions in which they occur, their polymorphism, and the absence of itching, together with enlarged glands, sore throat or tongue. In doubtful cases the whole cutaneous surface should be examined for characteristic marks or lesions. If the diagnosis is still uncertain, the Wassermann test should be applied.

Of all diseases of which the pustule is one of the manifestations, *small-pox* is that which presents the greatest difficulty in diagnosis. The lesion, occasionally preceded by a roseolar rash not unlike that of scarlatina, begins as a mere fleck, of pin-head size, flush with the surface and impalpable. In the course of a few hours it swells up into a pink papule, which can be felt embedded in the skin like a small shot. In a few days, the papule undergoes vacuolation, at the same time getting bigger, and becoming grey and translucent. So the papule passes into the vesicle, which is loculated, so that if it is punctured the contained fluid is not entirely discharged. As a rule, the smaller vesicles are hemispherical, the larger flat-topped, and occasionally the crown is indented. After about twenty-four hours the contents become turbid and the covering dull and whitish.

and so the pustular stage is entered upon. While the lesion is undergoing this transition, the grey translucent centre is encircled at the periphery of the crown by a white or yellow ring. By the sixth day from its birth, the lesion has become yellow throughout and the crown dome-shaped; the pustule so attains maturity, and if of full size measures about three-eighths of an inch across. Even in unmodified small-pox, however, the lesions often fail to reach those dimensions. As the pustule develops, the erythematous zone, the areola, which encircled the papule and was biggest and brightest in the vesicular stage, begins to wane, and has disappeared by the time the pustule reaches maturity. This occurs about the ninth day. As the pustules dry up or burst, scabs are formed, which on separation leave dark stains, scars and 'pits,' the number and depth of the pits usually being determined by the severity of the disease. In mild attacks the pustules remain discrete, in severe cases they run together, confluent small-pox (*Figs.* 237, 238). In bad cases, hæmorrhage takes place into the skin and the interior of the pustules. The mucous membranes of the air-passages may be invaded, the extent to which they are involved being determined by their susceptibility rather than by the severity of the attack. In modified small-pox the eruption may resemble that of the unmodified disease, as here described, the difference being that the lesions are less abundant and are seldom confluent.

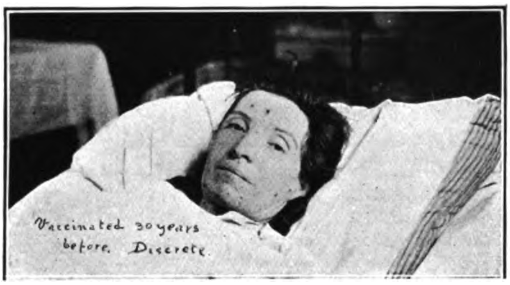


Fig. 237.—Discrete small-pox.
(*Photo by Dr. D. S. Davies, M.O H., Bristol.*)

It has been usual in the diagnosis of small-pox to lay the chief stress upon the solidity and hardness of the papule, the umbilication of the vesicle, and the loculation of its cavity; but in his masterly monograph ("The Diagnosis of Small-pox"), to which I owe the following description, Ricketts has shown that the distribution of the lesions is of more diagnostic value than their character, as also is it more easily observed. The parts most liable to the eruption are the face and hands; and of the two, the face is more liable than the hands. Next to the hands in susceptibility come the upper limbs, then the trunk, then the lower limbs. As to the trunk, the rash is thicker behind than in front, and thickest on the shoulders. The incidence is smallest on the great flexures of the body, while the extensor surfaces of the limbs, and especially the elbow, receive a disproportionate share of the rash. The neck fares better than either the head or the shoulders; the back of it suffers more than the front. On the flank the rash is less profuse than on the adjoining parts of the chest-wall, either in front or behind. On the foot, the distribution is marked by great inconstancy. Usually the back of the foot receives more attention than the sole; between the toes, and in the folds beneath the toes, there is comparative immunity; and the parts for which the eruption shows most preference are the instep, especially the tendinous ridges and the bony eminences, the tendo Achillis, the balls of the toes, the toe-pads, and the heels. In the hand, the palm, and especially the hollow of it, suffers little, and the brunt of the attack is borne by the extensor surface; the rash is thickest on the back of the wrist and hand, and over the heads of the metacarpals. To these usual characters the distribution offers exceptions, some of them difficult of explanation; but they are neither so numerous nor so considerable as materially to lessen its diagnostic importance.

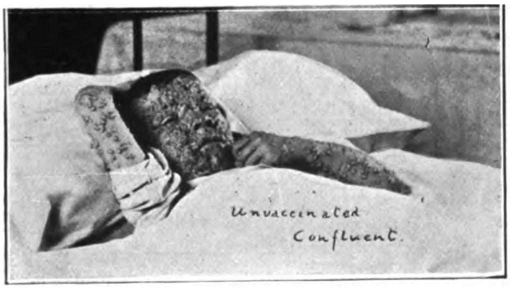


Fig. 238.—Confluent small-pox.
(*Photo by Dr. D. S. Davies, M.O.H., Bristol.*)

The diagnosis of small-pox from chicken-pox—the disease with which it is most often confused—and from vaccinia, has been set out under VESICLES (p. 757). The eruptions of measles and of German measles differ from that of small-pox in that, instead of being papular, they are macular, and that they never pass into a vesicular or a pustular stage. In German measles, further, there is enlargement of the posterior cervical glands, which is never the case in small-pox at an early stage. In scarlatina, the 'strawberry tongue' is a sign which is quite different from the condition of the tongue in small-pox. The rose-red lenticular spots which make up the rash of enteric fever are neither so hard nor so prominent as the papules of small-pox, and they appear chiefly on the trunk, and elect the abdomen and chest rather than the back; the arms and legs, and especially the face, almost always escape.

If the pink, slightly elevated macules of simple purpura are mistaken for the eruption of small-pox, the error is soon corrected by the deeper colour which the macules take on; nor, even though the macules may become papules, have the lesions the characteristic hardness of variolous papules. Another point of difference between simple purpura and small-pox is, that in the former affection the face and trunk are seldom attacked, the sites of election being the limbs. In erythema multiforme, although the rash makes its chief attack upon the limbs, it may be widely diffused and may even invade the face. In such cases, however, the diffusion will usually be less general than that of the variolous eruption, nor is the order of incidence the same. With the involution which the erythematous lesions undergo, the resemblance to small-pox ceases. Even in cases of acute febrile erythema, in which the whole cutaneous surface is covered by a profuse eruption, the distribution is quite different from that of the small-pox eruption.

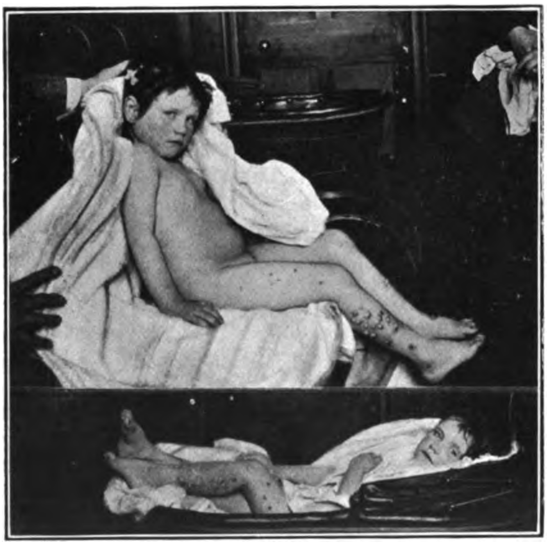


Fig. 239.—Case of septic dermatitis wrongly diagnosed as small-pox. (*Photo by Dr. D. S. Davies, M.O.H., Bristol.*)

Confusion between small-pox and syphilis is much more likely to arise when the syphilide is pustular than when it is vesicular or papular. The erroneous diagnosis may be assisted by the fever and aching symptoms which may precede pustular syphilides, and by the fact that the lesions may begin as papules. In syphilis, however, the constitutional symptoms are less severe, the eruption runs a more indolent course, and appears in successive crops, whilst the vesicles which form on the summits of the papules have an indurated base. Sometimes, too, the syphilitic eruption is indifferent in distribution, and often it comprises various types of lesions, even when it is not distinctly polymorphic, whereas in small-pox the departure from homogeneity is much more limited.

Occasionally, impetigo vulgaris is mistaken for mild modified small-pox (*Fig.* 239), but attention to the points which mark off the former affection from pustular eczema (see above) should prevent the mistake. Further differentiating features as between impetigo vulgaris and small-pox are, that in impetigo there is no fever, and that the lesions begin as vesicles or bullæ and dry up into flat yellowish crusts. In those cases of sudden and acute eczema which may mimic small-pox, guidance is to be found in the small size and superficiality of the eczematous lesions, and the œdema and infiltration of the underlying skin. In scabies, again, the vesicles are superficial, burrows will generally be found, and the heterogeneity of the secondary lesions will aid the diagnosis. In all these affections, the distribution is quite different from that of small-pox, the incidence being partial or patchy. Thus, in impetigo the lesions are frequently confined to the face and extremities, and if the trunk is invaded, it is the front more than the back, the lower part more than the upper. In scabies, except in children, the face escapes, and the commonest sites are the hands and fingers, buttocks, and feet.

In Ricketts' experience, no affection, except chicken-pox, is so frequently confused with small-pox as acne vulgaris, in spite of its chronic, afebrile character, and the absence of subjective symptoms. If, however, the rash is limited to the upper part of the body and a few characteristic acne lesions such as comedones are found, small-pox may be excluded.

I have seen copaiba eruption mistaken for small-pox. The absence of constitutional symptoms such as pain in the lumbar region and fever, the mixed character of the lesions, and the history are the chief points in the diagnosis.

It is seldom that bromide or iodide eruptions are mistaken for the rash of small-pox. In doubtful cases, attention must be paid to the larger size of the pustules, as compared with those of small-pox, and to the symmetrical or patchy distribution. *Malcolm Morris.*

PYREXIA, PROLONGED.—A pyrexia may be considered prolonged if it lasts more than ten days. In most cases, no doubt, there are signs and symptoms, or facts in the history, which enable one to make a diagnosis before this; but difficulties often arise from the absence of the distinctive characters of any one of the diseases commonly accompanied by such pyrexia. In most cases such a prolonged pyrexia is the result of one of the infectious diseases, and it is by a careful consideration of the more probable among these that one may often arrive at a definite opinion. The *general infections* most likely to give rise to a long-lasting fever are:—

Typhus fever
Typhoid fever
Paratyphoid fever
Mediterranean fever
Influenza
Tuberculosis
Meningitis
Malignant endocarditis
Septicæmia from deep-seated foci of disease, such as: Empyema, Cerebral abscess, Pylephlebitis, or other form of suppuration
Malaria
Syphilis
Bacilluria
Bronchopneumonia.

A high temperature of very long duration occurs often in connection with *diseases of the blood and blood-forming organs*, such as:—

Addison's anæmia
Leukæmia
Hodgkin's disease.

It also occurs much more often than has been commonly supposed in *sarcoma* and *carcinoma* of different organs, and has been observed in *cirrhosis of the liver*. There are two other forms of pyrexia which should be borne in mind, namely, a *prolonged pyrexia occurring in children*, often very difficult to explain; and so-called *neurotic pyrexias.*

Modern research has a tendency to rely upon its own methods alone, and to ignore the older clinical differences. But this involves a separate investigation for each disease as it comes to be considered; and thus it may happen that one patient may have to undergo, in addition to a thorough bedside examination of all his organs, a lumbar puncture, and the removal of blood for the Widal test or for the cultivation of organisms; and these may have to be repeated. A full knowledge of the history of the illness, of exposure to infection, and of the clinical changes as far as they are manifested, is desirable in order that the researches of the bacteriologist may be directed as early as possible into the right path.

Typhus.—On the score of prolonged pyrexia (*Fig.* 268, p. 638, and *Fig.* 269, p. 639) little need be said of this fever. The eruption is generally distinctive, and shows itself before the fever has attained any duration; but it is capable of being confounded with typhoid fever, and even with malignant endocarditis. The distinction from typhoid fever is given elsewhere; and a confusion with malignant endocarditis is only possible if the latter should produce a very uniformly distributed petechial eruption over the skin while the cardiac murmur is of slight intensity; or if a person already the subject of cardiac murmur should contract typhus, and have an ill-defined eruption. In either case, if the pyrexia were prolonged beyond the twelfth or fourteenth day, typhus would be unlikely.

Influenza.—In most cases of influenza uncomplicated by definite visceral changes, such as pneumonia or gastro-enteritis, the fever is of short duration, but it is often as long as a week or ten days, and sometimes three weeks or more (*Fig.* 240). The longer period, accompanied as the fever is by few distinctive signs, is sufficient to lead to a confusion with typhoid fever, tuberculosis, or malignant endocarditis, either of which may proceed for two or three weeks, and the last two for many more weeks, without distinctive clinical signs. The constant presence of influenza amongst us, and the great variety in the characters it assumes, make it very difficult to exclude it until positive signs of another complaint have manifested themselves. Equally difficult, however, is it to prove the existence of the disease, since the organism, Pfeiffer's bacillus, is not found easily in the blood; and in the prolonged cases, the rather striking peculiarities of the intense acute attacks, such as severe pain in the head and back of the eye, and in the lumbar region, may be absent. The diagnosis can often be made positively only *via exclusionis*, when the bacteriological tests of typhoid fever and tuberculosis have failed, and if there is an entire absence of rose spots, diarrhœa, or enlarged spleen on the one hand, or of pulmonary symptoms on the other. Especially must we bear in mind that an apparent attack of influenza may only be the pyrexial equivalent of early tuberculous infection, and if at any time during the illness sputum is available, it should be examined for tubercle bacilli.

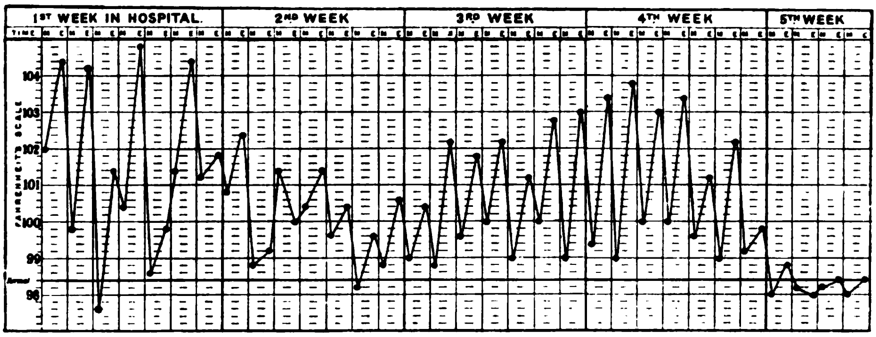


Fig. 240.—Temperature chart in a prolonged case of influenza in which the pyrexia lasted a month: the diagnosis was confirmed by bacteriological detection of Pfeiffer's influenza bacilli. The case was complicated by influenzal otitis media and transient cardiac bruits.

Typhoid Fever should be comparatively easy to diagnose in the present day. A fever commencing with frontal headache, perhaps with diarrhœa, generally compelling the patient to lie up in bed by the end of the first week, and showing within the first ten days rose spots on the abdomen and a slight enlargement of the spleen, while at that time the temperature is 101° F. or 102° F. in the morning, and 103° F. or more in the evening, and the pulse is relatively slow, namely from 80 to 100 in the minute, should be typhoid fever. The Widal reaction, that is, the agglutination of typhoid bacilli by the patient's blood serum, becomes positive about the tenth or twelfth day. All these signs may fail for a time: spots may be absent, the bowels may be persistently constipated, enlargement of the spleen may be difficult to prove, the Widal test may and often does fail. Examinations of the fæces for Eberth's bacillus are not easy; but cultivation of the bacillus from the blood taken from the patient's vein may yield the bacillus, and this at an earlier date than the Widal reaction can be obtained. Apart from such cultivation, the appearance of rose spots from the sixth to the tenth day, with additions to their number every day for five or six days, forms perhaps the most conclusive evidence of typhoid fever; and the diagnosis based on these grounds should not be upset by one or two failures to get a positive reaction with the Widal test. This test may require to be made with two or more strains of Eberth's bacillus, and failing them, paratyphoid bacilli should be used. A positive Widal reaction in a case otherwise unlike typhoid fever must be accepted with caution. If a case is devoid of spots, and gives no Widal reaction, the probability of its being typhoid fever might be asserted from its mode of onset, a characteristic chart of temperature (*Fig.* 241), with high readings morning and evening in the middle of the second week, and ending in twenty to twenty-five days after wide oscillations; and finally, a pulse always under 100. On the other hand, a pulse of more than 100 does not exclude typhoid, as it is common enough in the severe adynamic forms. Typhoid fever may be confounded with many acute diseases ; as a prolonged pyrexia it is especially pulmonary tuberculosis, malignant endocarditis, a long-lasting influenza, septicæmic processes, and occasionally tuberculous meningitis which will give difficulty.

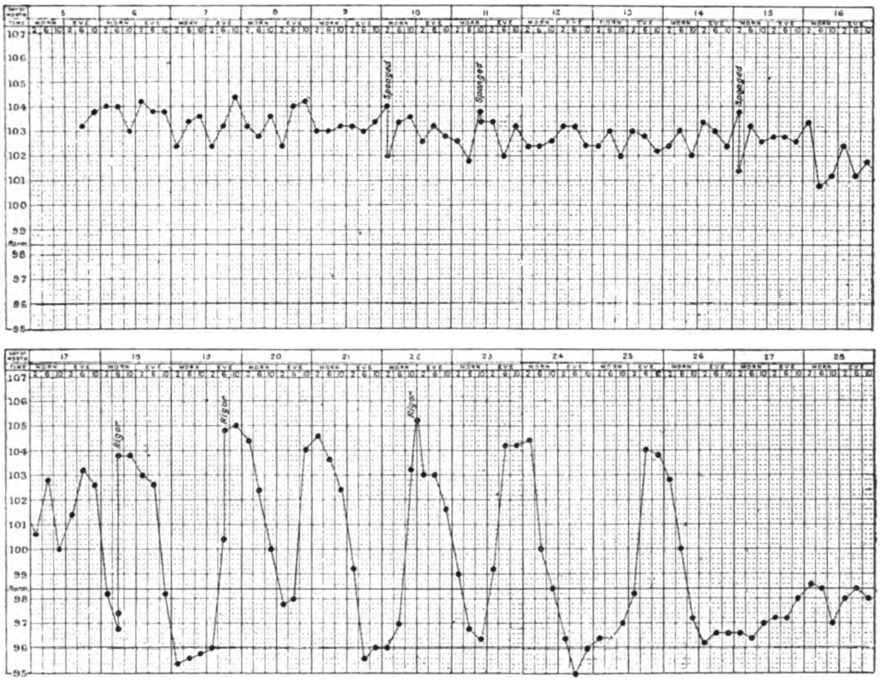


Fig. 241.—Temperature chart of a case of typhoid fever.

Paratyphoid Fever.—Of this it need only be said that it presents the features of a benign typhoid fever, in which all the distinctive characters are less marked. Like typhoid, it may be confounded with a mild long influenza, or with early tuberculosis. Its recognition depends ultimately upon the agglutination of paratyphoid bacilli by the patient's blood serum.

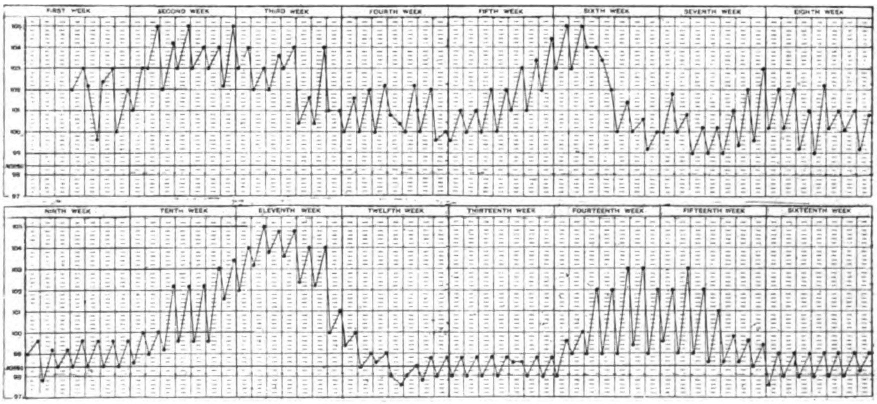


Fig. 242.—Temperature chart of a case of Mediterranean fever of undulant type.

Mediterranean Fever is one of the most prolonged of the fevers due to one recognized micro-organism ; for in the undulant form of the disease successive exacerbations of pyrexia may carry on the illness into the fifteenth or sixteenth week (*Fig.* 242). It resembles typhoid fever closely, including the enlargement of the spleen, but is proved to be due to infection with *Micrococcus melitensis*, conveyed, almost invariably, in goat's milk. In British practice its occurrence is unlikely, except in one who has been in the parts of the world where it is rife—namely, the Mediterranean coast and islands, whence it has found its way into Spain, Portugal, and other countries, and even to such distant parts as South Africa. It differs from typhoid fever in the absence of spots and of characteristic diarrhœa, but as these often fail in typhoid fever the distinction is not always available. However, the diagnosis can generally be made by the Widal test, since the blood serum will agglutinate the *Micrococcus melitensis* as early as the fifth day of the disease.

Tuberculosis.—The onset of general miliary tuberculosis, or of miliary tuberculosis of the lung, has often for its only symptom a moderate pyrexia, with accompanying physical weakness and anorexia. Such a condition may continue for weeks without any other sign; the breathing need not be quickened, and there may be an entire absence of abnormal signs in the lungs. The morning temperature is frequently near the normal, while the evening temperature alone is high; and it does not as a rule increase to a maximum and subsequently fall, so as to form the curve which is characteristic of typhoid, but continues nearly at the same level for long periods. In the absence of sputum the detection of tubercle bacilli is not available. Tuberculin tests may, however, be tried, and will probably give positive results. Of these the most suitable is the cutaneous tuberculin test of von Pirquet (*Plate XXXVII*, p. 768). Tuberculous infection in other localities may equally, when not accompanied by striking local signs, and only presenting the pyrexia, be taken for typhoid fever: for instance, tuberculous disease of the kidney, or pyelitis, and especially tuberculous meningitis. Headache and fever occur both in this and in typhoid and may be for a time the only facts in the case, until in the one case spots or loose motions assert themselves, or, in the other, optic neuritis, convulsions, paralysis, or retracted abdomen point to a cerebral localization.

Meningitis.—The duration of a tuberculous meningitis is often three or four weeks, during which there is pyrexia of moderate, or occasionally of severe type. In the sporadic, infantile form of cerebrospinal meningitis, first described as posterior basal meningitis, the fever is often prolonged for five, six, or more weeks. The continuous temperature is not generally very high, but in some cases there are sudden rises to 103° and 104°. followed by a fall to the normal within a few hours (*Fig.* 253, p. 591). These rises of temperature may occur daily, or every other day, or more frequently. As, in meningitis, by the time the pyrexia has become prolonged the cerebral symptoms, such as drowsiness, coma, and retracted head, are usually pronounced, there is not much difficulty in locating the disease in the meninges. To determine whether the inflammation is tuberculous or meningococcal or of other bacterial origin may require a lumbar puncture (p. 804) or blood culture.

Infective or Malignant Endocarditis may be mentioned next, because for days, weeks, or months the only prominent feature may be a continuous pyrexia with evening rises to 101° F., falling in the morning to 99° or 98·4° F. (*Fig.* 243). In most cases a cardiac murmur is present, and then one may, after a certain lapse of time, such as fourteen days, and excluding the possibility of typhoid fever, often make a diagnosis with confidence. This is confirmed by the occurrence of other symptoms mentioned on p. 34. An attempt should be made to obtain an organism from the blood by cultivation, but it is not always successful. In the early stages the difficulty is not uncommonly increased by the fact that no murmur can be heard, in spite of the fact that endocardial changes are present. The course of some of these cases of infective endocarditis, which may be called chronic,

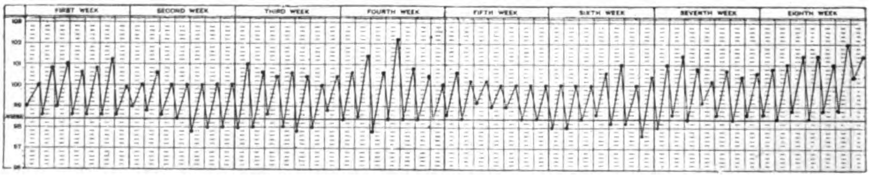


Fig. 243.—Temperature chart of a case of malignant endocarditis.

is remarkably prolonged, sometimes for six, eight, or twelve months, with little variations in the accompanying conditions. Long before that, of course, typhoid fever and tuberculosis would have been excluded ; and after eight or ten weeks the co-existence of cardiac murmur, with uniform prolonged pyrexia, would make the diagnosis certain. Cases in which the brunt of the disease falls upon the brain may actually have meningitis, as in pneumococcal cases ; or they may be mistaken for meningitis, or for typhoid fever, or, if petechiæ are present, for typhus. But in such instances the illness is generally a short one, and it would scarcely come into the present category.

Septicæmia.—In any prolonged pyrexia the possibility of a focus of deep-seated suppuration should be considered. In many situations the focus causes pain which at once directs attention to the origin of the trouble ; but in some cases pain is absent, and foci in some situations are habitually painless. The disorders to be thought of are *otitis, abscesses in the throat, a small empyema, cerebral abscess, deep-seated glandular suppuration, suppurative pylephlebitis, appendicitis, pelvic suppurations* in women, and *umbilical lesions* in the new-born infant. If the blood shows a leucocytosis this will be in favour of a suppurating centre. At any rate, it will exclude typhoid and miliary tuberculosis. Each possible centre of infection must be called to mind, and the locality concerned must be investigated. Suppurative pylephlebitis, in spite of the extent of the lesions and the size of the organ concerned, has frequently been overlooked, and has been mistaken for typhoid fever, pneumonia, peritonitis, or appendicitis. It is relatively rare, but it is a sequel of other suppurative lesions in the abdomen, and should therefore be thought of when appendicitis or other similar affection has been under treatment. Rigors are very inconstant in diseases of this class ; if they occur they compel attention to the possibility of pyogenetic centres (*Fig.* 244) ; but their absence must not be allowed to influence one in the contrary sense. They are most constant in visceral pyæmia, but this illness is not generally prolonged. In some cases the organism is only discovered after repeated cultural examination of the blood, the urine, or even the cerebrospinal fluid, and it may be found to be some rarity such as the *glanders bacillus* or a *leptothrix bacillus*.

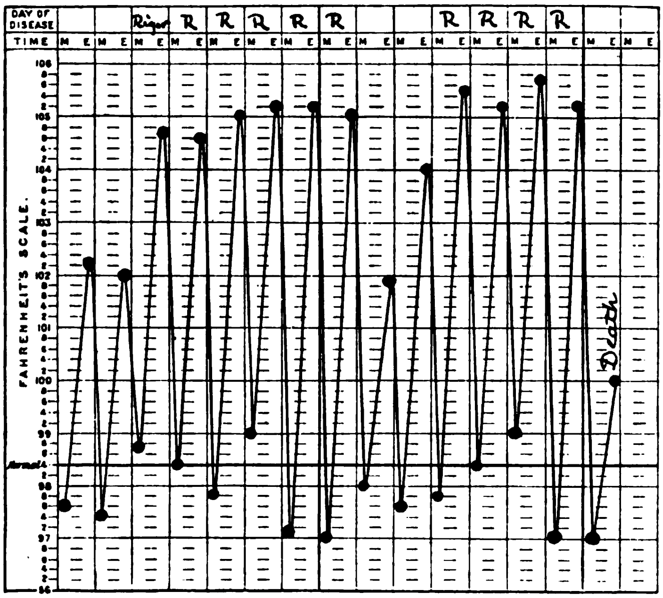


Fig. 244.—Temperature chart of a case of pyæmia, secondary to otitis media and lateral sinus thrombosis. There was a rigor almost daily.

Erysipelas, though generally of short duration, sometimes causes pyrexia lasting more than a fortnight (*Fig.* 245).

Syphilis.—Like other infections, this has its fevers, both in the secondary stage—that is, the stage of generalization of the infection—and in the late stages accompanying gummata and other local processes. The secondary fever is certainly not present to a marked degree in most cases; a little feverishness there may be, but it scarcely requires special mention. Only occasionally is there a really prolonged pyrexia, with decided evening elevations of temperature. The diagnosis is rarely in question, because the fever only accompanies the rash and sore throat; and the origin in a local infection is generally well known. The same help may be with us in the fever attending gumma-formation; but if the gumma is deep-seated in a viscus such as the liver, even though it should be painful, and recognized by palpation as an abnormal enlargement or thickening, some hesitation may be felt as to the diagnosis unless it is remembered that such pyrexia may be present. Moreover, a pyrexia of this kind has occurred in connection with the lesions of the inherited disease. Syphilitic pyrexia is frequently of decided character, with evening elevations to 103° F. or more, and morning falls nearly to normal, until stopped, as it may be at once, by the administration of potassium iodide. In both these cases the Wassermann reaction is available to make certain the nature of the infection.

Bronchopneumonia.—This may be mentioned because it has sometimes a duration of many weeks, and during the whole time an oscillating temperature is present. The local signs are, however, sufficient to explain the presence of fever, and the difficulty lies only in the fact that a general pulmonary tuberculosis may resemble almost exactly the more curable bronchopneumonia of pneumococcal or streptococcal origin.

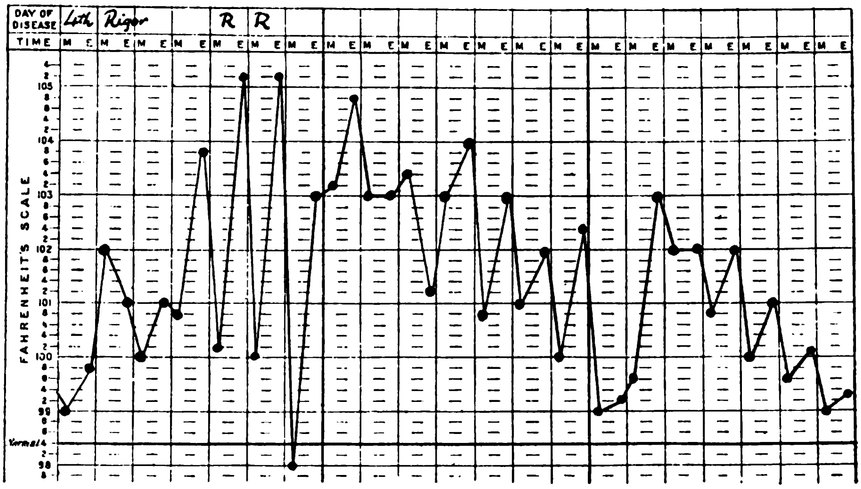


Fig. 245.—Temperature chart of a case of facial erysipelas following a septic scratch.

Malaria.—Important as is this disease, its diagnosis may be dealt with briefly here. The non-malignant quartan and tertian fevers produce isolated pyrexias of short duration (*Fig.* 6, p. 28, and *Fig.* 7, p. 29) which do not come within the scope of this paper. It is only the malignant or subtertian fevers, which do not arise in this country, and are little likely to be seen in other than regions known to be malarious, which cause prolonged continuous pyrexia (*Fig.* 11, p. 31). The diagnosis is based upon a consideration of the symptoms, upon the discovery of the parasite, and upon the results of treatment with quinine. The symptoms chill, fever, and sweating have less value in a continuous pyrexia than in the simple tertian and quartan fevers, and in a quotidian fever the resemblance to septic poisoning is well recognized. An enlarged spleen is present in malaria, but also commonly in typhoid fever. Most reliance is to be placed upon a microscopical examination of blood-films coloured with Romanowsky's or Leishman's stain; for in cases of pernicious malaria, it is rarely that the organisms (*Plate XXVIII*, *Fig. E*, p. 614) fail to be discovered by this means. Absence of leucocytosis and an increase of the large mononuclears to 15 per cent in a differential count of the leucocytes in the blood, are also in favour of malaria, at least in the first two weeks; after which the results may be very similar in typhoid fever. The third test is the administration of quinine, which, given in sufficient quantity, will stop malaria; and on the other hand, if the fever continues in spite of it, malaria is excluded. An adequate dose for this purpose is 3 or 4 gr. every three hours, day and night, for two or three days.

Bacilluria.—This is usually due to infection of the urinary passages with ***Bacillus coli communis.*** The symptoms may be slight or they may be those of cystitis or pyelitis in a marked degree (see BACTERIURIA, p. 69); with these there is a more or less continuous pyrexia (*Fig.* 193, p. 456). Especially in infants they may be few, or none at all other than the pyrexia, which is variable in degree, generally irregular from day to day, may be accompanied by chills and sweating, and may last several weeks. Hence in an obscure fever in infants the urine should be examined carefully.

Anæmia.—The several forms of pernicious, idiopathic, or Addisonian anæmia are frequently accompanied by a moderate degree of pyrexia, which may persist for many weeks (*Fig.* 246). Such an anæmia might be the manifestation of tuberculosis, of infective endocarditis, or of malignant growths; but an examination of the blood with the discovery of poikilocytosis and a colour-index above unity, the lemon-yellow tint of the skin in some

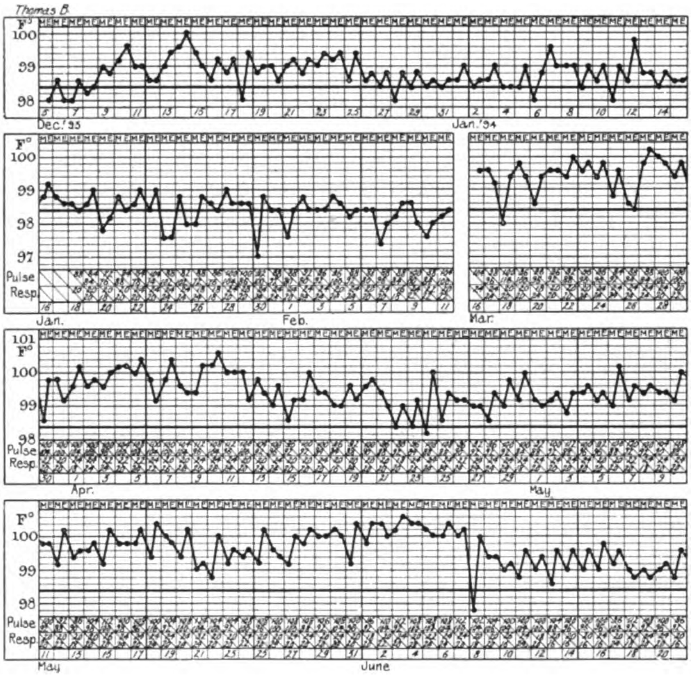


Fig. 246.—Characteristic temperature chart of a case of pernicious anæmia.

cases, and the history, will generally determine the diagnosis, though the differentiation of the particular form of anæmia may still remain to be considered (see ANÆMIA, p. 20).

Hodgkin's Disease.—In some, but by no means all, cases of this disease, a very remarkable form of pyrexia occurs. It consists of alternating periods of pyrexia and apyrexia, each of eight, ten, or twelve days' duration, lasting in all for six, nine, or twelve months. During the pyrexial period the temperature, beginning near the normal, becomes day by day higher and higher, till on the fourth or fifth evening it reaches 102° F. or 103° F., its highest point; it gradually declines during the next four or five days to the normal; and then for three or four days it becomes increasingly subnormal till a minimum is reached, and from this point there is a gradual return to the normal, when a second pyrexial curve begins (*Fig.* 247). If the cervical, axillary, or inguinal lymph-glands, apart from or in company with the spleen, are enlarged, and the condition of the blood is normal or of the simple chlorotic type, the diagnosis of Hodgkin's disease is safely made. If such a temperature is observed without any enlargement of the spleen or of the external glands, a lymphadenomatous enlargement of the internal glands, bronchial or mediastinal should be suspected, and endeavours should be made to demonstrate them by palpation, percussion, or x-rays. Leucocytosis should be absent ; but its occurrence in a late stage of the pyrexia would not militate against the diagnosis.

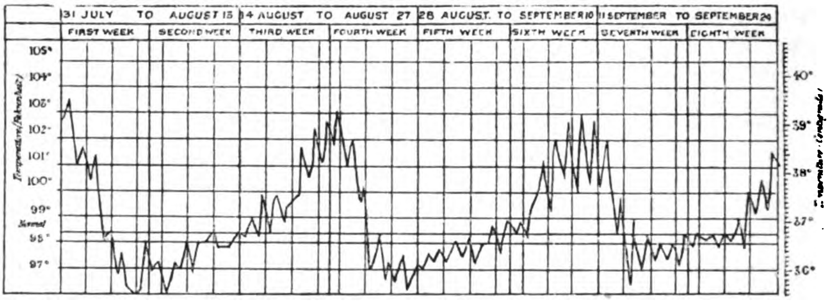


Fig. 247.—Temperature chart of a case of Hodgkin's disease.

Leukæmia.—Pyrexia is a common occurrence in the different forms of leukæmia. As a rule the great size of the spleen in one group of cases leads to an easy diagnosis, and in another group the glands are manifestly enlarged, though acute cases occur in which these changes do not appear early ; in all these forms an examination of the blood will show the excessive number of leucocytes, of one or other variety (p. 24), a sufficient explanation of the occurrence of pyrexia.

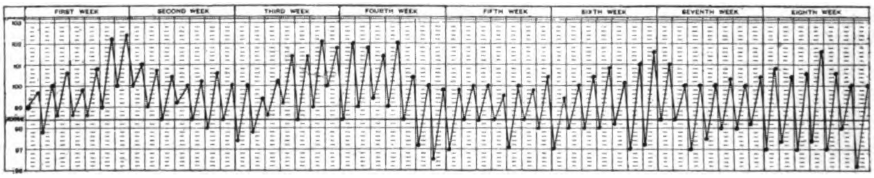


Fig. 248.—Temperature chart of a case of sarcoma of the neck and mediastinum.

New Growths.—New growths are not accompanied by pyrexia as a rule ; but many exceptions have been recorded, in regard both to sarcoma and to carcinoma. A case of sarcoma of the neck and mediastinum was accompanied for the four months preceding death by a pyrexia resembling in its variations from week to week that of Hodgkin's disease (*Fig.* 248), and carcinoma has now frequently been known to be accompanied for weeks or months by fever, especially where there are secondary deposits in the liver (p. 326). The diagnosis is not generally difficult ; the tumour is in evidence. A point of importance is that the pyrexia does not help to distinguish between syphilis and cancer.

Cirrhosis of the Liver.—The diagnostic relations of pyrexia in cirrhosis of the liver are on the same footing. It occurs in some cases (*Fig.* 167, p. 371) and is apparently due to cirrhosis ; perhaps because it is an inflammatory process, or because the cirrhosis is due to a toxæmia which may raise the temperature. The fact is of interest when we consider that ascites due to hepatic cirrhosis has often been mistaken for tuberculous peritonitis, and, perhaps less often, tuberculous peritonitis for cirrhosis. The absence of pyrexia might possibly be held to exclude tuberculous peritonitis, but its presence would leave the diagnosis open, since it might be due to either.

The curious pyrexial outbursts in *rat-bite fever* are described on p. 598, and the long-continued low fever of *pellagra* on p. 225.

Neurotic Pyrexia.—This term may be applied to two classes of case : in one the mercury is found at extraordinarily high levels at irregular intervals, often at different levels in different parts of the body, and falling again rapidly to the normal. This may be repeated many days, but hardly constitutes prolonged pyrexia. In the second variety the patient has a genuine pyrexia, lasting two, three, or more weeks, irregular in the elevations reached on successive days, but on the whole resembling the pyrexia of sepsis.

These cases are more often females, and the fever may be associated with symptoms referable to the pelvic organs, or may persist after the entire subsidence of such symptoms, so that skilled obstetric physicians have been unable to find any active local lesion. The neurotic origin of such cases may be open to doubt, but it is supported by the variability of the pyrexia, and by its occasional rapid cessation without adequate explanation, and without any local change. The diagnosis can only be made as a rule *via exclusionis*, and must always be considered provisional as long as there is any probability of an adequate cause being found.

The Unexplained Pyrexia of Children.—This form of pyrexia is in a somewhat similar position to the above. All students of disease in children are familiar with the fact that in patients under nine or ten years of age a mild pyrexia may occur, lasting two, three, or four months, for which no explanation can be found. Since it arises in quite young subjects, a neurotic origin is not so readily suggested, nor so likely, as in the adult female, and yet it is possible that some cases are of this nature. Tuberculosis should be considered carefully, but is often safely and rightly excluded. Gastro-intestinal disturbance, such as constipation ; the unsuitable nature of the food ; and acute intoxication from the alimentary canal, are other possible explanations of some cases. Since the prognosis is good, it is satisfactory to be able to recognize the clinical condition, even though the causation is obscure ; but the diagnosis can only be established after careful exclusion of typhoid fever, tuberculosis, and local inflammatory conditions such as otitis, bacilluria, and other definite lesions or infections. *Frederick Taylor.*

PYREXIA WITHOUT OBVIOUS CAUSE.—From researches carried out in trying to ascertain the mechanism by which the body maintains in health an average temperature (practically constant) of 98·4° F., and the factors that lead in disease, and also even in health, to a departure from that temperature, a number of general principles have been established, and these must be grasped before we can form a just estimate of the value of the reading of the thermometer in diagnosis. We must briefly state the more important of these without discussion.

1. *Sources of the Actual Production of Heat in Health* in order of importance : (*a*) Muscles at work ; (*b*) Glands at work ; (*c*) All minor tissues in which katabolic changes are proceeding.

2. *Provisions for the Escape of Heat.*—(*a*) Temperature changes in the air, especially that expired ; (*b*) Escape by the skin, radiation, and evaporation of sweat ; (*c*) Escape by urine and fæces ; (*d*) Warming of food, and anabolic tissue changes.

3. *Mechanisms for Regulating the Distribution of Heat generated Locally.*—(*a*) A main centre for regulating the distribution of the blood, situated in the medulla ; (*b*) Subsidiary centres for the same purpose situated in the cord and (?) elsewhere ; (*c*) The physical properties of the blood, lymph, and tissue juices as regards their circulatory movements and their capacity for absorbing and retaining heat.

These three belong to the province of pure experimental physiology and physics ; we have, then, the further observations shared by physiology and clinical medicine :

4. That the temperature in perfect health can be raised considerably (at least to 102° F. – Hill) by strenuous physical exertion ; such elevations disappear very rapidly, possibly with small oscillations.

5. That in exhaustion from violent effort or exposure (generally both, as in shipwrecks and similar accidents) the temperature can be, and is, very dangerously depressed, this being possibly the sole cause of death.

6. That in every form of microbic invasion of the body, the temperature may be altered. This alteration may be produced in several conceivable ways, e.g., by the direct action of the microbes or their poisons on the seats of the production of heat or on the centres regulating its distribution, or again by the action of the same poisons or microbes on the main sources of loss of heat. The exact and precise cause is very obscure, though extremely interesting to the pathologist.

Lastly, as purely clinical observations we have learnt :—

7. That in order that variations in temperature may kill, or indeed of themselves be of very serious import, they need to be very extreme (say 6 or 7 degrees above or below normal) or else very prolonged, the necessary duration varying with the degree of departure from the normal.

8. That in the wards of a hospital where the temperatures are taken and charted at regular intervals, it is scarcely possible to find a chart of chronic disease which does not show some oscillations in temperature, the causes of which are obscure or overlooked.

From these general propositions we may pass to the practical value of thermometric observations in diagnosis. We may again generalize, and say, that standing alone as a primary and isolated observation, the fact of a disturbance in temperature is of little use ; but when this observation is controlled by other factors in diagnosis, and we have made a complete diagnosis of a given disease, we are then in the position of knowing by experience approximately the course of the temperature for that disease, and we shall get many useful hints from the thermometer as to complications or the severity of the attack : a point to which we shall refer presently. Hence, it is useful in our present discussion to divide all patients into two main groups : (1) *Those who are well enough to visit their doctor*, and (2) *Those who are too ill to do so.*

Patients who are well enough to visit their doctor.—Unless some special complaint or physical sign soon leads to satisfactory diagnosis, let it be an accepted rule to take the temperature ; by doing so we shall not infrequently discover, early, a disease which we might otherwise have overlooked until something or some one else had drawn attention to it, greatly to our discomfiture and loss of credit. The three most typical illustrations of this position are perhaps *typhoid*, *phthisis*, and a condition which in our haste we label '*Influenza*.'

A headache lasting four or five days and associated with some degree of pyrexia, is, even standing alone, so suspicious of the first, that the patient must be ordered to bed to be watched. A nasty dry cough, with general anæmia, weakness, tiredness, and malaise, makes us very suspicious of the second, especially if there is also the slightest alteration in the breath sounds anywhere. The third is the recently developed refuge of the diagnostically destitute ; it leads to much mortification when suppurating gums or tonsils, gonococcal discharges, decomposing urine, or other critical points are discovered later : a position which may be avoided by examining all easily accessible orifices, and if the mouth, throat, and nose all seem healthy, by labelling the trouble fever of uncertain origin, and ordering the patient to bed. In fact, *if on examining the patient in the consulting-room no cause for the pyrexia is apparent*, I would lay down as a golden rule—*Remove the patient at once from Group* 1, *and place him in Group* 2 for further critical examination of his or her person, blood, and excreta.

Patients who request their doctor to visit them.—It must be admitted that in this group also, pyrexia by itself is not a very strong diagnostic point, for *ex hypothesi* the other symptoms are of considerable severity and importance, and therefore point pretty strongly in some diagnostic direction. Our residuum from Group 1, with a certain proportion of individuals originally in Group 2, will, however, together make up a by no means negligible number of patients in whom the cause of pyrexia is obscure to an ordinary examination. Here it is the first rule to examine systematically every orifice of the body for a possible source of trouble ; gums, and especially the roots of the teeth for pyorrhœa, ears, nose, throat, vagina, rectum, and urethra ; should all these prove healthy, the fingers and toes must be inspected for overlooked sores ; and the bones near joints investigated for a possible osteomyelitis.

The Blood.—It is by now almost certain that some clue will have been obtained, but should none have appeared (in some cases even when a complete diagnosis has been made) we must now proceed to have the blood examined by the best available scientific methods ; we may by this means prove that the blood itself is at fault, or that it contains some microbes, thus :—

1. *Leucocytosis* suggests leucocythæmia perhaps, in which disease outbreaks of pyrexia are not uncommon ; or some obscure focus of suppuration ; or perhaps it proves that an obvious focus requires the surgeon's assistance ; or per contra, it provides a slight argument against typhoid or tubercle, and in favour of gross parasitic worms.

2. *Poikilocytosis* suggests a severe anæmia, which, like leucocythæmia, is known to have pyrexial periods.

3. *Widal's Test*, if positive, practically proves the presence of typhoid.

4. *Cultivation* of it may prove a microbic invasion, the name and nature of the invader, and possibly the point of invasion.

5. *Microparasites* of protozoal form (malarial or other) may be recognized in the blood under the microscope.

The Urine may be found to be thick or decomposed, and thus give a clue to tubercle of the genito-urinary tract, to calculous trouble, or to *B. coli communis* invasion. It is well to remember that *B. coli* may exist in what otherwise appears to be a healthy urine (p. 12).

The Fæces may yield information: melæna, fatty stools, gall-stones, or even eggs of parasites may be found, thus clearing up the cause of an obscure pyrexia.

Tests such as Calmette's or von Pirquet's may be tried for *latent tubercle*, but the dropping of tuberculin into an eye is not without its own risks, and in my opinion had better be avoided, especially as by the time tubercle has caused pyrexia it has probably advanced so far as to be discoverable by careful physical examination of the chest. Moreover, the test is alleged to be so delicate as to discover obsolete tubercle, which is not the cause of the pyrexia, and so may lead one astray.

When physical examination, clinical methods of investigating secretions and excretions, and bacteriological aids have thus all been exhausted, there will still remain a few rare cases in which the cause of the pyrexia is undiscoverable. These are chiefly abdominal: thus gall-stones and their complications, pylephlebitis, ovarian abscess, and deep-seated phlebitis have all presented instances to the writer; but he knows of no rules by which such cases can be discovered; time or an autopsy can alone clear them up. Apart, however, from such obscurities, there still remain some interesting and useful observations to be made on certain clinical thermometric measurements.

PYREXIA OR SUBNORMAL TEMPERATURES IN CHILDREN.

In children we know that the heat-regulating function, like other nervous mechanisms, takes time to develop proper and complete connections with, and control over, subordinate centres of regulation, whether these be foci of production or surfaces of loss; hence we are not surprised, still less alarmed, at temperatures above normal in children which in adults may have a very different significance. It is rather the reverse with those below normal, because we know that the growing child requires very large supplies of heat to carry on the anabolic functions of growth and development.

Subnormal Temperature.—Suppose we find a child in this condition, the immediate diagnosis is great exhaustion; such temperatures are generally found in marasmic children who have been starved, or its equivalent, i.e., badly fed, or exposed. Subnormal temperatures are also found in summer diarrhœa, or sometimes in conditions associated with intense pain; also, at an interval after convulsions (not immediately), and in many other conditions, the differential diagnosis of which will generally be obvious.

Pyrexia, on the other hand, owns an infinity of causes, of which we can only glance at a few which may give rise to trouble in diagnosis.

Excitement.—In children's wards, visiting day almost invariably causes a plentiful crop of high temperatures, even as much as 103° F., and the private practitioner has to remember this when a loving aunt or an injudicious visitor of any sort has come in contact with his little patient at home. A bad night may easily send the temperature up to any height.

Febricula.—This is merely a Latin name for the fact of pyrexia, but before using the term let every system be examined carefully for signs of disease. It may be that there is absolutely nothing else wrong. The following is a useful and ordinary average clinical rule: for each 1° F. the temperature rises we may expect the pulse to increase in frequency 10 beats (or in children 15) per minute, and the respirations 2 to 3 per minute, and if this ratio is maintained it is fairly safe to say that heart and lungs are neither of them specially threatened, and hence, *if there be nothing else to attract attention*, it is safe to say that the child is certainly feverish, and we must nurse and watch it. I have known a delicate boy of 9 or 10 thus to have a temperature of 102·5° to 103·5° for three or four days at a time for no reason that I could ever detect; he ate well, slept well, and felt well; and this aspect of affairs should be remembered. On the other hand, such temporary attacks of pyrexia may be aroused by the presence of some micro-organisms which have hitherto escaped detection, but *quâ* the pyrexia in itself nothing more can be said. Deep-seated tubercle, perhaps in internal lymphatic glands, may be suspected, but it will often be exceedingly difficult to settle the diagnosis.

Convulsions.—These must be remembered as a possible cause of a temperature of even 103° F. if the thermometer be used at once.

Specific Zymotic Diseases.—On discovering a child with fever, suspicion will naturally be aroused that one of these troubles is at hand. The only thing to be said here is: Do not attempt to give a diagnosis on the thermometer alone; the temperature is raised long before a diagnosis is possible. It is well to note that a temperature of 102° F. only, or less, is a point in favour of diphtheria against a tonsillitis of other causation, which frequently has a pyrexia of 104° F. or 105° F. Slight pyrexia, with headache and vomiting, makes us suspect tuberculous meningitis, whereas a temperature of 103° F. or 104° F. with similar associations renders pneumonia probable.

IN CHILDREN AND ADULTS.

Here we may draw attention to some of those cases in which the fever of known average departs from its usual course.

Empyema.—Suggested by the crisis of a pneumonia followed in a few days by intermittent or remittent fever (*Fig.* 71, p. 160).

Endocarditis.—Suggested in rheumatism by a temperature of 100° F. to 100·5° F. or 101° F. following the initial fall from the administration of salicylates; though the absence of such pyrexia does not exclude the existence either of endocarditis or of pericarditis.

Hyperpyrexia.—Met with in rheumatism, typhoid, and other septic conditions; its onset is easy to detect in the latter conditions; in the former, restlessness with *less* complaint of pain may cause suspicion. The diagnosis must be confirmed by a frequency of registration proportionate to the severity of the initial observation; two-hourly, hourly, or even quarter-hourly observations may be necessary, that treatment may be controlled.

In addition to such obvious causes, it must be remembered that in cerebral diseases and injuries of almost any kind, the temperature may rise to most unusual heights; one over 106° F. almost certainly heralds a fatal event; the same is sometimes found in uræmia.

Attention may be drawn to the fact that *malaria* is not quite extinct in England, and a regular succession of pyrexial attacks at the same time of day suggests this cause.

Sudden Drops in Temperature.—These are met with in many diseases at times, e.g., typhoid, and the patient must be examined carefully for other suggestive factors—blanching, severe abdominal pain, etc., indicative of hæmorrhage, perforation, or other complication. Do not forget that the crisis in pneumonia is a great deal more of a crisis for the patient than it is for his microbes, and be ready with the appropriate stimulants.

Pyrexia and Malignant Disease.—When the nature of a swelling in the liver or elsewhere is being considered, the discovery of a temperature hovering about 101° F., or even higher, does not militate against a malignant growth, for these frequently give rise to pyrexia (*Fig.* 145, p. 326), and so, too, but more rarely, may cirrhosis of the liver (*Fig.* 167, p. 371).

In the differential diagnosis of *cerebral abscess* from *tumour* it must not be overlooked that a subnormal temperature, contrary to the usual rule of suppuration, suggests an abscess rather than tumour.

It is well to bear in mind that *after an operation*, a few degrees of pyrexia, even for forty-eight hours, are not a conclusive proof of infection of the wound.

When the source of a continued pyrexia is being sought for, the heart must be examined carefully every day, or even more frequently, for *infected endocarditis* is by no means an infrequent result of gonococcal and other microbic invasions which may be most difficult to detect.

Fred. J. Smith.

PYROSIS.—(See HEARTBURN, p. 296.)

PYURIA.—Pus appears in the urine in all suppurative conditions affecting the urinary tract, and occasionally from the rupture of an extra-urinary abscess into the urinary apparatus. It may be present in large or in microscopic quantities; when in bulk it forms a thick, greyish, tenacious sediment, which must be distinguished from phosphates and from urates; urates are of a pinkish colour, and will be cleared by heating the specimen; phosphates will be dissolved by the addition of acid, whereas pus will remain unaltered by either test.

In alkaline urine, the pus cells tend to run together into a dense viscid deposit, leaving the upper layers of the urine slightly turbid. Microscopically each pus cell is multinuclear, rounded, and about twice the size of a red blood-disc. The contents are granular, but the addition of acid clears the cell and makes the nucleus stand out more distinctly. Urine containing pus will always contain at least some albumin and frequently epithelial cells from some part of the urinary tract. If the specimen containing pus be shaken gently with ozonic ether, a slight effervescence will be produced, or if mixed with liquor potassæ, a ropy precipitation occurs.

The following is a classified list of the causes of pyuria :—

(*A*). From Diseases of the Urinary Organs.

1. *Renal* :
    - Pyelitis
    - Pyelonephritis
    - Renal abscess
    - Pyonephrosis
    - Tuberculosis
    - Calculus.
2. *Ureteric* :
    - Calculus.
3. *Vesical* :
    - Cystitis
    - Tuberculosis, acute or chronic
    - Calculus
    - Ulcer—Simple, Epitheliomatous
    - Tumour—Sloughing papilloma, Villus-covered carcinoma
    - Bilharzia hæmatobia.
4. *Prostatic* :
    - Prostatitis, acute or chronic.
5. *Urethral* :
    - Urethritis — Gonorrhœal, Septic, Gouty
    - Stricture.

(*B*). From Diseases outside the Urinary Organs.

- Leucorrhœa.
- Balanitis with phimosis.
- From the extension of inflammatory processes to the bladder, or the rupture into the bladder or urethra of an abscess such as :—
    - Prostatic abscess
    - Appendicular abscess
- Iliac or pelvic abscess
- Psoas abscess
- Pyosalpinx
- Carcinoma of the uterus, rectum, cæcum, sigmoid, or pelvic colon
- Ulceration of the small intestine—tuberculous or dysenteric.

It is impossible to determine the lesion producing pus in the urine simply by the examination of the latter. Due consideration must be given to the history and the other symptoms of any case, and particular care be taken not to lay too much emphasis upon any symptom which may point to a vesical lesion when in reality the trouble is in the kidney. This is perhaps most likely to occur in a hæmatogenous infection of the kidney by micro-organisms, in which increased frequency of micturition is a marked symptom, whilst the bladder remains quite free from disease. Occasionally, after pus has been present continuously in the urine for some time, it may disappear entirely, the change being accompanied by increase of pain in the side, by an elevation of temperature, or enlargement of the kidney in a case of pyonephrosis, when the obstruction to the flow of urine from that side has become temporarily complete. Very little help is derived from the character of epithelial cells accompanying pus in the urine. The shapes of the cells of the renal pelvis, ureter, and deeper layers of the bladder are so much alike, that it is usually impossible to differentiate them.

Some assistance in the determination of the origin of the pus in the urine may be gained by instrumental examination :

By Catheter.—If a catheter be passed and the bladder washed out with clear solution of boric acid, it will be found that the medium is soon rendered clear if the pyuria is of renal origin, but that it is much more difficult to obtain a perfectly clear medium if the bladder is the seat of the suppuration. If the medium is cleared quickly, but yet, after some ten minutes' retention in the bladder is again found to be turbid, the pus is almost certainly descending from the kidney.

The Cystoscope.—Much more certain evidence is gained, however, by a careful cystoscopic examination. By this means it can be determined in the great majority of cases if the bladder is infected or if any ulceration is present. In a few cases the bladder may be so affected that only a small dilatation is allowed, or bleeding is produced so easily that cystoscopy is rendered futile ; in these cases there will be little need for an inspection of the bladder. If the bladder be found to be normal, evidence of a suppurative lesion in the kidney may be obtained from the appearance of the ureteric orifices or by the variations in the character of the urinary efflux from them. Instead of the normal forcible flow of clear urine from each orifice, mixing with the medium in the bladder in a characteristic swirl, urine containing pus may be seen emitted, appearing in the field as a small smoky puff from the orifice (*Plate XV*, *Fig. B*, p. 282); pieces of muco-pus may be seen to pass from the orifice, or the turbid urine may be seen to leave the orifice in a gentle trickle instead of a jet if the renal secreting function is impaired or if renal dilatation is present.

Apart from the alterations in the urinary efflux from an orifice, the actual appearance of the orifice may show changes which indicate renal disease. Thus, in pyelitis, the margins of the orifice are slightly œdematous and congested, and appear to pout into the bladder (*Plate XV*, *Fig. C*. p. 282); the mucous membrane of the bladder, immediately below and internal to the orifice, is frequently congested or granular from the effect of the altered urinary flow upon it. If the renal pelvis and ureter are dilated, the orifice is usually elongated and patulous, whilst in tubercle or in diseases in which the ureter is thickened, the whole ureteric orifice is drawn upwards and outwards from its normal situation (*Plate XV*, *Fig. D*, p. 282), and is seen at the apex of a conical retracted area in the bladder base.

PYURIA CAUSED BY DISEASES OF THE URINARY ORGANS.

Renal Disease. *Diseases of inflammatory origin.*

Pyelitis and *pyelonephritis* may arise as an ascending infection from the lower urinary tract, especially when there is some obstruction to the normal passage of urine. Thus it is common in cases of prostatic enlargement and stricture. When cystitis is present, it is usually bilateral, although one kidney may show much more advanced disease than the other. Any growth or lesion in the bladder which is accompanied by suppurative infection, and which involves the ureteric orifice, such as vesical epithelioma, or the direct involvement of one or both ureters in the spread of uterine cancer, may set up pyelitis in the kidney, the infection ascending either by the ureter or by the peri-ureteric lymphatics.

In this group of cases the primary cause of the disease has usually advanced to a sufficiently late stage to be obvious, and the symptoms of suppurative ascending infection of the renal pelvis or renal tissues are usually overshadowed by the symptoms of the disease causing the obstruction. Aching in the loin, rigors or raised temperature, tenderness on deep palpation in the renal area, or actual renal enlargement, are usually indicative of renal infection. The urine is often increased in quantity, of low specific gravity, and the daily excretion of solids is lessened; the skin is dry and harsh and the tongue glazed.

Pyelitis and pyelonephritis may also arise as an infection of the kidney apart from any other disease in the genito-urinary organs. Infection is conveyed to the kidney by the blood-stream (hæmatogenous form), and is not uncommon in acute fevers, or with mild forms of suppuration in other parts of the body, or in association with pregnancy. In the less acute forms a pyelitis may result, as in typhoid fever, but in most cases the hæmatogenous infection produces first a suppurative process in the renal parenchyma, from which infection spreads to the calices and pelvis. This form of disease has been shown by recent work to be due most frequently to the colon bacillus in association with affections of the intestinal canal, less frequently to the staphylococcus, streptococcus, pneumococcus, typhoid bacillus, or *bacillus proteus*. The renal pyelitis which ensues when a calculus has ulcerated into the renal pelvis is truly a hæmatogenous infection.

Acute hæmatogenous infection of the renal pelvis without involvement of the renal parencyhma usually begins with slight rigors, tenderness in the loin, and increased frequency of micturition. The urine is faintly turbid and opalescent, does not settle to a pronounced sediment, but remains of a sheeny appearance. It contains numerous bacteria, a little pus, and a little albumin. When the infection first attacks the renal parenchyma as well as the pelvis, the symptoms are much more severe, and the patient may become uræmic rapidly. In the less acute cases, small foci of suppuration occur, which coalesce to form an *abscess*, with the general symptoms of suppuration. Renal abscess may also result from injury when an effusion of blood in the renal tissues becomes infected by pyogenic micro-organisms, or by the breaking down of a renal infarct.

Pyonephrosis—or dilatation of the pelvis and calices of the kidney with pus and urine—is caused when suppuration has occurred in a kidney which is at the same time subjected to some form of obstruction to the normal passage of urine. Pyonephrosis is caused most commonly by renal calculus or tuberculosis, but is by no means uncommon with a chronic cystitis, complicating urinary obstruction from an enlarged prostate or stricture. Carcinomatous ulceration affecting a ureteric orifice, either primary in the bladder or by direct extension of uterine cancer, is also a comparatively common cause of pyonephrosis. In contradistinction to suppurative pyelonephritis, the symptoms of pyonephrosis are less severe ; at first they are those of the obstructive lesion causing the disease, to which are added the general symptoms of suppuration. Pyonephrosis causes a renal tumour of variable size, whilst in the same patient distinct intermittence in size may be observed, a decrease being associated with the discharge of a larger amount of pus in the urine. In pyonephrosis due to calculous disease the urine may contain a large amount of pus, but there may be no lumbar pain suggesting a renal stone. In these cases a large calculus will usually be found in the renal pelvis, and will be shown on *x*-ray examination (*Fig.* 133, p. 279).

The urine in suppurative disease of the kidney and its pelvis requires careful examination. It may be normal with a localized cortical renal abscess or with closed pyonephrosis ; in all other lesions it contains pus and micro-organisms. If the pus-cells are found in the form of casts of the renal tubules, infection of the renal parenchyma is present, whilst in this latter the albumin in the urine is in excess of that due to the pus present. Polyuria, with a diminution of the total solids of the urine in a daily examination, is commonly present in inflammatory lesions of the renal tissue.

Renal Tuberculosis.—The miliary form of tuberculosis occurs in children as part of a general dissemination of tubercle, and causes no urinary symptoms. The kidney is, however, attacked not infrequently by primary tuberculous infection, beginning as a unilateral deposit of small tuberculous nodules. These enlarge and coalesce to form a caseating area, which eventually opens into the renal pelvis by direct ulceration of a calyx to discharge its contents by the urine, when the lining membrane of the renal pelvis and ureter become infected with tubercle and thickened by submucous infiltration. At first, before ulceration into the renal pelvis has occurred, the symptoms of the disease are very slight ; there may be aching pain in the loin and slight albuminuria, but as soon as the renal pelvis is involved, more marked symptoms occur—including persistent pyuria, lumbar aching, increased frequency of micturition, and polyuria. The urine is pale, of low specific gravity, and of opalescent turbidity ; by careful examination after centrifuging, the tubercle bacillus is usually found. A small amount of blood is generally present. The increased frequency of micturition occurs before any descending vesical infection has occurred, and this symptom, accompanied by pyuria, has frequently given rise to a diagnosis of vesical disease. The occurrence in a young adult patient of persistent pyuria which is not due to gonorrhœa, injury, or stone, should always be looked upon with grave suspicion, and a careful search made for the tubercle bacillus ; should this not be found by the microscope, inoculation experiments into guinea-pigs should be conducted. A careful examination of the bladder should also be made by the cystoscope, when early vesical tuberculosis may be seen (*Plate XV*, *Fig. E*, p. 282), or the characteristic changes in the ureteric orifice may show the presence of renal infection (*Plate XV*, *Fig. D*). By digital examination per rectum, the lower end of the ureter may be felt to be thickened and rigid in renal tuberculosis.

Renal tuberculosis is often confounded with *renal stone*, and the colic which is usually associated with stone may be present in tuberculosis if a piece of caseous débris be passed down the ureter. A skiagraphic shadow of a calculus shows well-defined margins (*Fig.* 133, p. 279), whereas a tuberculous focus in the kidney may give rise to a faint, blurred, indistinct shadow in the renal area (*Fig.* 134, p. 280). The presence of tubercle bacilli will, however, determine the existence of tuberculosis, whilst tuberculous lesions elsewhere in the body, most frequently in the testes, prostate, or vesiculæ seminales, may also serve to confirm the diagnosis.

The symptoms of renal calculus vary with the position of the stone and the changes that have taken place in the kidney in consequence of its presence. It may be situated in the renal parenchyma, and cause no symptoms beyond lumbar aching ; or in the renal pelvis, when, if movable, it may cause acute renal colic, due either to the attempted passage of the stone by the pelvic outlet or to the increased intrarenal pressure from blockage of the ureter. So long as the kidney remains aseptic the urine contains only a microscopic trace of blood ; but if it becomes infected with micro-organisms, pyelitis, pyelonephritis, or pyonephrosis may result, with their attendant symptoms. Pus only occurs in the urine in a case of renal stone when infection of the kidney has occurred.

Ureteric Calculus.—A small renal calculus may become impacted during its passage along the ureter, and may cause some difficulty in diagnosis. The usual situations of the obstructed calculus are in the upper few inches of the ureter, at the pelvic brim, or at the vesical end of the tube; in most cases the previous history of renal colic and symptoms of renal stone will be sufficient to indicate its partial ureteric descent. A calculus may, however, be present in the upper end of the ureter or at the pelvic brim, and give very few symptoms beyond a fixed pain in the course of the ureter; in the latter situation it has frequently been mistaken for ovarian pain or for chronic appendicitis. If the stone blocks the ureter completely, the kidney of the same side—in the absence of septic infection—becomes functionless and atrophies; but if the calculus only partially occludes the lumen of the tube, renal distention will occur, with resulting uro- or pyo-nephrosis. If, however, the calculus becomes impacted in the vesical segment of the ureter, a train of symptoms occurs simulating vesical stone or vesical tuberculosis; namely, increased frequency of micturition, penile pain following micturition, and often a small amount of blood and pus in the urine, in addition to the aching pain in the loin. A ureteric calculus impacted in this situation may often be felt in the ureter upon a rectal or vaginal examination; it may be demonstrated by the *x*-rays (*Fig.* 192, p. 455); whilst the changes seen around the ureteric orifice, and the absence of a vesical lesion on cystoscopic examination, will confirm the diagnosis.

Vesical Diseases.—Pyuria may occur in any lesion of the bladder which is associated with inflammatory changes. The fact that urine is retained in the bladder renders the latter much more liable to septic infection, so that cystitis is common with urethral stricture or prostatic obstruction. Any ulceration of the bladder, tuberculous or malignant, is also accompanied by inflammatory changes, when pus will be present in the urine.

Cystitis may be acute or chronic, and the essential factor of either form is the infection of the bladder by some micro-organism; any agent which produces either congestion of the bladder or retention of urine, acts as a predisposing cause.

With *acute cystitis* the mucous membrane of the bladder becomes œdematous and highly congested, and epithelial desquamation and formation of pus rapidly follow. Hæmorrhage may occur from the congested mucosa, or small abscesses develop in it and rupture into the bladder, to leave small areas of ulceration. In severe cases, patches of the mucous membrane may become gangrenous. The symptoms of acute cystitis are usually distinctive: frequent and painful micturition, elevation of temperature, pain in the perineum and suprapubic area, with the presence of pus and blood in the urine, which is commonly of an acid reaction. Usually, some distinct cause for the onset of acute cystitis is apparent, such as some form of acute urethritis or of previous instrumentation, and there is little difficulty in the diagnosis. The same symptoms are, however, produced by an acute inflammation of the prostate which, in nearly all cases, is preceded by acute urethritis; the presence of swelling of the gland, and acute pain on rectal palpation, will determine the presence of prostatic inflammation.

Chronic cystitis may succeed acute. The symptoms are less marked, but increased frequency of micturition is always present. The urine is alkaline, contains pus and mucus, and the disease is commonly associated with some form of urinary obstruction, or with retention or incontinence due to some nervous disease, such as tabes dorsalis or transverse myelitis. The possibility of retroversion of a gravid uterus should not be overlooked. The association of pyuria and increased frequency of micturition, which is present in chronic cystitis, must be distinguished carefully from that due to pyelitis or pyelonephritis, for increased frequency of micturition may be present without any vesical infection. In renal pyelitis, the urine is usually acid in reaction, pale in colour, and shows a general turbidity, with little inclination towards a deposit at the bottom of a specimen. The urine of chronic cystitis is alkaline, and rapidly deposits a greyish sediment of pus. In pyelitis and pyelonephritis, the urine contains more albumin than the pus would account for, and on microscopic examination renal or pus casts are frequently found, whereas in cystitis the albumin is less, and vesical cellular elements are present, without casts unless the kidneys are affected also. Further evidence may be obtained by the use of the *cystoscope*. In cystitis the bladder wall is trabeculated and the mucous membrane thickened; it has lost the normal iridescent appearance, and the vessels of the mucous membrane are obscured. With pyelitis, the bladder wall is normal, but the ureteric orifice of the affected side shows the thickened or pouting lips and slightly raised area of thickened mucous membrane whilst the urine flowing from the orifice may be seen to be turbid or to contain small particles of muco-pus.

Chronic cystitis may be simulated by an inflammation of the posterior urethra. In such a case there is almost always a history of urethral infection, and the diagnosis can be made by urethral irrigation. The patient is directed to retain his urine for some three hours, and after irrigating the anterior urethra as far as the compressor muscle with sterile water or boric acid lotion, the urine is passed into two glasses. With posterior urethritis, the urine contained in the first specimen will contain shreds of muco-pus, whilst that of the second specimen is clear ; whereas, with cystitis, the second specimen will be as turbid as, or even more turbid than, the first.

Tuberculous cystitis occurs usually in young adults. The characteristic symptoms are increased frequency of micturition during both day and night, pyuria, with pricking pain in the glans penis at the end of micturition, and the appearance of a few drops of blood in the last drops of urine. The same symptoms are often present with vesical calculus and with vesical epithelioma, when ulceration has taken place. *Vesical calculus* is usually present in older patients, and during the early part of the illness, before cystitis has set in, the calculus only gives rise to penile pain and desire to micturate during movement. When cystitis supervenes, the frequency of micturition will be marked during both day and night. *Vesical epithelioma* also occurs in older patients, and when ulcerated may cause hæmaturia ; frequently the diagnosis may be made by palpation per rectum of an indurated area in the bladder base, or of some enlarged glands in the pelvic lymphatic space. Tuberculous cystitis in the early stages, when the disease is characterized by the deposition of greyish tubercles in the submucous coat of the bladder, may give rise to increased frequency of micturition without other symptoms, but in the progressive advance of the disease the tubercles enlarge, coalesce, and ulcerate on the surface, by which time pus and blood will be present in the urine, and tubercle bacilli should be found. It may be taken as a general rule that in any patient of young adult life with increased frequency of micturition and pyuria, a careful search should be made for tubercle bacilli in the urine, and for other tuberculous lesions, especially in the testes, prostate, or vesiculæ seminales.

Tuberculous cystitis is much less often a primary disease than secondary to other lesions in the genito-urinary apparatus—most commonly to tuberculous disease of one kidney, when, after the primary focus has ruptured into the renal pelvis, the lining membranes of the latter, of the ureter and of the bladder become affected successively. The diagnosis between primary renal and primary vesical tuberculosis is very often difficult, for when the renal focus has ulcerated into the pelvis, and descending infection has commenced, the symptoms of the two affections are very similar. Thus, with renal disease, persistent pyuria, increased frequency of micturition, and penile pain at the termination of urination, may be present before the bladder shows any sign of disease ; blood is usually present in small quantity in the urine, but its amount is not so definitely greater in the urine passed at the end of micturition as is the case in vesical disease. In renal tuberculosis there may be tenderness in the loin, the kidney is usually enlarged, and the lower end of the ureter can be felt distinctly thickened upon rectal or vaginal examination. The two conditions can usually be diagnosed by a careful cystoscopic examination. In vesical tuberculosis the deposition of submucous tubercles, together with the shallow ulceration in the bladder mucous membrane, may be seen (*Plate XV*, *Fig. E*, p. 282), whilst in renal tuberculosis, changes may be seen in the ureteric orifice of the affected side (*Plate XV*, *Fig. D*). At first the orifice becomes thickened, œdematous, and slightly patulous ; but later it is rigid and patent, or drawn up by the shortening of the ureter to occupy a position above and outside the normal situation in the trigonal area of the bladder, or drawn up to the apex of a conical retraction of the bladder base. When tuberculous cystitis is secondary to lesions in the testes, prostate, or vesicles, the disease commonly begins in the epididymis of one side, and spreads to the vesicle or prostate, whence a focus may directly ulcerate into the bladder. The patient will first notice increased frequency of micturition and vesical pain, followed by an attack of hæmaturia when actual ulceration into the bladder base occurs ; the formation of a tuberculous ulcer in the bladder leads to pyuria and the other symptoms mentioned above. This sequence is by no means uncommon ; the history of testicular disease and the evidence obtained by rectal examination will serve to indicate the nature of the condition.

Vesical calculus may give rise to pyuria when it is accompanied by cystitis, but may be present a long time before any inflammatory infection occurs. When cystitis is present, the urine shows no features which will distinguish it from that of patients suffering from some other form of cystitis, except that there may be a constant presence of crystals, or an increased amount of blood after exercise. The constant symptoms of vesical calculus are vesical irritability during the day time, penile pain after micturition, and hæmaturia, especially after any exercise. If a calculus in the bladder is suspected, examination by the x-rays (*Fig.* 135, p. 282), a sound, or the cystoscope, will reveal it; the cystoscope may detect a stone that is in a diverticulum, partially encysted or lying in the pouch behind an enlarged prostate, where it may easily be overlooked in searching the interior of the bladder with a sound.

Ulceration of the bladder, apart from tuberculosis and epithelioma, may occur as a simple ulcer, consecutive to chronic cystitis, or as the result of injury. A single non-tuberculous ulcer, similar to gastric ulcer, has been described as occurring in young adults in the neighbourhood of the ureteric orifices, causing hæmaturia and painful frequent micturition. Later, the surface of the ulcer becomes encrusted with phosphatic material, when the urine contains muco-pus, and often small flakes of phosphatic débris from the surface of the ulcer. This single ulcer is rare, and can only be diagnosed by the use of the cystoscope. Ulceration may also occur in the bladder as a result of severe cystitis, when necrosis has occurred in the mucous membrane. This condition is present occasionally in a case of obstinate cystitis, giving rise to painful and frequent micturition, and may be diagnosed by means of the cystoscope. Both the simple and the consecutive ulcer must be differentiated from tuberculous ulceration of the bladder; in the latter, hæmorrhage is usually slight, and occurs at the termination of micturition; tubercle bacilli may be found in the urine, or other deposits of tubercle found in the epididymis, prostate, or seminal vesicles. The cystoscopic appearance of tuberculous disease, and its more generalized distribution in the vesical wall, will afford the strongest evidence in the diagnosis.

Malignant ulceration of the bladder occurs in two distinct forms: (*a*) The infiltrating epithelioma; (*b*) The villus-covered carcinoma.

(*a*). *The infiltrating variety* occurs as an ulcer, with raised edges and uneven necrotic surface, usually at the base of the bladder. It is usually met with in men over fifty years of age, causing increased frequency in micturition, pain at the glans penis following micturition, with blood and pus in the urine. The bladder-wall in the vicinity of the ulcer is densely infiltrated, and frequently can be felt on digital examination per rectum, whilst at the same time the lymphatic glands in the pelvic space may be felt to be enlarged.

(*b*). *The villus-covered carcinoma* of the bladder is not uncommon, and gives rise to irregular profuse hæmorrhages. The tumour is attached to the bladder by a broad pedicle, or may be entirely sessile and covered by blunt villi, presenting a coarsely mammillated surface. It occurs in elderly patients, and the tumours are frequently multiple. The surface is often necrotic, giving rise to pyuria. The diagnosis is not difficult, the frequently recurring hæmorrhages in the urine, associated with increased frequency of micturition, pain, and pyuria in an elderly patient, being fairly distinctive. Not uncommonly there is unilateral renal aching from the interference, by the position of the growth, with the flow of urine from one ureteric orifice, so that renal disease may be suspected; but in all cases a careful cystoscopic examination will show the nature of the disease. Difficulty may be experienced in obtaining a satisfactorily clear medium for a cystoscopic view, but in most cases this can be accomplished by gentle manipulations, or by the use of a styptic such as adrenalin 1–1000, or silver nitrate 1–1000. Difficulty may be found in distinguishing cystoscopically between a benign papilloma and villus-covered pedunculated carcinoma; but the broad attachment of the latter to the bladder, the stunted villi covering it, and the multiplicity of the tumours, will be signs of malignant disease (*Plate XVI*, *Fig. F*, p. 284). In rare instances a *benign papilloma* may begin to slough on the surface, or may be accompanied by cystitis, when pyuria will be present. A cystoscopic examination will reveal the diagnosis. Microscopical examination of the urinary deposit may show distinctive fragments of new growth.

Bilharzia hæmatobia may cause pus in the urine in advanced cases. When the small nodules in the submucous tissues (*Plate XVI*, *Fig. K*, p. 284) of the bladder ulcerate, small fungating masses are found in the bladder. The typical ova in the urine (*Fig*. 26, p. 79), in addition to pus and blood, will be found on microscopical examination of the urinary sediment.

Urethral Causes.—Any condition which sets up a purulent urethritis will cause pyuria. If the urethritis is recent or profuse, the local condition will be enough to indicate the diagnosis, but it must be remembered that cystitis may complicate a case of urethritis by direct backward infection. If, in addition to urethral discharge, there is increased desire to urinate, suprapubic pain, or hæmaturia, acute cystitis is probably present. The anterior urethra should be irrigated well with sterile water or boric acid lotion, and the patient then directed to pass urine into two glasses. If the first portion passed contains pus and the second is clear, infection is present in the posterior urethra and not in the bladder, but if both specimens are turbid with pus, cystitis is present.

The onset of acute prostatitis complicating urethritis gives rise to increased desire to micturate, and perineal and suprapubic pain, in addition to pyuria, or may cause retention of urine. Digital examination of the prostate, per rectum, will show the prostate to be enlarged, and very painful.

A small amount of pus may be present in the urine in cases of chronic urethritis which is not sufficient to cause any visible discharge from the meatus. The anterior urethra should be irrigated well, and the urine again passed into two separate glasses, when, if the first washings from the urethra contain pus, there is infection in the anterior urethra ; if the first specimen of urine contains pus but the second is clear, there is infection in the posterior urethra ; whilst if both contain pus, cystitis is present. In any case of urethral discharge, a bacteriological examination should be made for the organism causing the infection, for it is far from uncommon to find that an apparent gonorrhœal urethritis is in reality due to staphylococcal infection.

Pyuria is commonly present in cases of stricture of the urethra, from the co-existing urethritis or cystitis.

PYURIA CAUSED BY DISEASE OUTSIDE THE URINARY ORGANS.

Pus may be present in the urine, apart from any disease in the urinary apparatus, either by accidental contamination of the urine, or by the direct spread of inflammatory or carcinomatous processes from neighbouring organs to the urethra, the bladder, or more rarely the ureter. In the male, the accumulation of pus behind a *phimosis* may account for pyuria, or in the female a *leucorrhœal discharge* may contaminate the urine. In the latter case the vulva should be cleansed well with an antiseptic, and a catheter passed to obtain a specimen for examination.

The spread of inflammatory processes, or the actual rupture of an abscess into any part of the urinary tract, will cause pyuria, and may create considerable difficulty in diagnosis. If symptoms pointing to urinary trouble, such as markedly increased frequency of micturition or slight hæmaturia, be followed by the sudden appearance of a quantity of pus in the urine, there is strong probability of the *rupture of an extra-urinary abscess* into the bladder or urethra, provided that the sudden emptying of a renal abscess or a pyonephrosis can be eliminated. Frequently the history of any case will give some indication of the primary trouble, of which the most frequent are prostatic abscess, appendical abscess, pyosalpinx, psoas, iliac, or pelvic abscess.

Prostatic abscess is most frequently a sequela of an acute urethritis which has infected the posterior urethra and caused an acute prostatitis. It may be due to a gonorrhœal or to a septic venereal infection, or may result from septic instrumentation in the urethra. An acute prostatitis is very prone to result in the formation of an abscess which may rupture into the urethra, bladder, or rectum, unless appropriate surgical measures be undertaken, The onset of acute prostatitis is marked by increasing desire to micturate, pain in the perineum and hypogastric areas, and raised temperature, whilst, per rectum, the prostate is felt to be uniformly enlarged and very tender. If an abscess result, there may be rigors and increased difficulty in micturition, even retention of urine, whilst a soft area may be felt in the prostate from the rectal aspect. A prostatic abscess may occur more rarely in connection with a *prostatic calculus* ; or may be present in advanced *genito-urinary tuberculosis*, when a prostatic focus may caseate and ulcerate into the trigonal area of the bladder, a condition which is usually accompanied by a sharp attack of hæmaturia. A tuberculous focus in the prostate is commonly a comparatively late feature in the disease, and the presence of nodules in the epididymis or seminal vesicles, or the previous knowledge of vesical tuberculosis, will assist very largely in the diagnosis.

Pyuria in Inflammation of the Vermiform Appendix.—In the usual position of the appendix the bladder is commonly not affected ; but if the appendix passes downwards across the pelvic brim, it is not uncommon to find that should it become inflamed, the patient complains of frequent and painful micturition. The appendix may be adherent to the bladder, when the latter will show on cystoscopic examination a localized area of acute congestion on the right lateral wall, and both pus and blood may be present in the urine ; further, a small abscess may be formed in the adhesions between the appendix and the bladder, ulcerating into the latter and giving rise to pyuria. Two such cases have come under the care of the writer, when the association of frequently recurring attacks of pain low down in the right side of the pelvis, with increased frequency of micturition and pyuria, had given rise to the suspicion of ureteral calculus. In each case a cystoscopic examination showed a normal ureter, and a small ulcer in the right lateral wall of the bladder, surrounded by an area of acute cystitis. The diagnosis of these cases is by no means easy ; in the first place the situation of the pain is lower in the pelvis than is usual with appendicitis, whilst the association with urinary symptoms rather points to vesical disease ; but the character of the onset of the trouble, with elevation of temperature and pulse-rate, and right-sided abdominal rigidity, will point to an acute intra-abdominal lesion. In other cases, again, an abscess resulting from appendicular suppuration may track down into the pelvis and, if unopened, may rupture into the bladder. In these cases, there will be the usual history of acute appendicitis, followed by a tumour in the right iliac fossa or pelvic space, with a continuance of pyrexia, or even rigors, which subside on the appearance of a large quantity of pus in the urine.

A *pyosalpinx* may rupture into the bladder or cause cystitis from direct spread of the inflammatory process to the bladder. There will usually be a history of leucorrhœa, with constant aching or dragging pains in the lumbo-sacral region, with more severe attacks of pain and malaise at intervals. The periods may be profuse and associated with more pain than usual, and on vaginal examination a distinct fullness or tumour may be felt in one or both fornices.

Psoas or *iliac abscess* may rupture into the bladder, and a psoas abscess has been known to open into a ureter ; but the swelling in the iliac fossa or inguinal region, together with signs of spinal caries, will point to the condition.

Carcinoma of the neighbouring organs in the pelvis frequently attacks the bladder by direct spread of the growth. This is most common in carcinoma of the uterine cervix and of the rectum, but may result from cancer of the pelvic colon, sigmoid, or cæcum. In any case, the spread of the disease to the bladder occurs late in the disease, so that symptoms of the initial trouble are sufficiently manifest to point to the diagnosis. The implication of the bladder is shown first by an increased desire to pass urine, and by pain during the act ; later, when the growth has actually infiltrated the vesical mucous membrane, ulceration into the bladder occurs, with the passage of pus and blood in the urine. If the growth has extended from the uterus or vagina, there may be a leakage of urine into the latter ; or if from the rectum or colon, some fæces or flatus may be passed per urethram.

Tuberculous or *dysenteric ulcers of the small intestine* have in some instances become adherent to the bladder wall, and caused cystitis by direct spread, or have even perforated into the bladder. *R. H. Jocelyn Swan.*

RAINBOW VISION.—(See VISION, DEFECTS OF, p. 762.)

RASHES.—(See ERYTHEMA, p. 222; PUSTULES, p. 557; VESICLES, p. 753, ETC.)

REACTION OF DEGENERATION.—In testing muscles and nerves electrically, two different kinds of current are employed, namely : *faradic*, in which there is a very rapid alternate making and breaking of the current, and the *galvanic*, in which the current flows continuously until it is voluntarily interrupted by the operator. The faradic current excites the nerve and muscle continuously all the time it flows ; the galvanic current only excites when it is made and when it is broken ; not whilst it is flowing. In the case of the faradic current there is no difference between the poles, each being alternately an anode and a kathode many times a minute ; in the galvanic current, on the other hand, the pole connected to the zinc of the battery is known as the kathode, and it is by this that the current leaves the body, whilst the other pole is known as the anode, and by it the current enters the body. When testing muscles or nerves, it is usual to have one pole in contact with an indifferent part, such as the spine, and the other over the motor point of the muscle or nerve to be tested. Broadly speaking, the best spot for stimulating a nerve is the place where it is most superficial, and for a muscle, over the site of entry of its motor nerve. It is important to have the skin well wetted, to minimize its resistance to electrical conduction ; and the strengths of current required to produce contractions should be measured by a galvanometer, without which the relative excitabilities of the nerves and muscles of the two sides of the body cannot be compared.

Under normal conditions, both faradic and galvanic currents produce brisk contractions of a muscle when applied either to it or to its nerve ; and with galvanism it is found that a weaker current will suffice to evoke a contraction on making the circuit when the kathode is on the muscle or nerve than when the anode is similarly employed. This is usually summarized by the formula K.C.C. > A.C.C., which means " the kathodal closure contraction is more easily obtained than is the anodal closure contraction." When the nerve is degenerated, however, there is a change in these electrical reactions, and when there is complete reaction of degeneration—often written and spoken of as R.D.—stimulation of the nerve itself evokes no muscular contractions whether the faradic or the galvanic current is employed, stimulation of the muscle evokes no contraction when the faradic current is used, whilst with galvanism the muscle can still be made to contract, though its method of response differs from the normal in the following respects :—

1. It may be evoked by a strength of current less than the healthy minimum.
2. The twitch of the contraction is slow and sluggish, instead of brisk and quick.
3. It may be evoked at least as readily when the pole upon the muscle is the anode as when it is the kathode ; this is expressed by the formulæ A.C.C.=K.C.C., or A.C.C. > K.C.C., the latter meaning that the anodal closure contraction is obtained from a smaller current than the minimum required for the kathodal closure contraction.

In this connection, however, two considerations require to be understood clearly. In the first place, if a given nerve were cut across with a knife, there would be no immediate R.D. ; it takes a week or more for the process of nerve degeneration to reach the stage that produces R.D. ; it then depends upon what happens to the nerve how long the R.D. persists ; if regeneration occurs, it takes from twelve weeks onwards to complete itself, and R.D. will be found all that time ; if the nerve does not regenerate, then R.D. may persist for two or three years or more, provided that the muscle fibres are kept, by massage and electrical treatment, from becoming mere strands of fibrous tissue. Should the latter change ensue, there will be no more electrical response in the fibrous tissue that used to be muscle than there would be in any other fibrous tissue.

In the second place, it happens, as often as not, that when some fibres in a nerve trunk degenerate, others do not, and the same applies to the corresponding muscle fibres. It follows that there will then be a mixed reaction, the normal fibres giving a normal response, the degenerated fibres giving R.D. ; the greater the proportion of degenerated fibres, the nearer will the reactions obtained approach to complete R.D., and vice versa. The result is spoken of as *partial R.D.* ; some excitability both of the nerves and of the muscles to faradism remains, but it is less than normal ; the nerve responds to galvanism, but not so readily as does the muscle when the latter is stimulated directly ; the response of the muscle will be less brisk than normal, and yet K.C.C. may still be more easily obtained than A.C.C. It is by no means easy to be sure of the interpretation of a partial R.D., but partial is commoner than complete R.D.

The chief use of R.D. is in distinguishing muscular atrophy due to organic changes in the lower neuron from other cases of atrophy, especially when the latter is due to general wasting from cachexia, or to arthritis, or disuse, or a primary muscular dystrophy. When R.D. is present there is a lesion in the lower neuron, either in the anterior cornual cells, in the anterior nerve roots, or in the peripheral motor nerve fibres. The differential diagnosis of the various affections of these parts is discussed under ATROPHY, MUSCULAR (p. 59).

It remains to add that there are a few maladies in which the electrical reactions are peculiar, though they do not present R.D. In tetany, for instance, Erb has shown that A.C.C. is often greater than K.C.C., although in other respects the reactions are normal In Thomsen's disease there is variability in the polar responses, the original contraction produced on closure lasting a long while, and sometimes developing into a series of wave-like movements during the continuance of the passage of the constant current ; but excitability to faradism remains. It is a rare malady, but one so characteristic that it is recognized easily; the chief feature of it is slowness of the relaxation of the muscles when they are first used after a period of rest. When the patient starts to rise from a chair, for example, he does so very slowly and as though he were stiff; the muscles are unduly rigid, and the first few steps he takes are consequently awkward and very slow ; after a few seconds the peculiar delay in relaxation passes off and ordinary walking becomes possible. After sitting down again for a while, the same difficulty of rising and starting to walk ensues; and the trouble is generally persistent. There is no pain as a rule, which distinguishes the condition from true stiffness, ankylosis, or rheumatoid arthritis, which might otherwise be diagnosed in error. The legs are nearly always affected more than other parts, and the main complaint is that of difficulty in starting to walk or otherwise use the legs, this difficulty passing off after a few seconds or minutes.

In some cases of Raynaud's disease, and in angio-neurotic œdema and allied vasomotor neuroses, there may be variations from the normal galvanic reactions. In myasthenia gravis (*Figs.* 111, 112, p. 235) it is characteristic that, whereas the affected muscles respond readily to the first few faradic stimuli, the contractions diminish rapidly in size and cease after a few minutes, notwithstanding the continuance of stimulation. After a period of rest this myasthenic reaction is obtainable again, and so on. This type of electrical response corresponds precisely to the rapid fatigue of the voluntary muscle movements, and the diagnosis is not difficult, though the disease is rare. *Herbert French.*

RECTUM, ABNORMALITIES FELT PER.

Method of Examination.—The patient should be placed in a good light on a couch of convenient height. With male subjects the best position is the knee-elbow, with females the right lateral with the knees flexed and the right arm behind the back. The examination should be made with the left hand, leaving the right free for manipulations. Most diseases of the rectum are situated within two inches of the anus. It is advisable, therefore, that to begin with the finger should be inserted as far as the first joint only, and the lower inch of the bowel examined thoroughly. The examination must not be concluded until the finger has been passed up as high as possible and the whole of the rectum within reach explored, as well as the coccyx, sacrum, ischio-rectal fossæ, and adjoining viscera. The rectal speculum and the sigmoidoscope may also be needed to complete the examination.

If any abnormality be felt, the first thing to ascertain is (1) *Whether it lies free in the lumen or is attached to the wall of the rectum* ; (2) *Whether it is some abnormality of an adjoining structure or viscus that can be felt through the rectum.*

I. ABNORMALITIES LYING FREE IN THE LUMEN OR ATTACHED TO THE WALL OF THE RECTUM.

Foreign Bodies.—Though fæces can hardly be considered as foreign to the rectum, yet a hard, scybalous mass, enterolith, or hair-ball may amount to an abnormality. True foreign bodies include those that have been introduced through the anus, and those that have been swallowed. Examples of the first class are seldom met with, and then are generally in persons of weak intellect. Thieves sometimes employ the rectum as a hiding-place for stolen goods. The majority of foreign bodies felt per rectum have been swallowed —for instance, fishbones, pins, needles, or splinters of wood. Their importance lies in the fact that they may cause a rectal or ischio-rectal abscess, and in treating such a case their discovery and removal is essential for a complete cure.

Swellings of the Rectum projecting into the Lumen:

Internal Hæmorrhoids are rarely palpable to the finger unless chronically inflamed, thrombosed, or gangrenous. If palpable, they will be felt immediately inside the anus, and can be hooked out with the finger and made to protrude through the anal orifice for inspection. The existence of piles having been diagnosed, an effort should be made to see if there is any causative condition, such as a carcinoma in the bowel above.

Abscess (submucous) gives rise to a more or less elongated, smooth, elastic swelling in the rectal wall. It is intensely tender, the slightest pressure causing great pain. The mucous membrane may feel hot, and pit on pressure. If the abscess has burst or bursts during examination, the finger on withdrawal will be covered with pus. An abscess that has already emptied itself feels like a small pea or bean in the submucous tissue.

Polypus is a term used to designate, without reference to its histological characteristics, any benign tumour that is pedunculated. Almost all innocent tumours in this position, even if sessile at the beginning, become pedunculated owing to the downward pressure of the fæces. The passage of blood and mucus, combined with the absence of piles and carcinoma, should lead one to suspect the presence of a polypus. It may not be easy to feel, because its consistency is much the same as that of the mucous membrane, and further, its peduncle may allow such free movement that it may easily be mistaken for a small mass of fæces. The best way of fixing these growths is to sweep the finger round and round the whole circumference of the rectum up to the highest point attainable. The growth is then arrested by the pedicle, and the finger can be hooked round it, so that the growth is drawn down and, if possible, made to protrude through the anus. If the polypus is large, a rectal speculum may be of service. It is to be remembered that polypi are often multiple.

Ulcers, unless malignant or chronically inflamed, can rarely be felt with the finger; they must be exposed to view with the speculum. They may be tuberculous, gummatous, traumatic, or due to ulcerative colitis or dysentery.

Carcinoma occurs usually in people over forty. Its commonest site is within the four terminal inches of the bowel. It is generally hard, fixed, irregular, and nodular. Its extent varies with its stage: it may involve only part of the circumference of the bowel, or may extend right round so as to occlude the lumen and cause a stricture. The surface is usually ulcerating, so that it is friable and bleeds easily. There is nearly always a belt of normal mucosa between the internal sphincter and the neoplasm. Not only the lateral but the upper limit of the growth may sometimes be ascertained by inserting the finger to its extreme limit, care being taken not to split the mass. Another point to be gauged by a rectal examination is the degree of infiltration as measured by the fixity of the tumour to the neighbouring structures, e.g., sacrum and coccyx. Following the rectal examination, the abdomen is to be palpated for evidence of infection of the inguinal, pelvic, or lumbar glands, and the existence of secondary deposits in the liver.

The clinical symptoms of carcinoma of the rectum are very suggestive. The patient generally complains of diarrhœa, the bowels being open five to twenty times a day, and this may have followed on a period of constipation. Notwithstanding the apparent diarrhœa the total amount of fæces passed is very small, and no sense of satisfaction is obtained by the patient after stool. The evacuation may be so rapid as to merit the description 'explosive diarrhœa.' Hæmorrhage from the bowel is common, and in the later stages there is a discharge of mucus. Pain is complained of—a dull aching pain in the rectum and at the bottom of the back, which is not made much worse by the passage of a motion, quite unlike the sharp temporary excruciating pain associated with an anal fissure or ulcer. Emaciation is rapid, and a history of wasting and diarrhœa in a middle-aged patient should always lead to a careful examination of the rectum, and if nothing is to be felt with the finger, a sigmoidoscope should be used. A carcinoma is likely to be overlooked from carelessness and from not making an examination. Mistakes may, however, arise between carcinoma and an adenomatous polypus or ulceration, either traumatic or tuberculous, around which much long-standing inflammation has caused thickening. The facts that a carcinoma is hard, the surface often excavated, and the edges nodular and everted, are generally sufficient. If real doubt exists, a piece of the ulcer may be removed for microscopic report.

Intussusception.—Occasionally a piece of intussuscepted bowel may come down so far as to be felt per rectum. This condition is associated with the passage of blood and mucus, and therefore might be mistaken for a disease of the rectum proper. The fact that intussusception occurs nearly always in children, especially at the age of nine months or thereabouts, and causes intestinal obstruction, should make such a mistake easily avoidable.

Stricture due to a carcinoma is dealt with above, but a few remarks remain to be made about fibrous stricture. This may be present at the anal orifice, at the level of the upper border of the internal sphincter, or three to four inches up the rectum. It may be annular or tubular. The finger meets with a firm cord-like constriction, which perhaps will not allow the entrance of more than its tip ; there will be no bleeding unless the finger is forced through the stenosis and the mucous membrane torn.

Fistulæ, either recto-vaginal or recto-vesical, whether congenital or acquired, may be felt with the finger. The passing of urine or fæces by abnormal passages indicates the complaint.

Malformations of the Rectum.—Some children are born without an anus, or without the lower portion of the rectum, or the finger introduced may be stopped by a membrane separating the upper from the lower portion of the bowel. The diagnosis is obvious. The usual types of abnormalities are shown on the accompanying diagrams, *Figs.* 249–252, reproduced from the *Medical Annual*, 1910 : the figures are a summary by Dr. A. Keith of a series of 54 cases in male, and 52 cases in female, children.

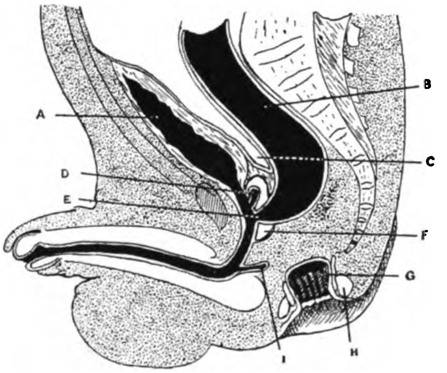


Fig. 249.—Sagittal section of the pelvis of a male child, showing the rectum opening into the prostatic part of the urethra. A bladder ; B rectum ; C recto-vesical pouch ; D uterus masculinus ; E intracloacal anus ; F prostate ; G proctodæum ; H external and internal sphincters ; I Cowper's gland.

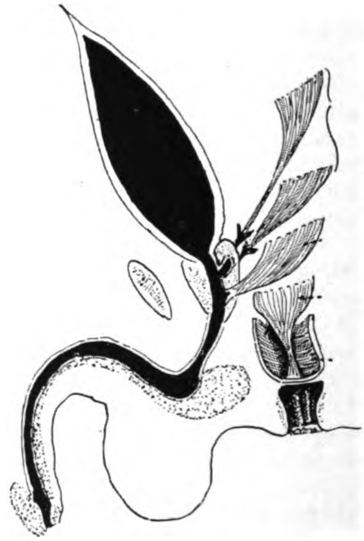


Fig. 250.—Diagram illustrating the degrees of imperfection in the male. The rectum is shaded ; the various degrees make up a series between the proctodæum and base of the prostate.

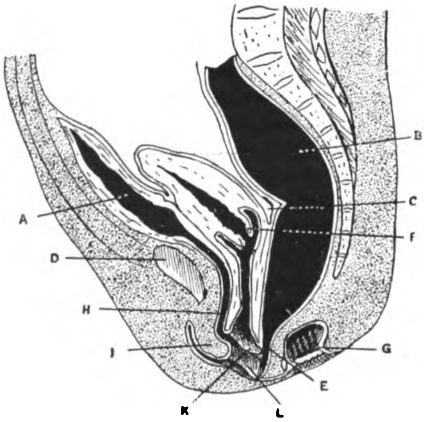


Fig. 251.—Sagittal section of the pelvis of a female infant, showing the rectum opening into the navicular fossa of the vulva. A bladder ; B rectum ; C recto-uterine fold ; D symphysis pubis ; E vulva-anus ; F cervix ; G proctodæum (rarely present if the rectum opens into the vulva) ; H urethra ; I clitoris ; K hymen.

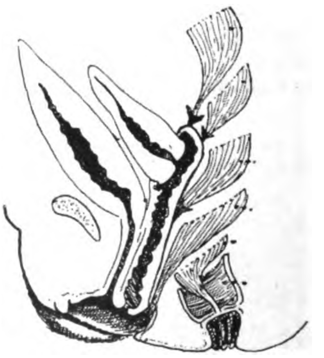


Fig. 252.—Diagram illustrating the common varieties of malformation in the female ; the various degrees make up a series between the proctodæum and the recto-uterine fold.

II. ABNORMALITIES OF NEIGHBOURING STRUCTURES FELT PER RECTUM.

It does not lie within the scope of this article to give the differential diagnosis of all the morbid conditions that can be felt through the rectum ; it suffices to take the structures within reach of the finger, and indicate the varying conditions in which a diagnosis may be aided by a rectal examination.

On the Anterior Wall the structures that can normally be felt are the prostate in the male, and the uterus in the female.

Prostate.—Any enlargement is easily felt. An *adenoma* is the commonest form. This is soft, elastic, and has a groove in the middle line. A *carcinoma* or *sarcoma* is hard and fixed, and the outlines are blurred. A *prostatic abscess* causes a marked painful protrusion into the rectum.

The Vesiculæ Seminales are not palpable normally. The fact that they can be felt is almost sufficient to declare them diseased. They are most commonly affected in connection with tuberculosis of the testes or from present or past gonococcal vesiculitis.

The Bladder is not felt if healthy. If greatly distended it may form a tense resistance in the anterior wall of the rectum. Rarely, a large stone or a malignant growth of the floor may be felt.

The Uterus is easily palpable. Enlargement or retroversion can be recognized ; the pressure of a fœtal head may occlude the rectum.

The Vagina cannot be felt unless it is occupied by a foreign body such as a pessary, or is the seat of a growth.

The Ovaries, if enlarged by cystic disease or by new growth, may come within reach of the finger ; *pyosalpinx* is often a bilateral affection in which the inflammatory masses can be felt per rectum in Douglas's pouch ; they can be detected more readily by vaginal examination, however, when this route is permissible.

Through the Posterior Wall the only structures that can be recognized are the coccyx and sacrum.

The Coccyx may be found bent in and pressing on the rectum. In coccydynia any movement of the coccyx may cause great pain.

The Sacrum may be the seat of either a growth or an abscess, which will cause a bulging into the posterior wall.

On the two Lateral Surfaces no structures are normally recognized. The ischiorectal fossæ are common sites for *abscesses*, and these can be felt as tense swellings pushing in the wall. Rarely an *aneurysm* of the internal iliac artery or a *stone in the lowest portion of the ureter* may be felt.

If anything is felt with the tip of the finger through the upper portion of the rectum, it will usually be something distending Douglas's pouch. This may be blood coming from a ruptured or leaking *ectopic gestation*, or a localized *abscess*, either parametric or arising from a septic Fallopian tube or the vermiform appendix. Some surgeons state that they are able to detect the appendix if it is hanging over the brim of the pelvis, but to do this the finger must be long and the senses very acute.

Sometimes, when there is doubt as to whether symptoms arising in connection with a more distant organ, e.g., the stomach or the gall-bladder, are due to malignant disease or not, rectal examination affords valuable evidence of malignancy even when there are no pelvic symptoms at all. When secondary deposits have arisen, they develop not infrequently in the pelvic peritoneum, presumably as the result of gravitation of malignant particles into Douglas's pouch. These latent secondary deposits can sometimes be felt very definitely as a firm band or shelf—the 'rectal shelf'—if the observer's index finger is a fairly long one. Rectal examination is sometimes of value also in verifying the existence of free fluid in the peritoneal cavity. *George E. Gask.*

RECTUM, BLOOD PER.—(See BLOOD PER ANUM, p. 75 ; and MELÆNA, p. 385.)

REDUPLICATION OF HEART SOUND.—It seldom happens that the diagnosis in a particular case is influenced to any marked degree by the presence or absence of reduplication of either heart sound ; nevertheless, the reduplication is sometimes so definite that it attracts special attention and needs interpretation. It has to be distinguished from other triple sounds, particularly from the canter-rhythm that occurs most commonly with acute pericarditis, and less often with dilatation of the heart from fatty change, especially in pernicious anæmia and other conditions of oligochromæmia ; and from the beginning of a mid-diastolic bruit at the impulse in a case of acute rheumatic endocarditis of the mitral valves. One can lay down no rules as to how these various sounds are to be distinguished; it can only be done by having heard them in other cases; sometimes, indeed, opinions differ as to whether the sounds heard in a given patient are due to a bruit or to a reduplication.

Reduplication of the first sound is rare, and to all intents and purposes it never occurs except at or near the impulse; it indicates some abnormality, but does not specify exactly what that abnormality may be. If there is no bruit, the commonest cause is great hypertrophy of the left ventricle from granular kidney or arteriosclerosis, indicated by the big heart, high blood-pressure, urinary and retinal changes.

Reduplication of the second sound is common, especially in the pulmonary area (second left intercostal space close to the sternum). It generally indicates great relative increase in the intrapulmonary blood-pressure, so that the pulmonary valves close a fraction sooner than the aortic; the reduplication may alternate with simple accentuation (see p. 1), the commonest cause being mitral disease, especially mitral stenosis. Similar reduplication of the second sound may be heard at the impulse also in these cases, though more often the second sound here is weak or inaudible. The commonest cause for reduplication of the second sound at the impulse is great relative increase in the systemic blood-pressure—especially in cases of arteriosclerosis or granular kidney. The second sound in the aortic area (second right intercostal space close to the sternum) is generally very loud and ringing, or even reduplicated at the same time. There are no other really important causes of reduplication of either of the heart sounds. *Herbert French.*

REFLEX, PLANTAR.—(See BABINSKI'S SIGN, p. 68.)

REFLEX, PUPILLARY.—(See PUPIL, ABNORMALITIES OF, p. 551.)

REGURGITATION OF FOOD THROUGH THE NOSE may be but a temporary accident, the result of an unsuccessful attempt to stave off a sneeze, a cough, or a burst of laughter when the mouth is full of food or fluid; or it may result from an explosive return of gas from the stomach or œsophagus, particularly after drinking gassy fluid such as soda-water, champagne, ginger-beer, cider, or beer. In such cases the diagnosis is generally obvious. Pathological regurgitation of food through the nose results from two main groups of causes, namely:—

A. Structural Imperfections of the Palate:—

(*a*) Congenital: cleft palate
(*b*) Acquired perforation: (i) traumatic, (ii) syphilitic, (iii) malignant, (iv) tuberculous.

B. Paresis or Paralysis of the Soft Palate or of the Pharynx:—

(*a*) Post-diphtheritic
(*b*) Post-operative
(*c*) Syphilitic
(*d*) The result of bulbar paralysis
(*e*) The result of pseudo-bulbar paralysis
(*f*) Cases of undetermined cause.

Simple inspection of the roof of the mouth is generally sufficient to decide whether the cause belongs to group *A* or to group *B*. The median and symmetrical imperfection of a congenital cleft palate is obvious, and there is the history of the trouble dating from birth. There may be a harelip or other congenital abnormality at the same time. When an ulcerative process is still in progress there may for a time be some doubt as to whether it is syphilitic, malignant, or tuberculous. The history may help, or the healing of the ulcer under the influence of mercury or iodide of potassium or salvarsan may indicate its syphilitic nature. If it is important to arrive at the correct diagnosis as early as possible, a small portion of the pathological tissue may be excised and examined microscopically, or Wassermann's serum test applied, or scrapings from the ulcer examined directly for the *Spirochæta pallida* or for tubercle bacilli. Tuberculous ulceration of the palate is very rare, and is generally associated either with lupus or with definite phthisis. A new growth of the palate may be either epithelioma, endothelioma, or sarcoma, the distinction between these depending mainly on the microscope.

Diphtheria.—If there is no structural defect of the palate, the regurgitation of food through the nose being due to paralysis, by far the most likely cause is previous diphtheria.

The existence of the latter may have been recognized at the time, but quite often the diphtherial attack has been so slight as either to have caused no definite illness, or else to have been regarded as simple sore throat. The palate alone may be paralyzed, giving rise to a nasal alteration in the character of the voice as well as to the regurgitation ; or there may be paresis of the ciliary muscles and the eyes as well, causing difficulty in reading ; less commonly, there is further evidence of peripheral neuritis affecting the limbs and heart. The trouble may not come on for three or four weeks after the diphtherial attack, and therefore it may no longer be possible to detect Klebs-Löffler bacilli in swabbings from the tonsils or fauces ; but in every such case it is important to look for them, both directly and by means of cultures. Probably not a few cases ascribed to 'influenza,' or to undetermined causes, are really post-diphtheritic. The paresis recovers in time, sometimes quickly, but often not until three months or more have elapsed.

Post-operative Cases.—The history in these cases will point to the diagnosis ; the accident is rare, and as a rule the effects are temporary ; it may happen during the removal of tonsils and adenoids.

Syphilitic Paralysis of the Palate is not common, and it hardly ever occurs by itself. It is a general rule that luetic affections of cranial nerves are multiple and often asymmetrical ; thus there may be strabismus, or a laryngeal paresis, in addition to that of the palate ; or there may be a history or other evidence of syphilis.

Bulbar Paralysis.—When this affects the palate and causes regurgitation of food through the nose, there have generally been other symptoms for some time. The malady is slowly progressive, and starts with paresis of the lips and tongue ; swallowing is difficult, not so much because of the regurgitation as because the tongue is unable to thrust the bolus back between the fauces. The constant dribbling of saliva from the angles of the mouth is characteristic of some cases. The title labio-glosso-pharyngolaryngeal paralysis indicates the usual sequence of events. Bulbar paralysis may be associated with progressive muscular atrophy (p. 61), and it may be distinguished from pseudo-bulbar paralysis by the atrophy of the tongue, which occurs in the former but not in the latter. Bulbar paralysis is due to a lesion in the medulla oblongata, whereas pseudo-bulbar paralysis has very similar symptoms due to bilateral cortical softening. In either case the patients are generally elderly.

Undetermined Causes.—As regards such cases, it may be repeated that the majority are doubtless post-diphtheritic, so that it is important to examine swabbings from the throat of all such patients for the Klebs-Löffler bacillus. The symptom is very rarely hysterical.

Herbert French.

RETENTION OF URINE.—(See MICTURITION, ABNORMALITIES OF, p. 393.)

RETRACTION OF THE ABDOMEN.—(See RIGIDITY OF THE ABDOMEN, p. 592.)

RETRACTION OF THE GUMS is occasionally a symptom which troubles patients very much, but in itself it seldom indicates more than a local affection. In a mild degree it may be due to excessive use of a hard tooth-brush ; in most cases it results from a local infective process, especially tartar, caries of the teeth, or pyorrhœa alveolaris. These conditions are discussed under the heading of BLEEDING GUMS (p. 72), though very often retraction may be present, even in an extreme degree, without actual bleeding.

Herbert French.

RETRACTION OF THE HEAD may be a marked symptom in the following conditions :—

Acute meningitis :
  1. Suppurative
  2. Tuberculous (basal)
  3. Meningococcal (posterior basal)
  4. Meningococcal (epidemic cerebrospinal)
Cerebellar or other subtentorial tumour or abscess
Superior longitudinal sinus thrombosis
Acute encephalitis
Bronchopneumonia with partial asphyxia
Laryngeal obstruction, especially diphtheria in children
Strychnine poisoning
Tetanus
Hydrophobia
Catalepsy
Spasmodic torticollis
Paramyoclonus multiplex
Hysteria and hystero-epilepsy.

In arriving at a diagnosis in any given case, the probability is that *strychnine poisoning* *tetanus*, and *hydrophobia* will either suggest themselves at once on account of other circumstances in the case, or else will not need to be discussed at all. *Hysteria* can only be diagnosed when all other possibilities have been excluded, and probably not until the case has been watched anxiously for a time; there may be other functional symptoms in the case (p. 465); the patient is generally a young adult, more often female than male. *Catalepsy* and *hystero-epilepsy* will be suggested by the mental symptoms, or obvious insanity.

These things being excluded, the first thought that marked and maintained retraction of the head arouses is that the patient has some serious intracranial lesion, probably *meningitis*. Before coming to this conclusion, however, it is important not to forget that extreme dyspnœa in children sometimes produces considerable head retraction, so that the physical signs in the lungs and heart should be noted carefully, *bronchopneumonia* and *capillary bronchitis* being kept specially in mind, and any signs of *laryngeal obstruction* looked for, especially stridor and spasmodic up-and-down movements of the thyroid cartilage, with sucking in of the thorax above and below the clavicles, along the attachments of the diaphragm, and in the intercostal spaces. *Diphtheria*, *foreign body in the larynx*, and *retropharyngeal abscess* have all been mistaken for meningitis.

If there is no evidence of sufficient throat or lung trouble to account for the symptom, an intracranial lesion is probable; and by far the most likely, especially in a child, is acute meningitis, either tuberculous or posterior basal. Symptoms common to all the intracranial affections are headache, vomiting, and giddiness; pyrexia, generalized convulsions, coma, incontinence of urine and fæces, retraction of the head, and optic neuritis; or even local symptoms, especially twitchings, convulsions, or paralysis of individual limbs or parts of limbs, according as one part of the brain or another is more irritated or softened than the rest. If there is an obvious source of sepsis in connection with the cranium, such as otitis media, mastoid abscess, facial erysipelas, a septic scalp wound, boils, pediculi with sores, suppuration in the orbit, nose, antrum of Highmore, frontal, ethmoidal, or sphenoidal air-cells, or nasopharynx, the probability is that any acute meningitic symptoms are due to staphylococcal or streptococcal *suppurative meningitis*; *pneumococcal meningitis* may occur without local sepsis, either alone or as part of a general pneumococcal septicæmia; suppurative meningitis due to the *Bacillus diphtheriæ*, the typhoid bacillus, influenza bacillus, or the *Bacillus coli communis* may occur, but it is decidedly uncommon and clinically indistinguishable from other forms of suppurative meningitis, in all of which marked pyrexia and a fatal ending in two or three days are the rule. *Tuberculous meningitis* is much commoner in childhood than it is at any other age; it is always part of a general tuberculosis, and it is very rare in adult life. At first there may be no pyrexia, though this depends on the caseous glands and tuberculous lesions in the lungs and elsewhere more than upon the meningitis. The early diagnosis is apt to be uncertain, but as the days go by the serious nature of the complaint generally becomes obvious; the effortless vomiting, the irregular pyrexia, severe headache, optic neuritis, retracted head, possibly choroidal tubercles (*Plate XX*, *Fig. w*, p. 418) or evidence of tuberculous foci elsewhere, serve to clinch the diagnosis. The chief difficulty, after the stage of retraction has been reached, is to decide between tuberculous meningitis on the one hand and *meningococcal* (*posterior basal*) *meningitis* on the other. The duration of the disease is often of assistance in this respect—suppurative meningitis kills in two or three days, tuberculous meningitis in two or three weeks, whilst posterior basal meningitis ends in recovery in a variable percentage of cases, even after continuing for two or three months. The tendency to head retraction is greatest with the posterior basal, least with the suppurative forms. Optic neuritis barely has time to develop in suppurative meningitis, but it is present more often than not in both basal and posterior basal meningitis. The way in which the heels touch the occiput in some cases of the latter may by itself decide the diagnosis. Another point in favour of meningococcal meningitis, is the occurrence of periodic spike-like rises of the temperature chart—pyrexial 'crises' lasting twenty-four hours or less (*Fig.* 253), and superposed upon what is otherwise a chart of but moderate type. When doubt remains as to the fact of meningitis or as to its nature, microscopical and bacteriological examinations of the cerebrospinal fluid obtained by lumbar puncture will often serve to establish the diagnosis (p. 304). Bacteriologically, tubercle bacilli are the least easy to find. The Gram-negative meningococci (*Diplococci intracellulares meningitidis Weichselbaumii*) (*Plate XXVIII*, *Fig. X*, p. 614) are characterized by their occurrence within the leucocytes in pairs, like gonococci, but without the reniform shape of the latter. The organisms of suppurative meningitis may be discovered on direct staining, but more often cultural methods are required.

Where posterior basal meningitis ends and *epidemic cerebrospinal meningitis* begins, it is difficult to say; they are both meningococcal, and probably they are only different types of the same malady, connected together by sporadic cases in which posterior basal meningitis is associated with more or less severe spinal symptoms. The way in which the least touch or movement causes the patient to cry out with pain sometimes indicates how inflamed the coverings of the posterior nerve-roots are, besides which, the erythematous, vesicular, or purpuric skin eruptions that may accompany it often suggest the diagnosis. There is less difficulty during an epidemic; it is the sporadic case that may be missed. The clinching point in the diagnosis is bacteriological investigation after lumbar puncture, assisted perhaps by the beneficial effects of the specific antimeningococcal serum.

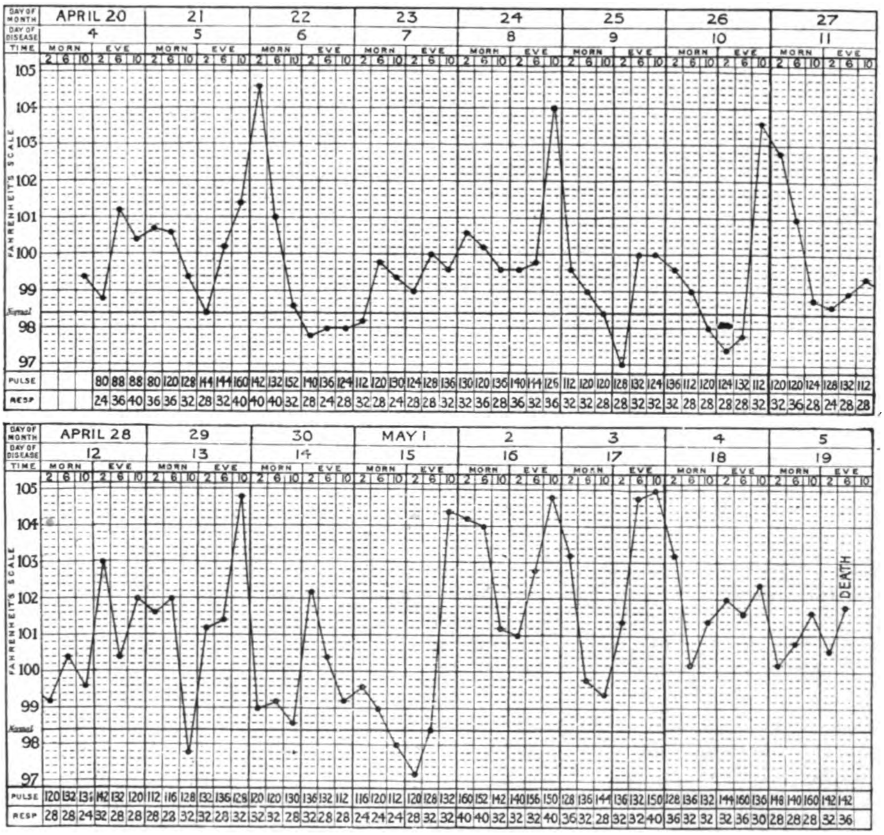


Fig. 253.—Four-hourly temperature chart in a case of meningococcal cerebrospinal meningitis. The pyrexial crises are even more marked in some cases. The diagnosis was verified at autopsy.

Superior longitudinal sinus thrombosis and *acute polio-encephalitis* are both nearly always diagnosed as acute meningitis in the first instance. It is when a case that has simulated acute and severe meningitis, with coma and apparently impending death, gets rapidly better after a few days and ends in speedy recovery, with or without some impairment of local or general brain functions, in a child or young person, that one changes one's diagnosis of meningitis to polio-encephalitis, though even then it remains one of opinion chiefly. Almost the same applies to superior longitudinal sinus thrombosis; though, if acute cerebral symptoms in a previously healthy child end in a gradual and but partial recovery, accompanied by permanent spastic paralysis of the legs, without much affection of the arms, it is very possible that the lesion has been thrombosis of the superior longitudinal sinus, with softening of the leg areas of cortex on either side of it.

Cerebellar or other *subtentorial tumours* or *abscesses* generally cause a much more gradual onset of symptoms than do any of the above. Head retraction is not present until the later stages. The diagnosis of tumour will rest on the slow increase in the signs of raised intracranial pressure, with nystagmus, optic neuritis going on to optic atrophy, and a tendency to fall always in one definite direction—forwards or backwards if the tumour is in the vermis, to the right or to the left according as it is in the right or left hemisphere. There is often marked ataxy, with exaggeration of the tendon reflexes, particularly on the same side as the tumour. If nystagmus is well marked this serves as a point of some value in distinguishing a cerebellar from a cerebral tumour. Abscess is distinguished from tumour chiefly by the existence of some obvious cause for intracranial abscess, especially otitis media on the one hand, bronchiectasis upon the other. Cerebellar abscess may give rise to no pyrexia and no leucocytosis ; but whether the temperature is raised or not, the pulse-rate is often absolutely slowed. *Herbert French.*

RIGIDITY OF THE ABDOMEN is a sign not to be regarded lightly, and one to find the true significance of which may call for the greatest care and skill. The patient should be examined lying on the back with the whole of the abdomen and lower thorax exposed. The observer, seated on a level with the patient, should watch the abdomen for a minute or so and see whether it moves or not with respiration, and whether one part moves more than another.

It should be remembered that some patients, whether from modesty or timidity, hold their abdomens intensely rigid in a wholly unnecessary way, a tendency which may create a false impression. This can be avoided by engaging them in conversation for a minute or two, by asking them to take a few deep breaths, or by making them draw their knees up and keep their mouths open, when the normal abdominal walls will generally relax.

There are varying degrees of rigidity. The whole abdomen may be rigid, the upper or lower part only, or one side, as in the presence of a localized appendicular abscess. Again, one part or one rectus muscle may be put, as it is termed, 'on guard,' whenever the patient thinks a tender spot is about to be touched. The rigidity over an inflamed gall-bladder, and that over a gastric or duodenal ulcer, are instances of this.

The most important cause of universal rigidity is *septic infection of the peritoneum*, which may follow external wounds, abdominal operations, childbirth, abortion, endometritis, parametritis, extension of inflammation from or perforation of the appendix, ulcer of the stomach, duodenum, or bowels, perforation of the gall-bladder, suppurating Fallopian tube, or abscess of the liver, spleen or kidney ; or which may be primary, as in some pneumococcal cases. It is a safe rule to believe there is peritonitis until the contrary is proved. As in the case of other disease, diagnosis must not be based on one clinical sign, and the patient must be examined for the other signs of peritonitis.

The history of the onset is important. In perforative cases, the beginning is marked by intense abdominal pain. This may be general and continuous, or by being referred to the stomach or appendix region give an indication of the primary seat of the mischief. The position taken up is on the back, sometimes with the knees drawn up to relieve abdominal tension, and the patient generally lies still, for any movement causes increase of pain. In colic, on the contrary, whether intestinal, biliary, or renal, the patient rolls about during the spasms. It is painful to use the diaphragm : therefore, respiration is superficial and costal in type. The abdomen gradually becomes distended, tense, and tympanitic ; the liver dullness which was previously present may disappear, and in some forms of peritonitis fluid may accumulate in the abdomen and be detected by the signs of shifting dullness in the flanks. The pulse is small and rapid, 100 to 150, and has the tendency to quicken. A friction rub may be heard over the liver or the spleen when the patient breathes. Borborygmi will generally be absent. Vomiting is an early, prominent, and almost constant feature. The contents of the stomach are ejected first, then bile-stained fluid, and later green or brownish fluid with a slight fæcal odour. The vomiting is often of a peculiar 'pumping' character. The bowels may be loose at first and then constipation follows, but is not complete as in intestinal obstruction. Micturition may be frequent, or there may be retention when the pelvic peritoneum is acutely inflamed. When the disease is well developed the appearance of the patient is very characteristic, exhibiting the 'Hippocratic facies.' The pulse is often of great assistance in arriving at a diagnosis of the need to operate urgently in these cases; it should be counted every ten minutes; if its rate falls or does not rise at successive counts, peritonitis is less probably present than when the pulse-rate is found to be rising each time.

In suppurative peritonitis leucocytosis, though it may not occur at all, is often marked (15,000 to 30,000 per c.mm.).

It does not necessarily follow, because the whole abdomen is rigid, that the peritonitis is general. For instance, in cases of perforative appendicitis it has been shown by operation that pus was only to be found around the cæcum, and yet there was general rigidity. Without operating, it is often impossible to tell. The best way of finding out whether there is pus in the abdomen, and the method to be employed at once if there is any doubt, is to open the abdomen and see.

Other Conditions associated with Abdominal Rigidity which may be mistaken for Peritonitis.

Pneumonia or *Diaphragmatic Pleurisy.*—In the early stages here, before the onset of dullness in the lungs and other physical signs in the chest, the most prominent features may be abdominal pain and rigidity. Laparotomy has often been performed on the mistaken diagnosis of peritonitis. If, however, the examination is thorough, signs pointing to implication of the lungs will usually be found. Rapidity of respiration, working of the nares, and blueness of the lips should receive particular attention. Examination of the blood may reveal a high leucocytosis (30,000 to 40,000); in suppurative peritonitis the numbers are rarely so high.

Colic.—The suddenness of the onset of pain, its intense character, and the abdominal rigidity, may render this condition extremely difficult to differentiate from peritonitis due to perforation of some viscus. Collapse may be marked, and the effect on the pulse is considerable; vomiting is common also. The temperature is raised slightly but rarely exceeds 100° F., and the pulse, though it may be rapid, does not tend to quicken progressively. The pain is spasmodic, not continuous as in peritonitis, and is generally relieved in a few hours. Biliary and renal colic are fairly characteristic, but that due to lead, the crises of tabes dorsalis, or gastro-intestinal disturbances may easily be mistaken. The gums are to be examined for a blue line, the knee-jerks and pupils tested, and a blood-count made. In uncomplicated colic there is no leucocytosis. In cases of extreme difficulty the abdomen may have to be opened. The persistence of borborygmi is in favour of colic rather than of general peritonitis.

Intestinal Obstruction.—The vomiting and constipation here present may lead one to think of peritonitis, and indeed the two conditions may be present at the same time, as in the case of an ulcerating carcinoma of the bowel. Usually the rigidity is not well marked, and the constipation, which is not absolute in peritonitis, is here complete.

Injuries of the Abdomen.—

1. Contusion of the abdominal wall, with laceration of muscle: Particularly in patients who have been run over across the abdomen, rigidity is a marked feature, and there must always be a doubt at first as to whether any of the viscera have been torn and are bleeding, or whether the escape of their contents is setting up peritonitis. In the case of mere contusion, if the patient is put to bed and kept warm, collapse will soon disappear, the abdomen will become less rigid, and the pulse-rate will fall.

2. Contusion of the abdominal wall with injury of viscera: The signs here will be more marked, and instead of tending to diminish rapidly will become worse. If there is internal bleeding the mucous surfaces will be pale, the skin cold and clammy, and the pulse small and frequent. If the contents of a viscus have escaped, the signs of peritonitis will develop rapidly. In all cases of doubt an exploratory laparotomy should not be delayed.

Ruptured Tubal Gestation.—This may simulate general peritonitis. The abdominal rigidity here is not well marked, and the signs of bleeding are. A moderate degree of leucocytosis is present (10,000 to 15,000), but the number of red cells is much diminished. If the patient is a woman of the child-bearing age, known to be a week or more overdue as to monthly period, and has begun to lose blood per vaginam synchronously with the onset of acute abdominal pain and pallor, the diagnosis will suggest itself at once.

Acute Hæmorrhagic Pancreatitis is usually diagnosed as intestinal obstruction or acute perforative peritonitis. The attack sets in with intense pain, usually in the upper and left part of the abdomen. Vomiting, constipation, and tympanitic distention are present. The condition is so rare, and the signs are so unreliable, that an exploratory laparotomy should be made, and the nature of the case becomes obvious directly the characteristic opaque yellow patches of fat-necrosis are seen in the omentum.

Rupture of an Abdominal Aneurysm, Dissecting Aortic Aneurysm, Embolism of the Superior Mesenteric Artery, may simulate peritonitis, and so also may *Acute Thrombosis of the Inferior Vena Cava* ; but all these conditions are rare, and they will be very difficult of diagnosis unless the existence of some cause for them, such as aortic aneurysm or fungating endocarditis, is already known.

Acute Suppurative Nephritis sometimes gives abdominal rigidity, and is associated with fever and vomiting. There is always marked tenderness in the loin on the affected side, and the urine will contain albumin, pus, blood, casts, and bacteria. The milder types of the infection (see BACTERIURIA, p. 69) may be mistaken for acute appendicitis, or for general peritonitis, unless the centrifugalized deposit from the urine is examined microscopically for pus.

George E. Gask.

RIGORS, or CHILLS, are common at the onset of the most various acute febrile disorders, and may occur at regular or irregular intervals in the course of many of the more severe of them. The chief sign of a rigor is shivering, the chief symptom a feeling of cold and general wretchedness. At its beginning, the patient looks chilly, pinched, and blue, and sits or lies huddled up, complaining of the cold ; his arteries are contracted, the pulse is rapid, small, and of raised tension ; the extremities are chilled superficially, but the internal temperature is above the normal. Very soon the sensation of cold induces involuntary shivering ; the patient shakes all over, sometimes so violently that the chair or bed is thrown into noisy vibration ; the teeth chatter, and even the muscles of the face twitch involuntarily. This shivering lasts for minutes, or even for an hour, dying away gradually as the patient feels himself to be warmed up. Thus the initial stage of the fever passes into the second stage or fastigium, in which the complaint is of sweating, thirst, and undue heat, and the body temperature rises still further. In children, general convulsions, with partial or complete coma, may occur at the onset of an acute infection, in conditions that would give rise to a rigor in adults. In adults, convulsions are not known to take the place of rigors. Cases may arise, however, particularly when only an imperfect history can be obtained, in which it may be hard to say whether a patient has had a rigor, or an epileptiform, hysterical, or epileptic fit. Should the patient have lost consciousness during the shivering, or have fallen down, bitten his tongue, or passed his water during the attack, or should he give a history of similar attacks on previous occasions, the diagnosis of epilepsy would be more than probable. Epileptiform fits that unskilled observers might confuse with rigors may occur in uræmic or eclamptic patients ; the history of the case, and the discovery of albumin in the patient's water, together with other evidences of acute or chronic renal disease, should make the diagnosis clear. Fits indistinguishable from rigors to the untrained eye may occur in hysteria ; in these, however, the shivering patient would be red in the face, or at least would not present the slightly livid and shrunken facial appearance characteristic of a rigor, the temperature would not be raised, and the signs or a history of other hysterical phenomena should be obtainable.

For their further consideration it is convenient to classify rigors according as they are single or multiple.

1. **Single Rigors.**—The occurrence of a *single rigor* at the outset of an acute infectious disorder is extremely common, and may be taken as evidence of the severity of the infection to some extent ; in lobar pneumonia this initial rigor is often particularly long and severe. No exhaustive list of the disorders that may be thus ushered in can be given ; but it may be stated generally that an initial rigor is common in :—

- Lobar pneumonia
- Small-pox
- Influenza
- Severe feverish colds
- Septicæmia
- Pyæmia
- Pneumonic tuberculosis
- Typhus
- Relapsing fever
- Erysipelas
- Cerebrospinal fever
- Acute poliomyelitis
- Malaria
- Yellow fever
- Weil's disease
- After catheterization.

It is less often seen in :—

- Scarlet fever
- Measles
- Diphtheria
- Tonsillitis
- Rheumatic fever
- Tetanus
- Miliary tuberculosis
- Sapræmia
- Glanders
- Sick headache
- Acute gastro-intestinal disorders
- Nephritis
- Cholelithiasis
- Renal calculus.

And is comparatively rare in : –

- Enteric fever
- German measles
- Mumps
- Gout
- Anthrax
- Hydrophobia
- Cholera
- Plague
- Dysentery
- Malta fever
- Beri-beri.

The diagnosis of all these different morbid conditions must naturally be made from the history of exposure to infection, and from the subsequent signs and symptoms. It is clear that the occurrence or non-occurrence of an initial rigor will rarely be of much practical assistance in determining the nature of the disorder from which any given patient is suffering.

A rigor after catheterization is not rare, whether the kidneys be sound or no, and in some cases is due to septic infection of the urethra or bladder. In others, however, it ensues when no infection has taken place, and is not followed by any evidences of urinary sepsis ; in these instances the rigor must be referred vaguely to nervous shock, and need not give rise to alarm.

2. **A Second Rigor** coming on in the course of any of these disorders, or a rigor occurring unexpectedly for the first time when the disease is well established or declining, is often evidence of the spread of the infection, or of the occurrence of some complication. For example, a second rigor occurring in the course of *lobar pneumonia* may coincide with the appearance of signs indicating the spread of the disease to the second and previously sound lung ; a second rigor happening after the crisis may indicate an empyema. In *enteric fever* a rigor is rare, though it may be due to such complications as perforation of the intestine, acute peritonitis, pleurisy, pneumonia, middle-ear disease, periostitis, and so forth ; but there is an abnormal type of enteric fever in which rigors occur for no apparent reason, followed by heavy sweats ; and rigors may be observed in cases with constipation, or during defervescence, or in enteric patients who have been treated with antipyretic drugs.

3. **Recurring Rigors.**—The occurrence of a *series of rigors* often gives information of more definite value, for they are seen in but a limited number of local or general infections, most of which have some characteristic or localizing signs. In themselves these rigors are no more than evidence of the severity of the infection, and of the extent to which bacterial toxins have been absorbed into the blood. The following are the chief disorders characterized by a series of rigors :

- Malaria—tertian, quartan, æstivo-autumnal or malignant
- Relapsing fever
- Acute leukæmia
- Acute blood-infections, including
  - Portal pyæmia
  - Pyæmia
  - Septico-pyæmia
  - Septicæmia

Special forms of these may be known as puerperal fever, malignant endocarditis, acute infective osteomyelitis, suppurative pylephlebitis, etc.

- Acute inflammations, e.g. :—
  - Pyelitis
  - Pyelonephritis
  - Cystitis
  - Cholecystitis
  - Empyema
  - Infective sinus thrombosis
- Abscess formation :—
  - Hepatic (tropical)
  - Appendicular
  - Subphrenic
  - Perinephric
  - Prostatic
  - Cerebral
- Pulmonary tuberculosis
- Bronchiectasis
- Enteric fever
- Erysipelas
- Rat-bite fever
- Influenza.

A very thorough physical examination of any patient presenting multiple rigors should be made ; the condition is always serious, and may be due to septic absorption from some deep-seated abscess that produces only the scantiest of physical signs. When no abnormal physical signs can be found bacterial cultures should be made from the circulating blood, care being taken to draw off a sufficient quantity of blood—5 to 10 c.c.—and to repeat the cultivation several times before it is decided that the blood-stream is sterile.

In *malaria* the rigors tend to recur at regular intervals of forty-eight (*Fig.* 6, p. 28) or seventy-two hours (*Fig.* 7, p. 29) in the benign tertian and quartan infections, at shorter intervals if the infection is mixed. In the æstivo-autumnal form the rigors and also the course of the fever are much less regular (*Fig.* 10, p. 31). The parasite (*Plate VI*, p. 32) may be found in the circulating blood, and the patient, if not in extremis, is cured by quinine: there is no leucocytosis, but a relative increase in large lymphocytes occurs.

In *relapsing fever* the onset is acute, with a rigor or a series of rigors. A fortnight later, when the patient has been convalescing for a week or ten days, relapse and a second rigor or series of rigors occurs (*Fig.* 5, p. 27). A second relapse may be noted at the end of the third week, and in a very few cases a third relapse. Relapsing fever has practically died out of the United Kingdom, but it is met with in Egypt, India, and other countries. It occurs in epidemics, and Obermeier's spirochæte (*Plate XXVIII*, *Fig. I*, p. 614) can be found in the patient's blood while he is feverish.

Multiple rigors occur exceptionally in the course of *acute blood-diseases*, such as acute leukæmia, pernicious anæmia, or Hodgkin's disease. Severe and progressive anæmia, wasting, fever, heavy sweats, and hæmorrhage from the mucous membranes, are likely to occur in these cases, with characteristic changes in the microscopical appearances presented by the blood (p. 24).

Multiple rigors are commonest in the various forms of *acute blood-infections*; special forms of these have received particular names. Thus *puerperal fever* occurs after delivery, and is due to bacterial infection of the uterus and its spread thence to the blood; the patient will probably have a sanious or offensive vaginal discharge as well as the evidences of septicæmia or pyæmia. In *malignant endocarditis*, attention is directed mainly to the condition of the heart, the presence of valvular murmurs and the signs given on p. 34. In *acute infective osteomyelitis* the first complaint arises from the acute inflammation occurring in the marrow of one of the bones. *Portal pyœmia* or suppurative pylephlebitis is seen in patients with various acute inflammatory intra-abdominal lesions, and is due to the spread of bacterial infection to the portal vein. The commonest precursor is mild appendicitis. The blood in the portal vein clots, the clot is infected with microbes, softens, and breaks up, to be dispersed throughout the liver in the form of infective emboli. Multiple hepatic abscesses result, with pain, swelling, and tenderness in the hepatic region; jaundice is present in less than half the cases, with more or less coloured stools, vomiting and diarrhœa are frequent, and there is hectic fever. *Pyœmia* is characterized by the formation of metastatic abscesses in any of the tissues or organs, oftenest in the lungs, in consequence of the lodgement there of multiple infected emboli. Before the days of antiseptic or aseptic surgery, pyæmia was the common outcome of serious surgical operations or severe wounds. Nowadays it is comparatively infrequent, and when it does occur is secondary to a severe infected wound, to ulcerations of the mucous surfaces, or to deeply-seated abscesses that are not amenable to surgical treatment. Occasionally it seems to be idiopathic, or due to some infective lesion that escapes discovery. Pyæmia oftenest begins suddenly; the main symptoms are hectic fever, rigors, leucocytosis, diarrhœa and vomiting, heavy sweats, prostration, and the formation of secondary abscesses due to the arrest of septic emboli. When the lungs become the seat of multiple abscesses, the breathing becomes rapid, and signs of bronchitis, pleurisy, or pulmonary consolidation appear. Abscesses in the more superficial tissues or joints make their presence known by the local evidences of pain, swelling, redness, and heat; in the deeper parts or organs, by pain and disturbance of function. The development of secondary subcutaneous abscesses is common in the less acute cases; abscess-formation in the heart, and suppurative pericarditis, are prone to occur when the primary lesion is a periostitis or an acute necrosis of bone. Pyæmia may be distinguished from enteric fever only with great difficulty if evidences of abscess-formation or some source of primary infection are not forthcoming, especially as the typhoid state is common in the later stages of both diseases; the occurrence of multiple rigors is rare in enteric fever, common in pyæmia; Widal's reaction should be tested for. From malaria, pyæmia is distinguished by not reacting to quinine; malarial parasites will not be found in the circulating blood. Pain and inflammation in the joints after childbirth or a miscarriage may be diagnosed as rheumatism when the condition is really one of pyæmia or puerperal fever.

The precise diagnosis between pyæmia and septicæmia is often impossible, and is, indeed, of academic rather than clinical interest. The necessity for it is in part avoided by the use of the term *septicopyæmia*, the evidences of which are much the same as those of pyæmia : all three conditions may arise from identical causes, and bacteria (streptococci, staphylococci, gonococci, pneumococci, *B. coli communis*, *B. typhosus*, *B. influenzæ*, *B. pyocyaneus*, etc.), may be cultivated from the circulating blood in any of them. Multiple rigors are far commoner in pyæmia—where several may occur daily—than they are in *septicæmia*. The latter condition is due to the growth of microbes in the blood without the formation of metastatic abscesses ; it originates in lesions very similar to those that underlie pyæmia, or results from infected but apparently trifling cuts or injuries, or even from neglected chronic suppuration about the teeth. Its main symptoms are pyrexia, debility, anæmia ; in severe cases rigors occur, and the patient may fall into the typhoid state. The bacteria causing it can be cultivated from the circulating blood ; septic rashes are often seen in both pyæmia and septicæmia, but they are not seen in enteric fever. It should be noted that, at the best, a deal of looseness attaches to the meaning of the term septicæmia ; for in lobar pneumonia, enteric fever, Malta fever, and many other acute febrile disorders, the specific microbes can habitually be cultivated from the circulating blood. Technically speaking, therefore, these are all instances of septicæmia. Two new terms have recently come into vogue in this connection, namely, *bacillæmia* and *bacteriæmia*.

Multiple rigors may result from *acute localized inflammatory infections* if the inflammation is sufficiently extensive and the infecting micro-organism virulent. It is often impossible to say how far such rigors are evidence of the absorption of toxins, and how far they indicate that living bacteria have gained access to the blood-stream. Situated in the genito-urinary tract, these inflammations are often associated with a history of *gonorrhœa*, *renal calculus*, or *gout*, and produce characteristic pathological changes (hæmaturia, pyuria, albuminuria) in the urine, or difficulties in micturition. If the gall-bladder or bile-ducts are the seat of the inflammation, jaundice, and pain in the hepatic region will probably be observed with the fever and rigors, and a history of gall-stone colic may be given, *suppurative cholecystitis* or *suppurative cholangitis* having supervened ; the gall-bladder will be tender and probably enlarged from the former, the whole liver swollen and possibly tender from the latter ; Charcot's hepatic intermittent fever is due to *chronic cholangitis*, with intermittent biliary obstruction due to a ball-valve stone often lying in the ampulla of Vater. The occurrence of rigors in a child convalescing from pneumonia, measles, scarlet fever, or pleurisy, may lead to the discovery of an unsuspected *empyema*. *Infective sinus thrombosis* occurs mainly in patients with otorrhœa, and indicates that the bacterial infection has spread from the ear to one of the cranial venous sinuses. Its symptoms are general—those of septicæmia or pyæmia, often with an initial rigor and vomiting followed by high fever, more rigors (*Fig.* 244, p. 567), and sweating ; and local—very severe pain about the ear, excruciating headache, and venous congestion of the optic disc, with others that vary with the site of the thrombosis. If the sigmoid sinus is thrombosed, œdema and tenderness over the mastoid appear, and should the clotting spread downwards a thrombus may be felt in the internal jugular vein. Thrombosis of the cavernous sinus is accompanied by squint, exophthalmos, and œdema of the orbits and eyelids. Thrombosis of the superior longitudinal sinus may set up œdema of the scalp near the sagittal suture. The diagnosis must be made from cerebral or cerebellar abscess, in which repeated vomiting is likely to occur, and the localizing signs and symptoms will suggest brain-disease ; and from meningitis, in which rigors are rare. In other patients, some acute inflammatory disorder may result in definite *abscess formation*, when rigors may develop from toxic or septic absorption ; here again, the virulence of the particular microbe causing the inflammation will be the chief factor in determining whether rigors occur or not. In many cases the rigors will really be due to a secondary and probably terminal septicæmia or pyæmia.

Tropical abscess of the liver, usually single, occurs in patients who have been abroad and have had dysentery, whether amœbic or bacterial. The early symptoms of liver abscess are often obscure, malaise, fever, sweating, rigors, and gastro-intestinal disturbances occurring, or a pleural effusion secondary to spread of infection through the diaphragm, while nothing particularly suggests implication of the liver. As a rule, complaint of dull pain in the right hypochondrium, axilla, or shoulder will be made. The diagnosis of this and other forms of suppuration in the liver is discussed on p. 369.

Multiple rigors may occur from septic absorption in various diseases of the lungs, the most important of which are *bronchiectasis*, and advanced *pulmonary tuberculosis* with secondary pyogenic infection of the bronchi or tuberculous cavities. In either case the sputum will be abundant, and will probably contain fragments of elastic tissue ; it is sure to be offensive in bronchiectasis.

High or irregular fever with recurring rigors has been recorded in a few unusual cases of *enteric fever* (*Fig.* 241, p. 565) and of *influenza* free from any complication, and in *erysipelas* (*Fig.* 245, p. 568).

Only about a dozen cases of *rat-bite fever* have been recorded in Great Britain so far, but it is probably commoner than it would appear to be, and habitually unrecognized. It occurs in persons who have been bitten by rats, or by ferrets, cats, or weasels that have killed rats recently : the rat-bite heals slowly, and after an incubation period of from two to four weeks the patient begins to suffer from a series of acute febrile attacks at fairly regular intervals of a few days. These attacks recur for from two to ten months in different instances (*Fig.* 254). The onset of each is abrupt, with headache, fever up to 102°–106° F., malaise, pains all over, often a rigor, severe pain and swelling in some of the muscles, recurrence of inflammatory phenomena, rarely suppurative, about the original wound, and urticarial, measly, patchy erythematous rashes on the face, limbs, and trunk. Each attack lasts for a day or two, the patient being fairly well in the intervals ; during the attack a varying leucocytosis is common. Rat-bite fever is not fatal ; the infecting agent, though often looked for, remains unknown.

A. J. Jex-Blake.

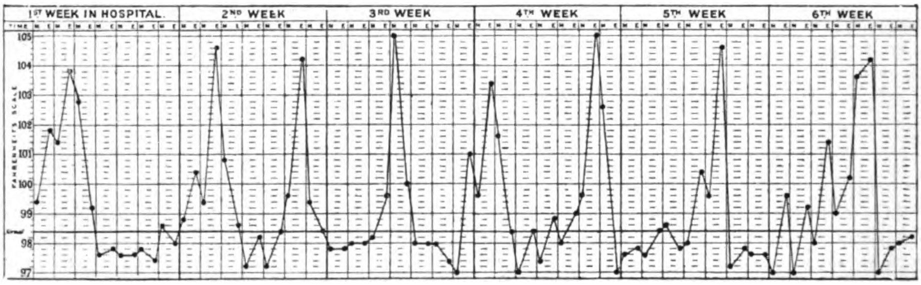


Fig. 254.—Temperature chart in a severe case of rat-bite fever ; only part of the chart is shown ; the recurrent attacks extended over a period of four months.

RINGWORM.—(See FUNGOUS AFFECTIONS OF THE SKIN, p. 246.)

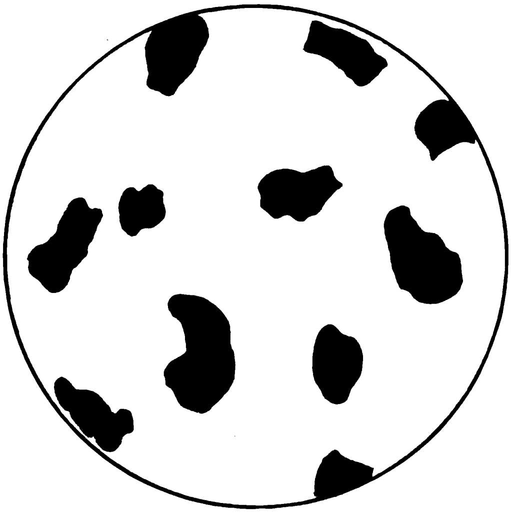
RISUS SARDONICUS is the fixed unmirthful grin that results from spasm of the muscles of both sides of the face. The angles of the mouth are drawn outwards and the eyelids raised by tonic contraction of the same muscles as produce the facial expression of smiling, but the spasm is maintained in a way that at once excludes natural smiling. The chief causes of the condition are tetanus, strychnine poisoning, malingering, hysteria, catalepsy.

Catalepsy.—The differential diagnosis is not, as a rule, difficult. A cataleptic case is chronic ; the facies is by no means always that of smiling, but if it should be, then the smile is a fixed one ; the chief characteristic of the condition is the maintenance for hours at a stretch of some attitude that would rapidly fatigue an ordinary person ; the history and the associated mental symptoms of melancholia or dementia point to the diagnosis, and tetanus and strychnine poisoning would be excluded by the absence of tetanic spasms.

Hysteria sometimes takes a form that may for a while raise doubts as to strychnine having been taken, but, as a rule, the multiformity of the contortions points to the correct diagnosis. The features may be kept fixed for a time, but sooner or later they become twisted into all sorts of shapes, and the tonic and clonic spasms of the body and limbs are not in any way regular, as they are apt to be in strychnine poisoning and tetanus. The patient is likely to be a woman, and there may be a history of previous hysteria (p. 465).

PLATE XXIII

INTESTINAL SAND



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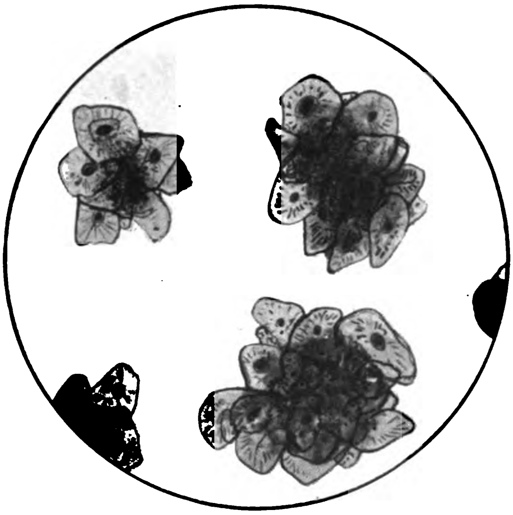

2

1, Microscopical appearance of true intestinal sand.

2, Microscopical appearance of false intestinal sand, following the ingestion of pears

By permission from 'The Fæces of Children and Adults.' P. J. Cammidge.

During a quiescent interval it may be found possible to stroke or touch the patient without bringing on a convulsion, whereas in strychnine poisoning and in tetanus the slightest touch is apt to evoke a violent and generalized spasm, even opisthotonos.

Malingering may take the form of imitated convulsions, during which the features may be kept fixed in one position or another, sometimes in that of smiling. The fixed voluntary contractions cannot be maintained long, however, on account of fatigue, so that although there may be some doubt at first, this generally disappears soon. The patient is usually a man who has something to gain by malingering: a night's lodging in a hospital, for instance.

Strychnine Poisoning and **Tetanus** are the two chief causes of typical risus sardonicus. The main point to rely on in distinguishing the two is the history, if it is obtainable—the injection of an overdose of strychnine hypodermically, or the taking of a rat-paste, on the one hand, or the occurrence of some small but penetrating wound by a rusty nail or earth-soiled knife or stick during the fortnight preceding the symptoms, on the other. The absence of any known wound, however, does not exclude tetanus. If lock-jaw and stiffness of the neck are prominent features, tetanus is more probable than strychnine poisoning, and vice versa. In strychnine cases, the patient will either die quickly, or the symptoms will subside rapidly, whereas in tetanus they may persist unabated for several days. In a few instances the diagnosis may only be settled by the discovery of strychnine in the gastric contents, or of tetanus bacilli (*Plate XXVIII*, *Fig. T*, p. 614) in anaërobic cultivations from the infected wound.

It only remains to add that a few cases of *facial sclerodermia* may simulate risus sardonicus, though more often there is complete smoothness of the features and lack of expression. There are no spasmodic contractions, the condition comes on gradually, is permanent, and the diagnosis becomes obvious at once when the hard smooth skin is palpated, for one cannot pick it up between one's fingers. *Herbert French.*

RUMINATION.—(See Merycism, p. 388.)

RUPIA.—(See Scabs, below.)

SALIVARY GLANDS.—(See Swelling of the Salivary Glands, p. 694.)

SALIVATION, UNDUE.—(See Ptyalism, p. 542.)

SAND, INTESTINAL.—This is seen in the motions, especially when they are fluid and the patient has membranous colitis. It is like the finest sea-sand; its colour varies owing to varying degrees of imbibition of fæcal pigment. Usually it is red, looking something like fine uric acid, and it varies from this to a pale dirty yellow. It is seen best showing up against the white of the bed-pan in which it lies. Analysis shows that it consists of from 30 to 70 per cent of organic matter, doubtless all derived from the fæces. The inorganic matter is invariably nearly all calcium phosphate, with traces of calcium oxalate, magnesium, iron, and perhaps silica. The amount of sand passed in a day may be four teaspoonfuls, but usually it is much less. Many patients pass it for years, but not always constantly even then; it may be passed daily for weeks, and then for weeks none is passed. It is far commoner in those who have membranous colitis than in other patients, but it has been seen with malignant disease of the large intestine; it always indicates some organic disease of the colon. It must be distinguished from false intestinal sand, which looks very like it and may be found in the motions of those who have eaten largely of pears. This is entirely vegetable, and can be distinguished easily from true intestinal sand by microscopical examination (*Plate XXIII*). *W. Hale White.*

SCABS.—The scab, or crust, one of the secondary cutaneous lesions, is a more or less irregular, dried-up mass of exudation on the surface of the skin. It may be produced by the desiccation of serum, pus, or blood, or of a mixture of these fluids, and commingling with these substances there may be epithelial débris, or fat, or fungous elements. Scabs form on matured vesicles, bullæ, and pustules, on ulcerations, erosions, and on every kind of excoriation, pathological or traumatic. If the exudation is thin, as sometimes in eczema, they are soft and friable; if it is thick, they may be tougher and more adherent, and successive layers may be formed, as in the rupial crusts of syphilis. Scabs composed largely of fungous elements are more or less friable, and these, like those resulting from the seborrhœic process, may partake of the character of scales as well as of scabs. Such formations, however, as for example the 'crusts' of favus, are in the nature of scales rather than of scabs, and are noticed elsewhere. (See SCALY ERUPTIONS, p. 601; and FUNGOUS AFFECTIONS OF THE SKIN, p. 246.)

Scabs vary greatly, not only in consistence, thickness, and adhesiveness, but also in colour and in form; and by attention to these differences the diagnosis of the affections in which they occur may be assisted. Some guidance may also be obtained from the condition of the surface from which the scab has been removed; it may be dry when the scab has been long adherent, as in some cases of impetigo, excoriated as in eczema, or ulcerated as in rupia. But the clinician will be guided much more by the primary than by any secondary lesion; and for the decisive diagnostic features of the diseases about to be mentioned, the reader is referred to the articles MACULES (p. 382); VESICLES (p. 753); BULLÆ (p. 96); PAPULES (p. 487); NODULES (p. 402); and PUSTULES (p. 557.)

In *irritative herpes* the vesicles on the skin shrivel up and form yellowish-brown crusts, which after a few days become detached, as a rule leaving no scar, but only a brownish stain, which slowly fades away. In *herpes zoster* most of the vesicles which do not abort reach the same termination; but others, instead of drying up, may burst and discharge a fluid, which then forms yellowish or brownish crusts. In zoster a scar is produced sometimes. In *erythema multiforme* there is often considerable scabbing, as is mentioned under VESICLES (p. 758). In *eczema* the lesions may dry up either into scales or into crusts. Crustation is usually the third stage in the evolution of the disease, the discharge from the vesicles drying into greyish-yellow scabs of varying thickness, which become detached and are succeeded by others until the 'weeping' ceases. When the lips are attacked, they may become so stiffened under layers of crusts superimposed one upon another that the patient can hardly move his lips without fissuring the skin. In the male, the 'bathing-drawers' area may be so covered with crusts that the patient cannot walk or sit down without breaking them. In the same region in the female the inflammation and crustation may be even more severe, and the scabs may be marked by much foulness. One of the characteristic features of what is called *papular eczema* is the appearance of a tiny dome of blood-crust on the papules, due to scratching. In *seborrhœic eczema* there may either be scaling, or the squames may be massed into fatty crusts (see SCALY ERUPTIONS, p. 601). The scabs in *eczema rubrum* are extremely thin, like goldbeater's skin; when they are torn off, a red, wet, raw, tender surface is laid bare. The crusts of *scabies* may be distinguished from those of eczema by their being isolated and distributed irregularly, instead of being grouped, and by the multiformity of the lesions with which they are mixed—vesicles, bullæ, pustules, hæmorrhagic scabs, etc. In most itching diseases there will be found blood-scabs, resulting from the scratching to which the patient is provoked.

In *cheiropompholyx* the bullæ into which the little sago-grain vesicles run dry up into crusts, the removal of which reveals a surface that is red and exquisitely tender. The appearance and sensitiveness of the underlying skin, together with the limitation to the hands and feet, and often to the hands alone, will help the diagnosis. The crusts of *sycosis vulgaris* also have a limited distribution; they may be confined to the upper lip, and in any case they do not extend beyond the hairy parts of the face. They are brown or yellow in colour, thin, and distinctly adherent.

In *impetigo contagiosa* the scabs which are formed from the dried-up fluid discharged by the pustules on rupture are yellowish; in uncleanly persons they are brown, or even black. A characteristic feature is that they have around them no hyperæmic halo, but look as if they might have been stuck on artificially. In the severe form of the disease styled *ecthyma*, however, the flat irregular scab formed from the ruptured vesicles is surrounded by a more or less pronounced areola. At first loosely attached, the scabs in impetigo contagiosa afterwards become so firmly adherent that their removal requires some force and gives rise to a little bleeding. In this affection there is what may be called a secondary scab, formed by the drying-up of the thick, purulent discharge—honey-like in consistence and appearance—from the surface left raw by the removal of the earlier crusts. The reddish stain that appears when the lesion heals is not permanent. In *pemphigus vulgaris* the crusts into which the bullæ shrink are brownish-yellow ; and when they fall off spontaneously the surface beneath is not raw, as in impetigo, but is found to be covered with newly-formed epidermis, at first purple, afterwards turning brown, and gradually becoming normal in colour. When the area covered by the scabs is extensive there is an unpleasant sense of tension, and if they are removed prematurely, excoriation may be caused. In the more serious affection known as *pemphigus foliaceus*, the crusts are yellowish, and as the disease proceeds, large scales are formed. In *pemphigus vegetans* the foul-smelling secretion from the patches of affected skin forms a thin crust, which can easily be stripped off, when a papillary excrescence, partly covered with a thin stratum of epidermis, is revealed. The process usually ends in gangrene and death. The excrescences are distinguishable from the condylomata of syphilis by always being surrounded by a zone of bullæ, while condylomata have an infiltrated border.

In *nerve leprosy* the bullæ, which have the same characters as those of pemphigus vulgaris, form, on rupture, a large crust, the removal of which exposes a grey surface consisting of altered rete, the epidermis being cast off by suppuration. In this way a succession of yellow scabs or crusts may be formed and fall off, leaving at last a granulating surface, which ultimately is converted into a white scar. If the bullæ abort, they are followed by a parchment-like scale instead of a crust, and this in turn gives place to a hyperæsthetic ulcer. The scabs of nerve leprosy have some resemblance to the *rupial crusts* of secondary syphilis, but there is little danger of confusion between the two. The rupial crusts, greenish or blackish, consist of several successive layers, each smaller than the one immediately below it, so that a pyramidal structure is formed, somewhat resembling the shell of a limpet. This very characteristic crust, which can scarcely be mistaken for that of any other condition, and distinguishable from the psoriasis rupioides of M'Call Anderson by the base being ulcerated, is formed from pustules usually, but may follow also on the drying up of bullæ. The scabs in *secondary yaws* form upon the yellow heads of large papules, and beneath them are found reddish raspberry-like granulations which secrete a little pus, and after a time become pale or even white. Healing usually takes place beneath the scabs, which fall off about the end of the second month from the onset of the secondary rash. The raspberry-like granulations, the characteristic lesion of yaws, will obviate confusion between these crusts and those of any other affection. The crusts of *lupus vulgaris* are greenish-black, like rupial crusts, but they do not consist of layers superimposed upon each other, and dotted around the ragged edge will be seen the 'apple-jelly' nodules which are the 'note' of lupus.

In *small-pox* the formation of scabs on the pustules begins in the centre and causes a secondary 'umbilication'; it is generally attended by intense itching. In from three to four weeks from their appearance the crusts fall off, leaving a reddened surface, made uneven by scars or 'pits.' The true nature of the disease will have been discovered, even in doubtful cases, before the crust stage is reached. (For the differential diagnosis, see under Pustules, p. 557). In the diagnosis of *ulcers*, as in that of small-pox, the crust is of little importance. These are dealt with under Ulceration of the Face (p. 735) and Ulceration of the Foot (p. 735).

Malcolm Morris.

SCALY ERUPTIONS.—The squame, or scale, one of the secondary cutaneous lesions, is a dry, and as a rule laminated, exfoliation of the epidermis. Disregarding the slight, imperceptible desquamation which is a purely physiological process, scales may be said to result either from inflammation, as in psoriasis and pityriasis rubra pilaris ; from an abnormal dryness of the skin, as in dry seborrhœa and keratosis pilaris ; or from an earlier acute hyperæmia, as in scarlatina and other erythematous eruptions. The process may consist in an over-multiplication of the epidermic cells or in interference with the normal horny transformation. In colour, scales are ordinarily white or grey, either dull and lustreless, as in seborrhœa, or silvery, as in psoriasis ; but they are sometimes a dirty yellow, as in some dry syphilides, or even reddish-brown, as in oily seborrhœa. They may be large and thin, as in pityriasis rubra, or small and branny, as in tinea versicolor ; even in the same affection (e.g., pityriasis rubra) they may vary greatly in size in different regions. They may consist of a single layer, as in squamous eczema, or of several adherent strata, as in psoriasis. In quantity they may be inconsiderable, as in tinea versicolor, or most profuse, as in psoriasis and pityriasis rubra. Usually they are dry and friable, but if mixed with an oily secretion, as in seborrhœa, or with a serous or seropurulent discharge, as in eczema, they may partake of the nature of both scales and crusts.

A brief description of the scales met with in various affections may be given here; but except in a few diseases of which they form a highly characteristic manifestation, such as seborrhœa, psoriasis, and pityriasis rubra, they have little diagnostic value, and, as I have said elsewhere of scabs, diagnosis must usually rest upon primary rather than upon secondary lesions.

Sometimes there is scale-formation in eruptions due to the internal administration of such *drugs* as quinine or belladonna, or to the external application of carbolic acid, iodine, etc.; but it has no significance in diagnosis. In *keratosis pilaris* (xeroderma) and other forms of *ichthyosis*, there is always more or less desquamation of the dry and roughened and sometimes warty skin; but here also it is without diagnostic import. Of the scales of scarlet fever, measles, German measles, and other *infectious fevers*, again, nothing need be said, for the diagnosis ought to have been settled before they appear. Nor need I speak of the scaliness of tinea tonsurans, tinea versicolor, and other *fungous affections*, for the differential diagnosis of these diseases has been given under FUNGOUS DISEASES (p. 246). The scales of papular syphilides, again, have been described under PAPULES (p. 487), and the diagnosis of the lesions, both primary and secondary, from those of psoriasis will be found in that article. In the unusual instances of *urticaria* in which desquamation is present, it is so slight as to be negligible. In most forms of *erythema*, scales occur; but here again they have little significance for the diagnostician, and it will be sufficient to say that in erythema simplex the desquamation is slight, and that in erythema scarlatiniforme it is more considerable. In *lupus erythematosus* the central scar-like depression of the primary eruptive lesions may be covered either with thin, papery, greyish scales, or with a firmly adherent scab. In parts furnished with sebaceous glands, the skin will usually be covered with small adherent scales of sebum, which at the margin of the patch plug the dilated orifices of the glands, so that numerous comedones are formed. From other forms of erythema, as also from ringworm, lupus erythematosus may be distinguished by the slowness and persistence of the process. The lesion itself, atrophic in the centre, with a well-defined red border, and studded with plugs, can scarcely be mistaken when it appears on its site of election, the face. When, however, it occurs on the hands, it may mimic chilblains so closely that only the lapse of time can make the diagnosis certain, lupus erythematosus being much more obdurate to treatment, and not disappearing in the summer. For the diagnosis between lupus erythematosus and psoriasis, see below; for that between lupus erythematosus and lupus vulgaris, see under NODULES (p. 402).

We now come to affections in which scales play a more important part. In *seborrhœa sicca* there is an excess of the solid fatty constituents of the sebum, and the excreted material takes the form of scaly but usually somewhat greasy masses. In *seborrhœa oleosa* there is an abnormal predominance of the oily part of the sebaceous secretion, which dries into yellowish or reddish-brown cakes of greasy scales, often with a hyperæmic base and a fringe of papules about the edge. In the face, oily seborrhœa is more often met with than the dry form; but seborrhœa generally, though it may occur on the trunk and limbs, almost invariably begins on the scalp. When not limited to the scalp, as usually it is, it spreads downwards to the face, round the neck, the chest, the centre of the back, and the limbs. In the light of this preference for the scalp, and the downward extension when the affection is not confined to that part, a typical case of seborrhœa is unmistakable. In cases of seborrhœa which resemble psoriasis, guidance may be found in the scales, which in the latter affection are silvery, and harder. The respective starting-points of the eruptions, however, furnish a safer indication, psoriasis almost always appearing first on the elbows and knees and spreading upwards.

In typical *eczema*, scaling forms the final stage of the pathological process. After the initial erythema comes the exudation, then the crustation (see SCABS, p. 599), next the dry stage, and lastly the desquamation, the epidermis being shed in scales that become progressively thinner and smaller until only a brownish stain is left to mark the site. All the stages are often present at once in a given case. Scaling is frequently a noticeable feature when there is a predominance of erythematous lesions, as it is also in *eczema folliculorum*. But it is in *seborrhœic eczema* that this phenomenon is most prominent, the latent catarrh with which the affection begins being followed by the agglutination of epidermic scales which are thrown off in the form of large lamellæ. In some cases the scales may increase in quantity, in others, as is mentioned under SCABS (p. 599), they may become massed into fatty crusts among the hairs. The differential diagnosis of eczema has been set out in the articles on the primary lesions. The secondary lesions in this affection may indeed be rather a hindrance than a help in determining its true nature, and in doubtful cases the first thing to do is to remove both scales and crusts in order that the underlying lesion may be examined carefully. As between eczema and psoriasis, however, just as between eczema and seborrhœa, the scales afford guidance in the diagnosis ; while in psoriasis the lower layers of scales are whitish or silvery and hard, in eczema they are yellowish, dull, and friable.

In *psoriasis* the scale has distinct diagnostic value. It enters, indeed, into the definition of the disease as an affection of the skin, characterized by flat, dry patches of varying extent, covered with whitish, silver-grey, or asbestos-like scales. The scaliness may vary from a thin film to a dense, heaped-up mass. If the scales are removed, a smooth, dry, shining hyperæmic surface is seen, studded with spots that show various gradations of colour, from a deep to a bright red, the bright-red points being the tops of inflamed papillæ. The eruption appears as papules of pin-head size, at first red, but becoming white as the scales form. Spreading centrifugally, the papules form patches, generally roundish or oval when small, and becoming more irregular as they grow larger. They may remain stationary for a long time, and slowly disappear, or, continuing to spread, may become confluent. While the disease is active the individual patch is encircled by a narrow zone of redness, but when it is not spreading this fades away. Sooner or later involution takes place, and the redness which the lesions leave behind them soon disappears, though in protracted cases a brown stain may persist, and in rare instances there may be superficial atrophy. The malady may come and go, recrudescing after nearly disappearing, for the greater part of a patient's life. It is often most abundant when the patient is in the best health, and may almost or quite disappear during serious illness. In distribution, psoriasis is almost invariably symmetrical. Like small-pox, it shows a marked predilection for surfaces that are exposed to friction. Almost always it starts on the tips of the elbows and the fronts of the knees. After the extensor aspects of the limbs, its favourite site is the hairy scalp, and then the trunk, especially over the lumbar region. In typical cases the clinical picture—the patches with sharply defined border, covered with hard, shiny scales ; the hyperæmic surface beneath, dotted with red points ; the distribution as just described ; the unimpaired health, the natural complexion, the proneness to attack blue-eyed fair-haired persons, and the absence of exudation—scarcely admits of misinterpretation. In all these particulars, except the unimpaired health, psoriasis differs from eczema, as well as in the less intense and less constant itching by which it is attended. On the scalp, while psoriasis usually occurs in patches and ends abruptly at or only just beyond the margin of the hair, seborrhœic eczema almost invariably extends over the whole surface, and often involves the face and the neck. Almost always, too, psoriasis spreads upwards from its sites of election, seborrhœic eczema downwards from the head. In very chronic forms of eczema, in which there may be no history of 'weeping,' the diagnosis from psoriasis may be difficult. In all doubtful instances, gentle scratching on the affected surface will bring out the silvery scales, if the case is one of psoriasis.

The papular stage of *lichen planus* may be mistaken for psoriasis. In lichen planus, however, the eruption shows a preference for the flexor aspects of the wrists and knees ; it consists of shining-smooth papules, without scales ; the ground-tint is bluish-red or violet ; and the patches are formed by the aggregation of a number of papules instead of by centrifugal extension. In doubtful cases the whole body must be searched for the typical lesions of either disease.

Lupus erythematosus differs from psoriasis in that, in the former condition, the scales are not abundant, the edge of the patch is more elevated, the cheeks are usually attacked, as they are not in psoriasis, and there are the distinctive plugs in the orifices of the sebaceous ducts, as described earlier in this article. Scarring, too, may be found in the patch, and there may be atrophy of the ears.

As between psoriasis and *papular syphilides*, the diagnosis is given under PAPULES (p. 487). The heaped-up crusts of the condition which has been styled psoriasis rupioides can be distinguished from the rupial lesions of syphilis by the base being ulcerated in the latter disease ; but syphilis mimics everything, and cases sometimes occur in which it can be distinguished even from so distinctive an affection as psoriasis only by attention to the history, and by the discovery of other lesions, the presence of cachexia, the influence of salvarsan, iodides, and mercury, or by the Wassermann serum test.

In *pityriasis rubra* the whole cutaneous surface is always inflamed and reddened, without infiltration or thickening, but accompanied by profuse desquamation (*Plate XXIV*), branny on the head, larger flakes from the trunk, huge scales from the hands and feet. Pityriasis rubra may occur as an independent disease—an extremely rare event—or may follow in the wake of erythema multiforme, eczema, psoriasis, lichen planus, dermatitis herpetiformis, and certain drug eruptions. Its most frequent precursor is psoriasis. The constant and profuse desquamation, the papery scales and sheets in which the epidermis is shed, are important diagnostic signs ; others are the vivid redness of the eruption, the rapidity with which it is diffused, its universality, the serious impairment of health—sometimes issuing in death—and the frequent absence of itching. From eczema it is distinguished by the absence of exudation and of crusts ; from psoriasis by its rapid spread and universal diffusion ; from pemphigus foliaceus by the absence of loose bullæ and of foul-smelling discharge, the less severe general symptoms, and the greater amenability to treatment ; from lichen ruber planus by the absence of papules, as well as by its rapid extension and involvement of the whole area of the body.

The essential lesions of *pityriasis rosea* are patches or circles, very slightly raised and thinly covered with small scales. The eruption usually shows itself first on the abdomen, though it may begin on the chest, the face, or the arms. It spreads less rapidly than pityriasis rubra, but in two or three weeks the trunk, the face, and the limbs may be covered, and though occasionally it is universal, it seldom extends below the elbow or the knee. The diagnosis is seldom in doubt, the characteristic 'herald patch' with which the rash begins, the pale-red tint, the slight elevation of the patches, the mingling of maculate and circinate lesions, the slight degree of scaliness, and the spontaneous involution, forming a distinctive *ensemble*. The differences it presents from pityriasis rubra have been indicated above. From psoriasis it is differentiated by its less inflammatory character, the more rapid onset, the slight scaliness, its neglect of the situations most vulnerable to psoriastic attack, and the absence of hyperæmic spots on the surface beneath the scales. From seborrhœa corporis, by the dryness of the scales, its much less chronic character, the lesions disappearing spontaneously in a few weeks. From tinea circinata, by the large number and wide distribution of the lesions, and the absence of the fungus which is the cause of cutaneous ringworm. From the maculo-papular syphilide, by the absence of infiltration, the lighter colour, the fact that the palms of the hands are usually spared, and the lack of concomitant syphilitic signs.

Pityriasis rubra pilaris may appear in the form of scaly patches, resembling psoriasis, on the palms and soles, or as a dry eruption, covered with eczematous-looking crusts ; but the papule which soon appears is a more characteristic lesion, and the diagnosis of the condition from psoriasis and other affections will be found under PAPULES (p. 488).

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SCLEROTICS, BLUE.—(See FRACTURE, SPONTANEOUS, p. 242.)

SCOLIOSIS.—(See CURVATURE, SPINAL, p. 153.)

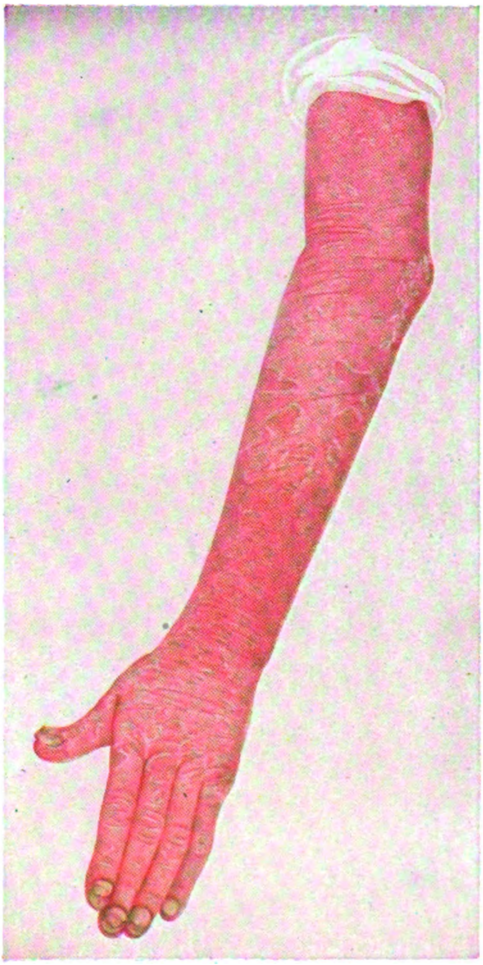
SCOTOMATA.—(See VISION, DEFECTS OF, p. 737.)

SENSATION, SOME ABNORMALITIES OF.—The abnormalities of sensation met with in disease are as numerous as they are important for the purpose of diagnosis. Under PAIN IN THE FACE (p. 446) ; PAIN IN THE UPPER EXTREMITY (p. 442) ; and PAIN IN THE LOWER EXTREMITY (p. 438), the question of subjective pain in relationship to diagnosis has been discussed, and reference will be found on p. 414 to another subjective abnormality of sensation to which the term 'acroparæsthesia' is applied.

Wherever sensory loss occurs, it should assist in forming a conclusion as to the site of disease, even if it does not indicate clearly anything with regard to its nature. In order that the sensory loss may give the necessary information, it is absolutely necessary that the

PLATE XXIV

PITYRIASIS RUBRA



From 'Diseases of the Skin,'
by kind permission of Sir Malcolm Morris, K.C.V.O.

physician should ascertain both the limit and the nature of the loss. He will be able to judge from the shape and locality of the anæsthetic area whether it conforms to a lesion of a peripheral nerve, a spinal root, or some part of the central nervous system. Analysis to show whether the loss is uniform to all forms of sensory stimuli, or whether it is limited to one or two forms only, will provide additional information for diagnosing the situation of the lesion. In order to utilize the information provided by the shape and size of the area of anæsthesia, it is necessary to know what are the areas on the surface of the body which correspond to the distribution of peripheral nerves on the one hand, and of spinal segments or spinal roots on the other. The accompanying diagrams (*Fig.* 255 and *Plate XXV*, p. 608) supply this information to some extent, but in order that it may be used to the best advantage, it is necessary to say a few words about various forms of sensory loss due to lesions in different parts of the nervous system. Before entering upon this part of the subject, we may explain the way in which we propose to use the terms anæsthesia, analgesia, and thermo-anæsthesia.

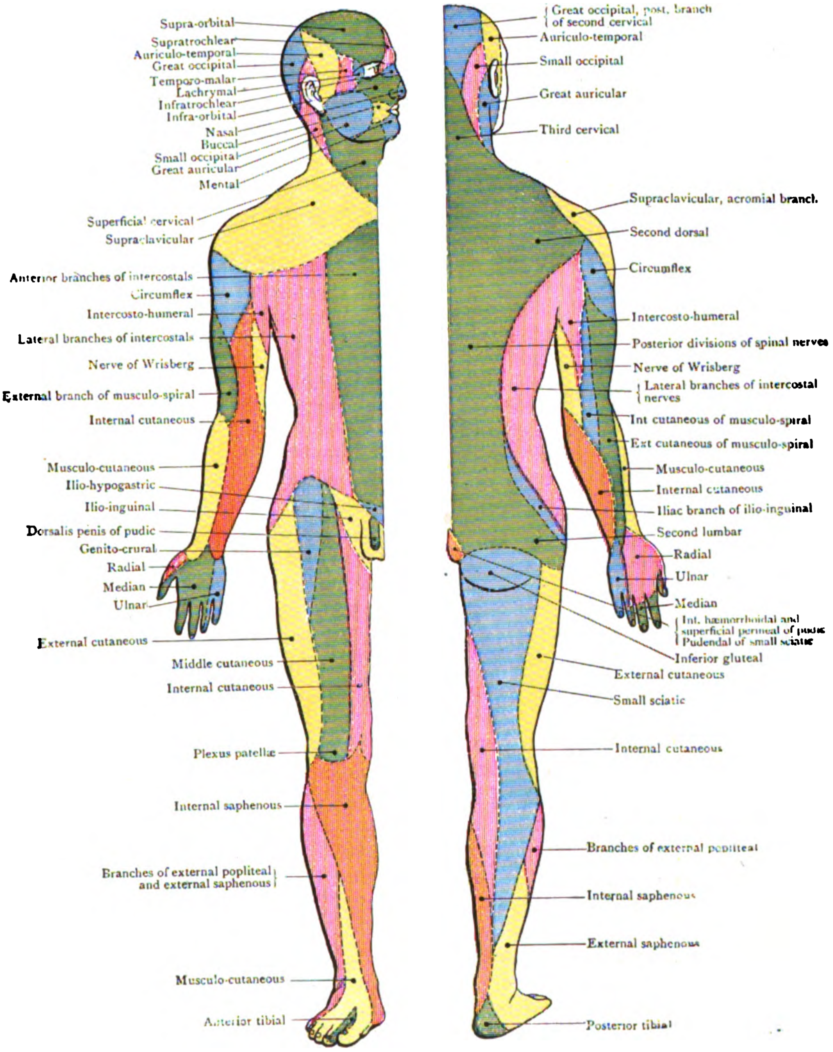


Fig. 255.—The Distribution of Sensory Nerves in the Skin.

Anæsthesia denotes impairment or loss of the cutaneous sensibility to cotton-wool touch, and it is important to remember that parts which are hairless should be chosen for accurate examination.

Analgesia refers to impairment or loss of pain-sense, the adjective 'superficial' being applied when the surface pain produced by the prick of a pin is interfered with, and the adjective 'deep' when the pain usually associated with pinching or squeezing the muscles and deeper tissues is no longer appreciated.

Thermo-anæsthesia indicates loss of appreciation of heat and cold; but the inability to distinguish between things which are warm and cool is not always associated with equal loss of sensibility in distinguishing between objects which are ice-cold and really hot.

Disturbances of Sensation in Peripheral Nerve Lesions.—The afferent mechanism of the peripheral nervous system consists of the following three sub-systems:—

1. *Deep Sensibility.*—This conveys impulses excited by pressure and by all movements of joints, tendons, and muscles. Painful impulses derived from excessive pressure are also carried by this sub-system. By its means a healthy person is able to recognize not only movements of joints, but also the locality of the stimulus and the direction of the movement. The fibres which conduct these sensory impulses run mainly with the muscular nerves, and are not destroyed by division of the sensory nerves to the skin.

2. *Protopathic Sensibility.*—This sub-system responds to painful cutaneous stimuli (pinpricks) and to the more extreme degrees of heat and cold. The appreciation of these stimuli is vague and inexact as to the locality of the spot stimulated.

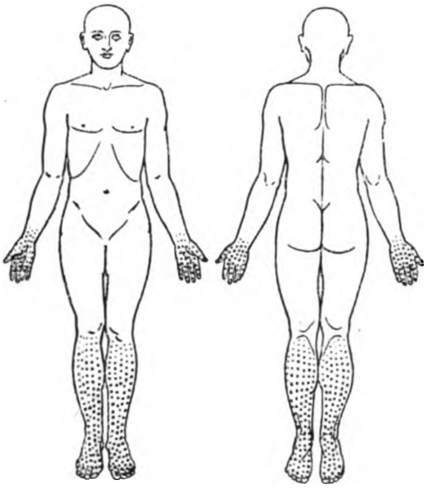


Fig. 256.—Peripheral neuritis. 'Glove and stocking anæsthesia.' Cotton-wool and pinprick sensibility impaired or lost over the dotted areas. This is associated with hyperalgesia of the underlying muscles.

3. *Epicritic Sensibility.*—To this sub-system is due the power of perceiving and locating light touches (cotton-wool), of discriminating between two points applied simultaneously to the surface, and of recognizing the finer grades of temperature called cool and warm.

It has been shown that when a *peripheral cutaneous nerve* is divided the area of epicritic loss is greater than that of protopathic loss; in other words, there is more overlapping of protopathic sensibility than of epicritic sensibility between neighbouring nerve distributions. Thus, if the *ulnar nerve* is divided near the wrist, there is complete loss to touch, superficial pain, heat, and cold, over an area including the little finger and part of the inner edge of the palm of the hand. This is the area of epicritic and protopathic loss. But epicritic loss extends over a wider area which includes half the ring finger and more of the hand (*Fig.* 257). In this area of epicritic loss pain can be recognized but cannot be localized exactly, while light touch is not appreciated, and the discrimination between cool and warm is absent.

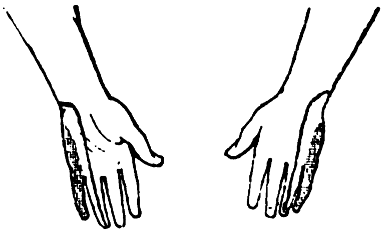


Fig. 257.—Division of ulnar nerve at the wrist. The dark area represents loss of epicritic and protopathic sensibility. The line indicates the limits of epicritic loss. (*After Head and Sherren.*)

Consideration of the above points shows how important it is to define accurately the exact nature of any sensory loss, and to be careful that the appreciation of pressure is not mistaken for the appreciation of light touch. If tactile sensibility is tested by the observer's finger or with the head of a pin, the results will be vitiated, because pressure sensibility is at once brought into action.

Another important diagnostic point depends on the fact that protopathic sensibility returns some months before epicritic sensibility in the process of regeneration after the division of a peripheral nerve. During the stage of protopathic repair there is often a considerable degree of hyperalgesia in the affected area ; that is to say, the pain produced by a pin-prick or a scratch is out of all proportion to the nature of the stimulus.

So far we have dealt with the disturbance of sensibility produced by the disease or injury of a single nerve. In the disease known as *peripheral neuritis* the sensory disturbances are very characteristic, and consist of pain, tingling, tenderness, and cutaneous anæsthesia. Spontaneous pain in the limbs is often complained of, but more important is the intense suffering produced by movements, and especially by handling the limbs or by squeezing the muscles. At the same time cotton-wool touch is often unperceived on the peripheral parts of the limbs, particularly in what are known as the glove and stocking areas (*Fig.* 256). This combination of deep hyperalgesia and cutaneous anæsthesia constitutes an important differential sign between peripheral neuritis and tabes dorsalis, in which superficial and deep analgesia are nearly always associated.

Disturbances of Sensation in Lesions of the Cord.—The impulses of the three peripheral sub-systems—deep, epicritic, and protopathic—combine in new groups soon after they enter the spinal cord. Some impulses cross to the opposite side immediately, others cross after running a short course on the same side, and others ascend to the upper extremity of the cord entirely on the side of their entry. This rearrangement may be summarized briefly thus :—

1. Impulses of pain, whether excited by cutaneous stimuli or by excessive pressure, run together in the spinal cord, and cross, probably soon, to the opposite side.

2. Impulses of temperature *of all degrees* cross to the opposite side and are closely associated, but not intermingled, with those of pain ; the impulses of heat are also separated from those of cold.

3. Impulses excited by light touch and by pressure, and those which subserve their localization, accompany each other, cross to the opposite side, probably less rapidly than those of pain and temperature, and ascend in a path which is distinct from that of the latter.

4. Impulses subserving the senses of passive position and movement are associated with those of tactile discrimination (compass points) in their ascent of the cord on the same side as their entry. These impulses pass up the posterior columns.

The accompanying diagram represents this rearrangement of impulses and their course in the spinal cord.

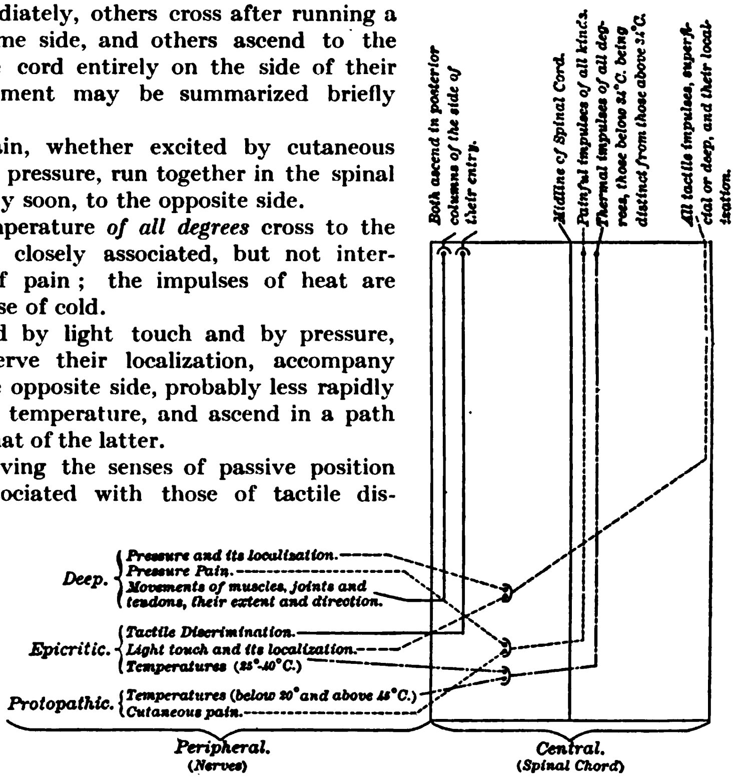

Both ascend in posterior columns of the side of their entry.
Midline of Spinal Cord.
Painful impulses of all kinds.
Thermal impulses of all degrees, those below 31°C. being distinct from those above 31°C.
All tactile impulses, superficial or deep, and their localization.
Deep.
Pressure and its localization.
Pressure Pain.
Movements of muscles, joints and tendons, their extent and direction.
Epicritic.
Tactile Discrimination.
Light touch and its localization.
Temperatures (25°-40°C.)
Protopathic.
Temperatures (below 20° and above 45°C.)
Cutaneous pain.
Peripheral.
(Nerves)
Central.
(Spinal Chord)

Fig. 258.—Diagram illustrating the course of the sensory impulses passing from the peripheral nerves into the spinal cord. (From the author's article in *Osler's] Modern Medicine*, vol. vii.)

The chief points of practical importance in clinical work to be deduced from the above considerations are as follows : In the first place, analgesia resulting from a cord lesion always includes deep as well as superficial pain, and so differs from the analgesia produced by a peripheral nerve lesion in which, as we have seen, superficial analgesia may be associated with deep hyperalgesia. Secondly, a lesion of the spinal cord may abolish the appreciation of thermal stimuli, but, if it does so, the discrimination between all degrees of heat and cold will be lost. This again differs from the effect of some peripheral lesions. In the third place, a lesion of the posterior columns may produce loss of the sense of passive position and movement without any loss of tactile, painful, or thermal stimuli, a combination which does not obtain as the result of a lesion limited to the peripheral nervous system.

In all diseases or injuries of the spinal cord, the degree of sensory loss depends, of course upon the severity of the lesion. On the other hand, the distribution of the sensory loss is of the greatest importance for the diagnosis of the level of the lesion. The distribution must be mapped out carefully, and then compared with the accompanying diagram (*Plate XXV*), which shows the sensory areas corresponding to the different spinal segments.

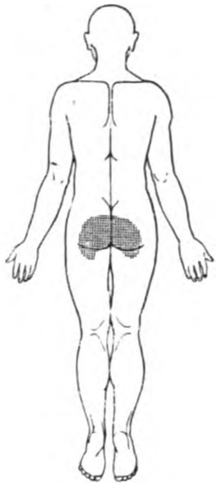


Fig. 259.—Comminuted fracture of the sacrum, with injury to the 3rd, 4th, and 5th sacral roots. Complete loss of sensibility to touch, superficial pain, heat and cold.

In a case of ***myelitis,*** for instance, it may be found that sensation is perfect above the level of the umbilicus, and impaired on the trunk and legs below that level (***Fig.*** **260**). We shall be justified in concluding that the highest point of the disease corresponds to the 9th dorsal segment of the cord. Take another example : *fracture dislocation of the vertebræ* is common in the cervical region, and may crush the spinal cord at the level of the 7th cervical segment. The resulting sensory loss is represented in the accompanying chart (***Fig.*** **261**).

In testing the sensibility of the skin it is always advisable to work from the anæsthetic area towards the normal, and to note not only complete anæsthesia, but all modifications of sensation. For instance, bordering on the region of complete anæsthesia there may be an area in which the patient is able to appreciate a touch or a pin-prick, but in which he describes the sensation produced as differing from the natural sensation elicited by these stimuli. Such modifications should be taken into account in diagnosing the level of the lesion.

As a result of disease or injury of one side of the spinal cord, a symptom-complex called *Brown-Séquard paralysis* is met with. This is discussed on p. 496. ***Fig.*** **204** (p. 497), illustrates the sensory loss in a case of this kind.

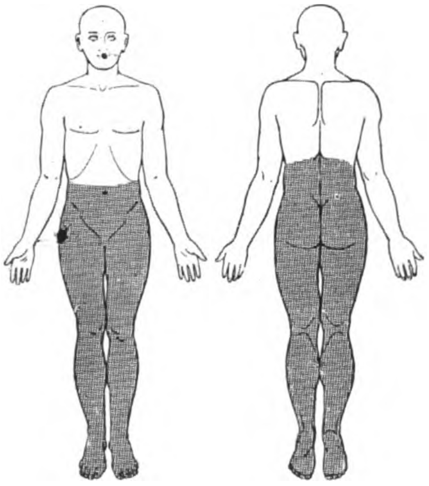


Fig. 260.—Dorsal myelitis affecting the cord as high as the 9th dorsal segment. The shaded parts are insensitive to touch, deep and superficial pain, and all degrees of temperature.

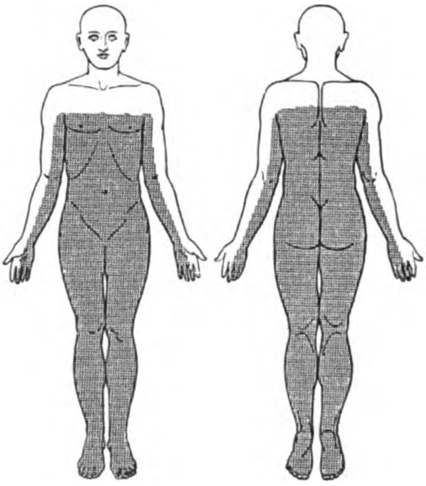
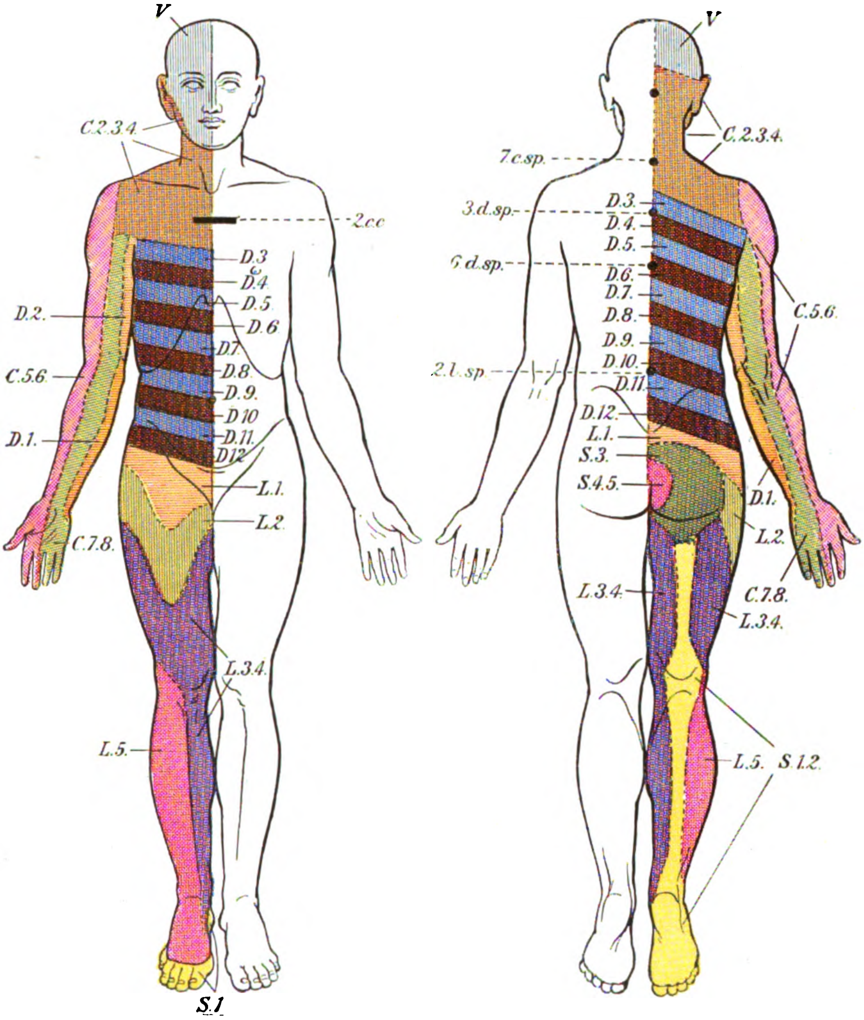


Fig. 261.—Fracture-dislocation of the cervical spine. The shaded area represents the loss of sensibility to touch, pain, heat and cold.

Syringomyelia and *hæmatomyelia* are other conditions in which dissociative anæsthesia is common (***Fig.*** **262**). In the former disease thermo-anæsthesia and analgesia are usually found first in the arms and thorax, and they tend to spread all over the body. In rare instances they begin in the legs or on the face. Their distribution is nearly always asymmetrical. The borders of the cutaneous loss are not sharp but shaded off, and correspond to the limits of spinal-root areas. On the other hand, charts sometimes show regions of dissociative anæsthesia which correspond laterally to one or more root areas, but do not cover their longitudinal extent. For instance, the sensory loss in one hand may be limited above by a line encircling the

PLATE XXV

DIAGRAM SHOWING THE RADICULAR SENSORY AREAS OF THE HUMAN BODY



Copyright. *By Dr. E. Farquhar Buzzard.*

This diagram has been compiled by the writer from a study of similar diagrams published by others, and modified in accordance with his own experience.

Although the various areas depicted in the diagram are essentially Root Areas, the information they supply can be used clinically for the purpose of localizing both radicular and intramedullary lesions. It must be remembered, however, that the deductions in the one case are different from those in the other. If, for instance, the character of the sensory change in one of these areas is of the peripheral type, a radicular lesion of the corresponding segmental level may be diagnosed; if, on the other hand, the sensory loss is of the central type in a particular skin area, the spinal lesion must be sought at a level several segments higher than that which corresponds to the sensory area.

For practical purposes it is important to remember that the uppermost level of sensory change (not the upper level of total analgesia) should be compared with the nearest corresponding line on this diagram.

If employed with an intelligent appreciation of these points, the diagram is of much clinical importance, but it should not be exploited blindly as a mechanical calculator. Individual variations alone are sufficient to demand a considerable margin of error.

forearm, so as to give it the appearance of a glove distribution. Similarly, on the face a central area, including the nose, mouth, and eyes, may preserve its sensibility intact, while the surrounding regions are completely insensitive to painful and thermal stimuli. Thermo-anæsthesia and analgesia are sometimes, but not always, co-extensive. Tactile loss also occurs, but usually supervenes in the later stages of the disease. Subjective sensations may form the initial evidence of the disease, and may be thermal, painful, or tactile. Lancinating pains and cramps are described. More curious are the subjective sensations of drenching sweat in a part which is dry, or of cold in a part which is quite normal in temperature.

In *tabes dorsalis* the disturbances of sensation are numerous and characteristic. Lightning and dull boring pains, tingling, numbness, girdle sensations, and various painful crises are among the subjective abnormalities. Impairment or loss of deep and superficial pain sensibility in various parts of the body is one of the earliest and most important physical signs for the purpose of diagnosis. The cutaneous analgesia is generally found on the legs, and often also in the root areas on the arms and thorax corresponding to the C 8 to D 5 segments (*Fig.* 263). Deep analgesia is nearly always present in the calf muscles.

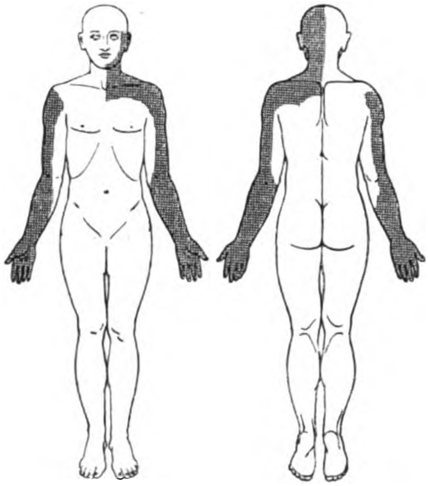


Fig. 262.—Syringomyelia. The shaded parts show the areas of dissociative anæsthesia, i.e., of thermo-anæsthesia and analgesia. This was associated with atrophic palsy of the upper extremities.

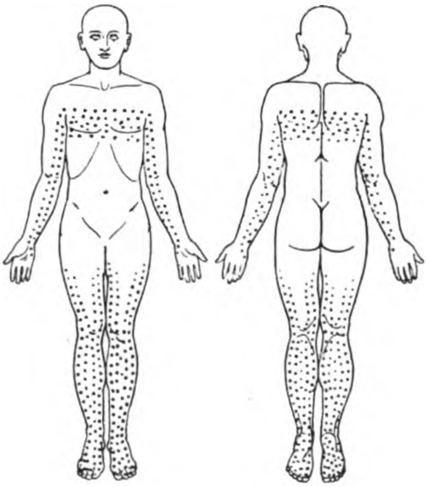


Fig. 263.—Early tabes dorsalis. The dotted areas represent a characteristic distribution of sensory disturbance. The loss is chiefly to painful stimuli, and the superficial analgesia is almost always associated with deep analgesia.

Superficial nerves such as the ulnar may often be found insensitive to rolling or pinching. Delayed sensibility is another phenomenon very characteristic of some cases of tabes dorsalis. Hyperæsthesia may be present, especially in bands around the abdomen, when gastric or intestinal crises form part of the clinical picture. Intolerance of hot or cold water on any part of the skin is described by some patients suffering from severe forms of the disease. Allocheiria, or reference of a sensory stimulus to the opposite side of the body, has also been observed. The sense of position and movement is nearly always disturbed in locomotor ataxy, and this results in varying degrees of inco-ordination and in the production of Romberg's sign. Astereognosis, or the inability to gauge approximately the size and shape of objects placed in the patient's hand, is another common sensory defect.

In *disseminated sclerosis*, sensory troubles do not as a rule constitute so prominent a feature as do the motor disabilities, but subjective and objective changes are by no means uncommon. Numbness of one limb lasting a few weeks or months, girdle sensations, and even pains of a neuralgic type, are sometimes complained of. I have known transient hemianæsthesia to be an initial symptom in one case, and astereognosis with loss of sense of position on one side, to be the earliest signs of disease in two or three cases. From the diagnostic standpoint these are important facts, because it is very tempting to assume, erroneously, that such sensory phenomena, occurring alone without any reflex or motor signs of organic disease, are hysterical in origin. They are doubtless due to patches of disease near the internal capsule.

This brings us to the consideration of the **Abnormalities of Sensation resulting from Disease of the Higher Parts of the Nervous System.** Hemianæsthesia is a common result of the various vascular accidents responsible for *apoplexy* and hemiplegia. It may be present with or without motor paralysis and with or without hemianopia ; sometimes all three phenomena are associated in the case of severe lesions of the internal capsule and optic radiations. In most cases of apoplexy hemianæsthesia is slight and transient. Tactile and pain sensibility may be impaired, usually more so on the limbs than on the trunk, and more especially in the distal portions of the arm and leg. Even when touches are perceived they are localized badly by the patient. In *lesions of the optic thalamus* the opposite side of the body may be the site of a curious sensory disturbance which consists in part of a lowered sensibility to painful stimuli and in part of a great exaggeration of the disagreeable effects produced by such stimuli when they are perceived. For instance, the patient may fail to recognize a light pin-prick so well on the affected as on the sound side, but a scratch may produce an intensely painful sensation referred to a very wide area and not localized to the spot stimulated. In such cases the patient often complains also of paroxysms of severe pain in the affected limbs.

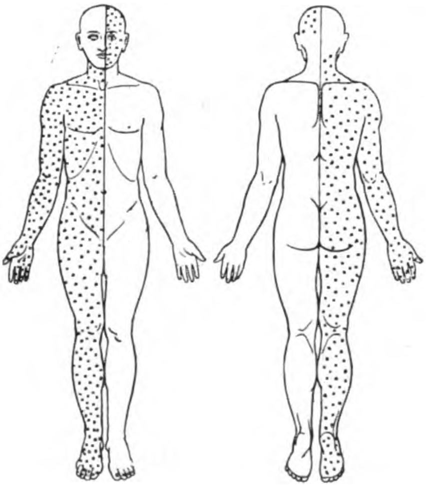


Fig. 264.—Thrombosis of left posterior inferior cerebellar artery. The dotted areas show the regions of dissociative anæsthesia, i.e., loss of sensibility to pain and temperature of all degrees.

The *hemianæsthesia of hysteria* is usually far more complete to all forms of stimulation than any hemianæsthesia due to organic disease of the brain. The hysterical patient is found to have lost taste, smell, and sometimes even hearing, on the anæsthetic side. The visual defect, instead of being hemianopic as in the organic cases, is generally a marked contraction of the visual fields, sometimes amounting to blindness, especially in the eye corresponding to the other sensory defects.

Lesions of the brain-stem may also be responsible for extensive loss of sensation. For instance, thrombosis of the posterior inferior cerebellar artery gives rise to a localized softening on one side of the medulla, which produces thermo-anæsthesia and analgesia on the same side of the face, and on the trunk and limbs of the opposite side (*Fig.* 264). This sensory disturbance is sometimes complicated by homolateral cerebellar ataxy and cranial nerve palsies.

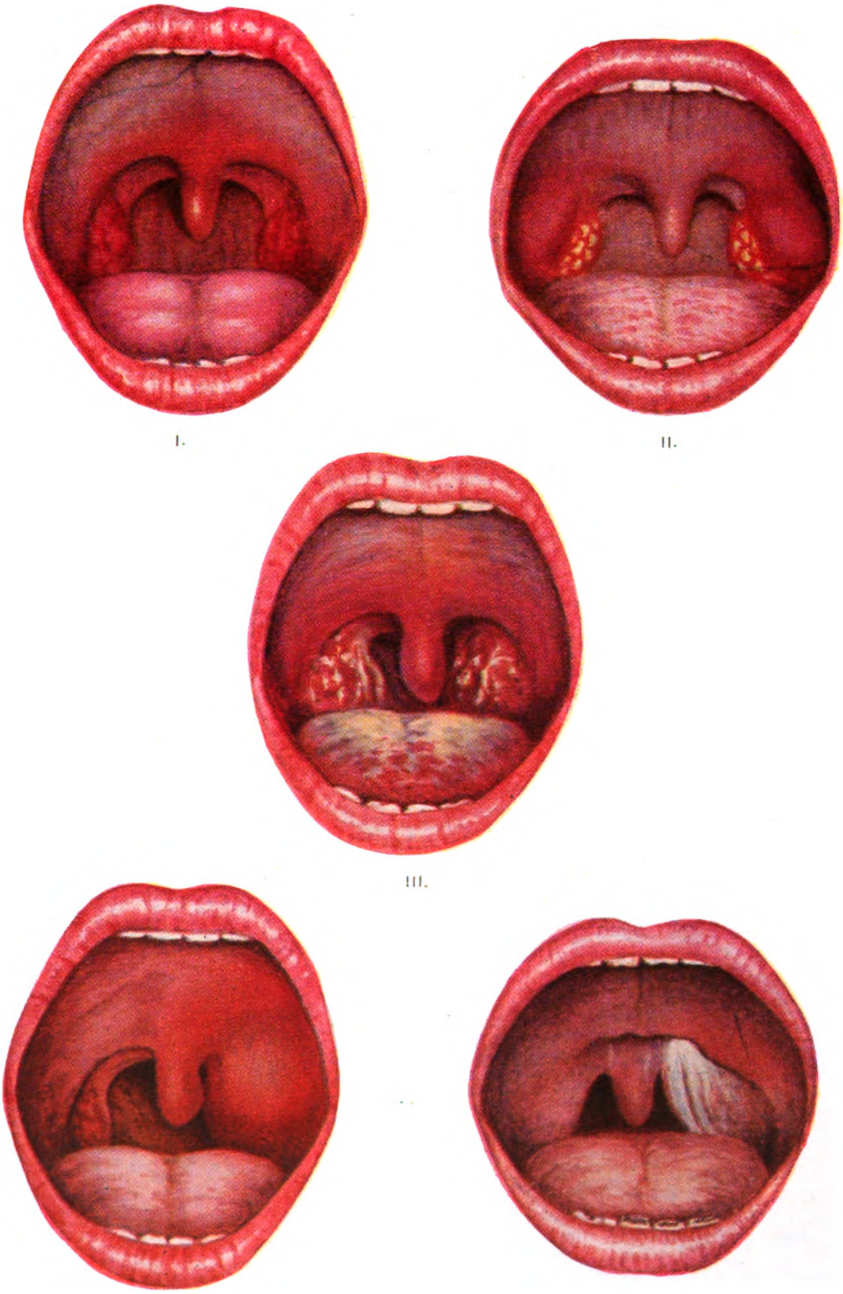
So far we have dealt chiefly with the various forms of lowered sensibility, and have given little attention to perversions of sensation, such as are indicated by the terms *hyperæsthesia* and *paræsthesia*.

Hyperæsthesia is observed in cases of tabes dorsalis and peripheral nerve lesions as described above, but it is also met with in other conditions of organic as well as functional disease. It is found, for instance, in root areas in cases of vertebral and intravertebral disease. In *spinal caries* and in *tumours of the spinal meninges*, a zone of hyperæsthesia may be detected just above the area of anæsthesia produced by the compression of the cord, or it may precede the appearance of compression signs. The increased sensibility is probably caused by pressure on, or irritation of, the posterior root fibres. A similar phenomenon is a frequent accompaniment and sequel of an attack of *herpes zoster*. The shape and situation of such hyperæsthetic zones afford a clue to the site of the lesion. Hyperæsthesiæ as well as paræsthesiæ are among the earliest signs of *subacute combined degeneration of the cord*, and are referred by the patient to peripheral parts of his four extremities. They may precede by many weeks or months the appearance of ataxic or spastic paraplegia and definite areas of sensory loss. Similar symptoms are also complained of in not a few cases of pernicious anæmia and other severe blood diseases, probably on account of scattered degenerations in the nervous system as the result of the Anæmia (p. 20).

Neurasthenic and *hysterical states* are responsible for hyperæsthetic areas which have

PLATE XXVI

SORE THROATS



I.

II.

III.

IV.

V.

I. Ordinary hyperæmic sore throat. **II.** Mild follicular tonsillitis. **III.** Severer follicular tonsillitis. **IV.** Left-sided quinsy. **V.** Syphilitic sore throat.

no relation to central or peripheral innervation. In neurasthenia, especially the traumatic variety, the patches are usually found on and around the spine and over the scalp. In hysterical conditions similar patches may be detected in the mammary and ovarian regions.

Hyperæsthesia in connection with *visceral disease* has been referred to in other articles, such as those on PAIN IN THE FACE (p. 446); PAIN IN THE UPPER EXTREMITY (p. 442); etc.

A very special peculiarity of sensation, known as Magnan's sign, is met with in sufferers from the cocaine habit; it consists in a subjective feeling as of multiple small worms creeping about under the skin, though in some cases the patient compares his sensation to that of fine sand under his skin. *E. Farquhar Buzzard.*

SHIVERING FITS.—(See RIGORS, p. 594.)

SHORTNESS OF BREATH.—(See BREATH, SHORTNESS OF, p. 87.)

SKIN ERUPTIONS.—(See MACULES, p. 382; PAPULES, p. 487; ETC.)

SKIN, PIGMENTATION OF.—(See PIGMENTATION OF THE SKIN, p. 527.)

SKODAIC RESONANCE.—When there is a basal and unilateral pleuritic effusion of medium degree, the pitch of the percussion note over the upper part of the thorax in front is often higher on that side of the chest on which the effusion is than on the other. It is not a question either of impairment of resonance or of hyper-resonance, but merely of pitch. This higher pitch of the percussion note over the upper lobe, when the lower lobe is compressed by an effusion, is named 'Skodaic resonance,' after the observer who first drew attention to it. Its importance is mainly twofold: in the first place it does not indicate disease of that part of the lung which affords the sign—for instance, the fact that, in a case of right-sided effusion, the right upper lobe gives a higher-pitched percussion note than does the left cannot be taken as evidence that there is a lesion, perhaps tuberculous, at the right apex; in the second place, it is erroneous to suppose that skodaic resonance is obtained only in cases of effusion; its occurrence cannot be taken as proof that dullness at the base is due to fluid there. It is true that *pleural effusion* gives it not only in most marked degree, but also most commonly; nevertheless it may also be observed in some cases of *basal pneumonia* without effusion, or as the result of compression of a lower lobe by such causes as *subdiaphragmatic* or *hepatic abscess, hepatic masses* such as *carcinoma, gumma* or *hydatid cyst, great enlargements of the spleen* such as occur in leukæmia, a *big heart*, a *pericardial effusion*, or a *mediastinal* or *pulmonary new growth*.

The cause of skodaic resonance has never been quite decided, and many theories have been propounded about it; clinically, the most serviceable view is that anything that lessens the degree to which the upper lobe is stretched, yet without actually compressing it, may produce a rise in the pitch of its percussion note. Bilateral compression of the bases of the lungs by such lesions as ascites, presumably causes bilateral skodaic resonance, but this is difficult to determine, because the latter is recognizable only when there is a difference of pitch between the two sides.

Skodaic resonance over an upper lobe when there is some lesion affecting the lower lobe on the same side, should not be confused with the tympanic note that may sometimes be heard over the other parts of the thorax. Stomach tympany is heard normally external to and below the precordial region over an area known as Traube's space, which is bounded above by the precordial dullness, behind by the splenic dullness, and below by the rib margin. When the stomach is dilated there may be an abnormal extent of this gastric tympany in the thorax. When the transverse colon is distended with gas, or when it is pushed upwards by something intra-abdominal, it may produce abnormal areas of thoracic tympany, particularly in the lower sternal region or on either side of this. Such conditions can scarcely be mistaken for skodaic resonance, for the latter concerns the upper lobe, and is not a definite tympany, but rather a moderate rise in the pitch of the ordinary percussion note, not as a rule obvious till the two sides are contrasted. *Herbert French.*

SLEEPLESSNESS.—(See INSOMNIA, p. 320.)

SMELL, ABNORMALITIES OF.—Abnormalities of the sense of smell fall into three main categories, namely:—(1) *Too great sensitiveness to smells which actually exist*;

(2) *Deficient sensitiveness to smells which actually exist*; (3) *Subjective sensations of smells which do not exist.*

Too great sensitiveness to existing smells is sometimes a nuisance to the individual, but is seldom a sign of disease. There are great differences in the powers of perception of different sensations in different persons, and just as some can appreciate very slight differences in sounds more than others, so can some detect smells that are indiscernible by others. This is natural idiosyncrasy.

Deficient sensitiveness to actual smells is often but the obverse of the above, and no sign of disease, although it may be a detriment to the individual, especially in certain commercial pursuits in which the varying qualities of products are judged partly by smell. When the power of smell, having been normal, becomes deficient or totally absent, the change may affect one nostril only, or both. The condition may be transient or persistent. The commonest cause of transient anosmia is *acute nasal catarrh*, whether the result of an ordinary *cold*, or of other affections such as *hay fever* (coryza e feno), oncoming *measles*, or the effects of drugs such as *iodide of potassium* or *arsenic*.

Persistent anosmia may be due to :—

(*a*). *Inability to get air freely, or at all, through the nostril,* as the result of :—

Adenoids
Polypi
Dislocated nasal septum
Nasal septal spur
Hypertrophic rhinitis
Syphilis
Necrosis of bones in the nares
Occluded anterior or posterior nares.

(*b*). *Alteration in the olfactory mucous membrane,* so that it no longer transmits impulses to the endings of the olfactory nerve, although the airway is free :—

Atrophic rhinitis
Paralysis of the fifth nerve, leading to undue dryness of the mucosa.

(*c*). *Abnormalities of the olfactory nerves* :—

Congenital absence
Hydrocephalus
Olfactory neuritis, either the result of overstimulation locally by strong noxious vapours, ammonia, or snuff; or part of a general peripheral neuritis
Post-influenzal changes.

(*d*. *Cranial lesions,* especially hæmorrhage, thrombosis, embolism, softening, injury, or tumour of the uncinate gyrus, which is the centre for smell.

(*e*). *General nerve diseases,* especially :—

General paralysis of the insane
Tabes dorsalis

(*f*). *Hysteria.*

There is little need to discuss the above table in detail, for each heading speaks for itself. When a case is being investigated, the history is very important ; it is next necessary to examine the nose carefully through a speculum, and to test the air-way through each nostril ; if there is any local lesion it will generally be obvious, and only after local affections have been excluded should conditions in groups *c*, *d*, *e*, and *f* be discussed. Anosmia will seldom if ever be a prominent symptom, except in connection with local affections of the nose ; when due to any other cause there will nearly always be some other symptoms which will attract attention more than the anosmia itself.

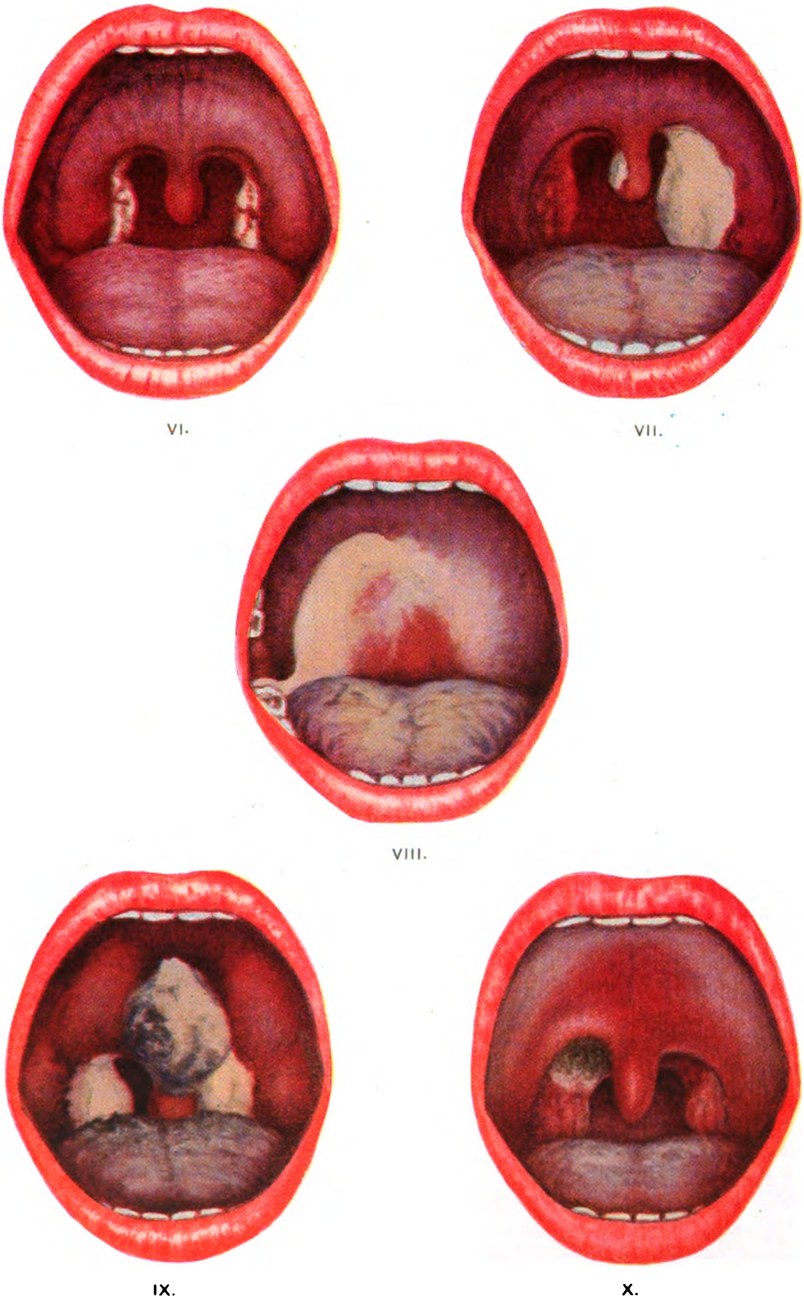
Subjective sensations of smells which do not exist externally may be due to :—

(*a*). Offensive or purulent inflammations of the nose or of the air-cells communicating with it, especially empyema of the antrum of Highmore, or of a frontal sinus.
(*b*). Local thickening of the meninges, tumour of the brain, or interference with the vascular supply, causing irritation of the hippocampal region.
(*c*). An aura preceding an epileptic seizure.
(*d*). Hysteria.
(*e*). Insanity.

In arriving at a diagnosis, it is chiefly important to exclude purulent affections discharging into the nose ; if it is possible to state with certainty that the abnormal sensations have no such organic basis, it is not difficult as a rule to decide between the

PLATE XXVII

SORE THROATS



VI. Mild diphtheria, simulating follicular tonsillitis. **VII.** Diphtheritic sore throat, of medium severity. **VIII** Diphtheritic sore throat of severe type, showing spread of the membranous exudate to the palate. **IX.** Phlegmonous diphtheria. **X.** Vincent's angina.

other causes. It is a curious fact that subjective abnormalities of smell are apt to be associated with delusional insanity concerning the genital organs, in which the prognosis is not free from acute dangers. *Herbert French.*

SNORING may be a very troublesome symptom in some patients, particularly to those who have to sleep in the same room with them; but it is often less an indication of disease than merely a bad habit. Most snorers sleep with their mouths open, and breathe partly through the nose and partly through the mouth; but it is possible for snoring to occur with the mouth completely shut, and nothing the matter with the nasal passages. The tendency is increased, however, by any obstruction of the nasal airway, so that particular examination should be made for such lesions as undue smallness of the nares or a tendency for the soft parts of the nostrils to collapse on inspiration, deflected septum, hypertrophic rhinitis, polypi, adenoids, acute or chronic nasal catarrh, inflammation of the accessory sinuses or of the pharyngeal tonsils, or even a fibrosarcoma or other neoplasm of the nasopharynx. *Herbert French.*

SORE THROAT may be due to one or other of many different causes:—

1. **Affections of the Tonsils:—**

Quinsy.
Acute Tonsillitis.
  (*a*). With reddening and swelling only:
    Acute inflammation due to various micro-organisms, especially to streptococci; staphylococci; pneumococci; pneumobacilli; diphtheria bacilli; Hofmann's bacilli; Vincent's spirilla and fusiform bacilli; spirochæta (treponema) pallida; micrococci catarrhales; bacilli influenzæ; tubercle bacilli. The sore throats of scarlet fever, rheumatic fever, German measles, and measles are probably not due to specific micro-organisms, but to streptococci, or others of the bacteria just enumerated.
  (*b*). With redness, swelling, and exudation:
    Follicular tonsillitis due to streptococci, etc., as above
    Diphtheria
    Vincent's angina
    Syphilis
    Kirkland's disease, or epidemic cervical adenitis.
  (*c*). With ulceration:
    Phlegmonous tonsillitis due to streptococci, etc., as above
    Syphilis
    Vincent's angina.
Chronic Affections of the Tonsils:—
  Recurrent inflammation often associated with adenoids, or tonsillar hypertrophy, especially in children
  Primary or secondary syphilis
  Vincent's angina
  Squamous-celled carcinoma (epithelioma)
  Sarcoma Gumma. Tubercle.

2. **Inflammation of the Soft Palate, Uvula, and Fauces:—**

Catarrh, occurring either by itself or associated with any of the varieties of tonsillitis enumerated above
Gumma
Squamous-celled carcinoma
Tubercle.

3. **Affections of the Pharynx:—**

Acute catarrhal pharyngitis due to any of the micro-organisms mentioned under the heading of acute tonsillitis
Chronic granular pharyngitis due to smoking in excess; or to over-use, as in clergyman's, costermonger's, or stockbroker's sore throat
Squamous-celled carcinoma of the pharynx
Post-pharyngeal abscess
Varicella
Variola.

4. **Laryngeal Conditions, especially:—**

Acute catarrhal laryngitis due to any of the organisms mentioned above
Tuberculous laryngitis
Syphilitic laryngitis
Carcinoma laryngitis
Acute perichondritis of the arytenoid, thyroid, or cricoid cartilages.

5. **Sore Throats the result of the Swallowing or Inhalation of Irritants:**

Corrosives, such as alkalies or strong acids
Ammonia vapour, chlorine fumes
Hot steam.

6. **Mumps.**

7. **Acute and Subacute Adenitis** of the lymphatic glands in the neck.

Notwithstanding the length of the above list the differential diagnosis of a sore throat in practice is not difficult as a rule. Inquiry into the history and inspection of the parts locally in a good light will generally serve to give one a shrewd notion of the nature of the complaint. The chief point in practice is to determine as soon as possible whether the Klebs-Löffler bacilli of diphtheria are present or not, for it has been established that there is no kind of sore throat which can be recognized clinically as non-diphtheritic. It is important that swabbings should be taken from the inflamed parts and examined by a bacteriologist both directly in films stained by Neisser's method or one of its modifications, and by means of cultures. Similar bacteriological investigations will serve to determine which of the organisms mentioned above is responsible for an acute or follicular or ulcerative sore throat other than diphtheritic, it being borne in mind, however, that the organism should be found in fairly pure cultures if it is to be regarded as causative and not merely as a secondary or even casual infection.

Affections of the Tonsils.

Quinsy is practically always asymmetrical, one tonsil being very much more bulged than the other ; the surface is reddened, generally without follicular suppuration, and the diagnosis is ultimately confirmed by the bursting of the abscess.

The presence of Klebs-Löffler bacilli in association with a sore throat may be regarded as conclusive proof that the lesion is *diphtheria*, even though there may be no typical diphtheritic exudate. Diphtheria having been excluded, the diagnosis of one of the other varieties of acute tonsillitis is rendered possible. The frequency with which *acute rheumatism* is associated with recurrent tonsillitis, especially in young people between the ages of five and twenty, should always be borne in mind ; the patient may or may not have suffered from other effects of acute rheumatism, such as joint pains, endocarditis (evidenced by the bruits), pericarditis, pleurisy, erythema nodosum, chorea ; or a history of such rheumatic affections may be obtained in other members of the same family. The tonsillitis is benefited by sodium salicylate, but by no means to the same extent as are the joint pains, so that the effect of treatment is not by itself conclusive evidence of the nature of the complaint.

When acute rheumatism gives rise simultaneously to generalized erythema and to tonsillitis, there will be very considerable difficulty in excluding *scarlet fever*, especially if there has been considerable nausea or actual vomiting ; in some such cases the diagnosis will be one of opinion only ; that which was regarded at first as acute rheumatic tonsillitis and erythema may prove to have been scarlatina after all, should the patient presently develop acute nephritis, or if other members of the family develop typical scarlatina ; the occurrence of extensive peeling of the skin is not conclusive evidence of scarlatinal erythema and sore throat. If the patient is known to have had scarlet fever formerly, the rheumatic nature of the case is more likely.

Follicular tonsillitis is not a final diagnosis, for it may be due to various different micro-organisms, and there is no doubt that the diphtheria bacillus may produce that which to inspection presents multiple foci of pus in the different tonsillar crypts formerly regarded as characteristic of follicular as distinct from diphtheritic tonsillitis. The only conclusive proof that a comparatively simple follicular tonsillitis is not diphtheritic is bacteriological examination. If clinical points alone have to be relied on, one would say that the higher the temperature, the greater the constitutional disturbances, and the more sudden the onset, the more likely is it not to be diphtheria.

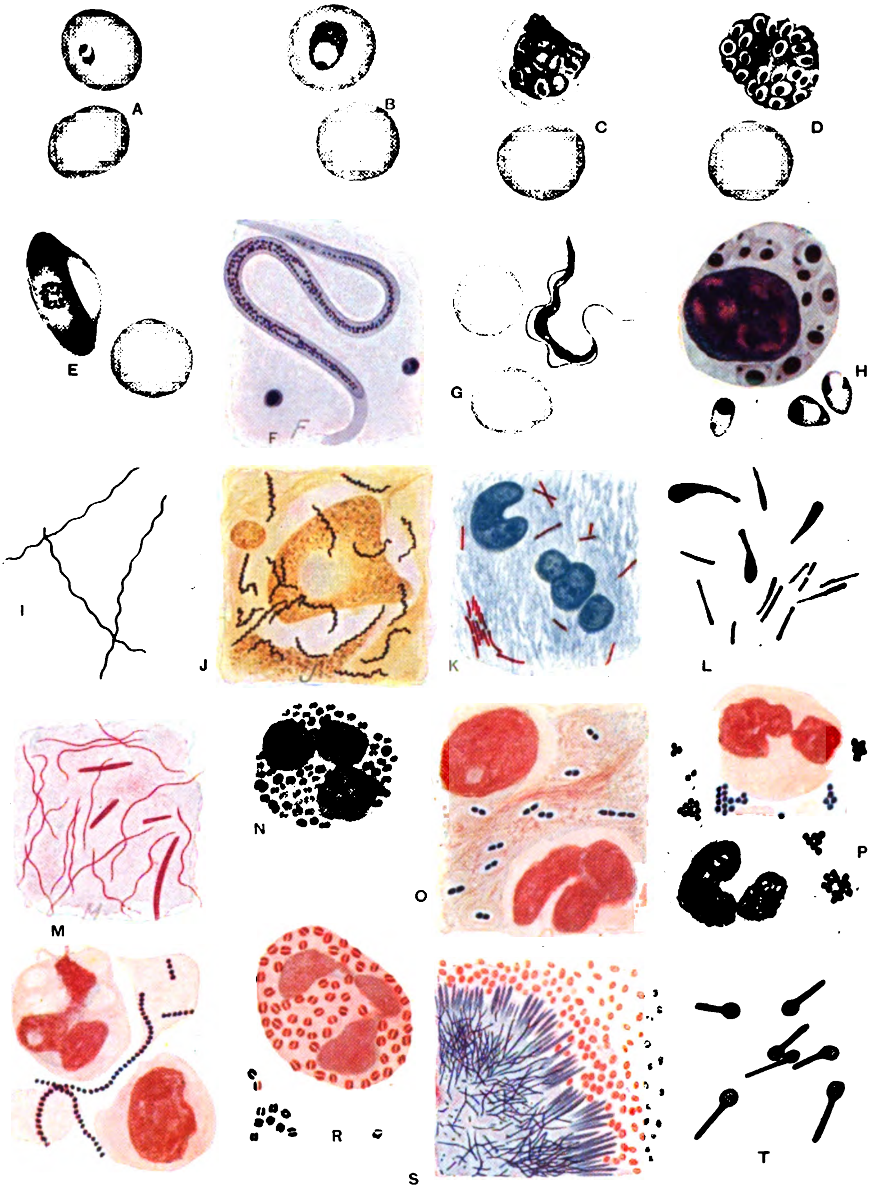
Vincent's angina has been differentiated bacteriologically, cases of this kind having formerly been regarded either as diphtheria or as follicular tonsillitis. The characteristic micro-organisms present are elongated fusiform bacilli symbiotic with spirilla (*Plate XXVIII, Fig. M*). The disease is, as a rule, more resistant to treatment than are other forms of acute sore throat, so that what has originally been a tonsillitis with exudation presently becomes an ulceration associated with remarkable fœtor of the breath, but without that tendency to fatal termination presented by most other varieties of acute ulcerative or phlegmonous sore throat. The disease cannot be recognized without the aid of the bacteriologist.

Syphilis may cause acute soreness of the throat in its secondary stages, and unless that possibility is borne in mind, one may diagnose as acute simple or acute follicular tonsillitis that which is really syphilitic. There is more or less pyrexia, and in most cases

PLATE XXVIII

BACTERIA, AND BLOOD PARASITES

As seen under the $\frac{1}{12}$th inch oil-immersion lens.



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J. R. Ford, del.

A, Malaria, early ring form; **B**, Malaria, ordinary ring form; **C**, Malaria, mature tertian; **D**, Malaria, tertian, ready to sporulate; **E**, Malaria, crescentic; **F**, Filaria embryo; **G**, Trypanosoma Gambiense; **H**, Leishman-Donovan bodies, obtained by splenic puncture; **I**, Spirochæta Obermeieri of relapsing fever; **J**, Spirochæta pallida of syphilis; **K**, Tubercle bacilli and pus cells; **L**, Diphtheria bacilli; **M**, Vincent's angina, Spirilla and Fusiform bacilli; **N**, Meningococci within a leucocyte; **O**, Pneumococci and pus cells; **P**, Staphylococci and pus cells; **Q**, Streptococci and pus cells; **R**, Gonococci, in and outside of pus cells; **S**, Antinomyces; **T**, Tetanus bacilli.

(*The magnification of* **F** *and* **S** *is much less than that of the remainder; the relative magnifications are indicated by the relative sizes of the white corpuscles.*)

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there will be tenderness of the scalp and of the bones, together with the well-known roseolous eruption upon the skin and the 'snail-track' ulcers upon the pharynx. The diagnosis may be more difficult in women than in men, for in the latter the remains of the chancre may still be obvious. Wassermann's serum test may assist materially in the diagnosis of doubtful cases.

Chronic affections of the tonsils in children are nearly always the result of recurrent acute attacks of non-diphtheritic tonsillitis, generally in association with adenoids, both affections arising from the habit of mouth breathing. Inspection of the bulging hypertrophied tonsils, with or without digital examination of the posterior nasal fossæ, will indicate the diagnosis. In an older person, simple hypertrophy from recurrent tonsillitis becomes progressively less common. Chronic tonsillitis in a young adult may be diphtheritic, syphilitic, or due to Vincent's angina, the diagnosis between these being arrived at in the way described above. It may be mentioned that in very rare instances an actual chancre appears upon one tonsil, giving rise, unless secondary symptoms are present, to much difficulty in diagnosis until the case has been watched. *Malignant disease of the tonsils*, whether *squamous-celled carcinoma*, or *sarcoma*, is fortunately not very common; when it does develop, its comparatively non-acute course and its unilateral distribution with progressive ulceration of the central parts and overgrowth of the edges of the neoplasm will point to the diagnosis. A *gumma* of the tonsil is rare, but may at first simulate squamous-celled carcinoma: in case of doubt a small portion of the suspicious mass might be excised, and examined microscopically; or if operative measures were not to be adopted at once, potassium iodide or salvarsan might be administered, and the lesion would be shown to be gummatous if it were thereby relieved or cured. *Tuberculous ulceration* of the tonsils is uncommon, practically never primary, but nearly always preceded by both phthisis and tuberculosis of the larynx. The diagnosis will be indicated by the discovery of tubercle bacilli in the sputum, though it should not be forgotten that carcinoma or gumma might affect the tonsil in a person who has phthisis.

Inflammation of the Soft Palate, Uvula, and Fauces.

This may be seen in many cases of common cold; in association with acute rheumatism; in persons who have recently returned to town from a holiday; in patients who have been subjected to the influence of motor-car dust stirred up from the roads after the latter have been dry for about three days—a longer period of dryness seems to lead to relative disinfection of the dust, whilst rain keeps the dust from rising, so that either continuance of fine weather or a return to wet leads to a disappearance of the sore throats; or in those who are subjected to the influence of relatively concentrated microbes, as in the air of old-fashioned hospital wards, of ill-ventilated much-inhabited rooms, of sewers, and the like. Often a rim of acute reddening is to be seen all along the edge of the anterior pillars of the fauces, and affecting much of the uvula and of the soft palate, producing, as a rule, but little pyrexia, though much discomfort in swallowing, and a raspy feeling at the back of the mouth on first waking in the morning. This inflammation of the palate and fauces may or may not be associated with tonsillitis, pharyngitis, or laryngitis; it is clearly microbial; and doubtless more than one of the varieties of bacteria mentioned above may produce the lesion. The diagnosis of the fact of inflammation is obvious on inspection; that of the nature of the micro-organism requires skilled bacteriological assistance.

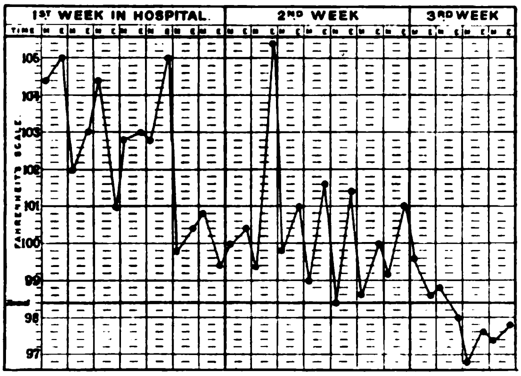


Fig. 265.—Temperature chart in a severe case of Kirkland's disease in which the sore throat and constitutional symptoms were more pronounced than the cervical adenitis. In an average case the temperature lasts less than a week.

During the last few years a new and common disease, epidemic in character, and with acute sore throat, fever, and enlargement of the upper cervical glands as its main symptoms, has been described by Kirkland, of Cheltenham; it is spoken of variously as *Kirkland's disease*, the *Cheltenham disease*, or *epidemic cervical adenitis.* In some respects it resembles diphtheria, in some rheumatic fever, and in some atypical scarlatina without rash, but it differs from all these according to most observers, and it is now regarded as a specific malady. It affects young and old alike, is very infectious, generally occurs in epidemic form, may be mild, medium, or very severe, but generally ends in recovery. Starting with sore throat and a rise of temperature to 102° F. or higher, the patient soon complains mainly of extreme tenderness of the neck, and on palpation acute tender swelling of the parotid, submaxillary, and jugulo-facial glands is found; the swelling may be very considerable, but it generally resolves without suppuration. Fever may last only a day or two, but in severe cases it may persist for a fortnight, especially if the malady relapses after a period of improvement as it is apt to do (*Fig.* 265). The tonsils, uvula, fauces, and pharynx may be merely red and injected; or on the other hand there may be an exudation simulating diphtheria. On cultivation of swabbings from the throat no Klebs-Löffler bacilli can be found, but as a rule streptococci in abundance. What is very remarkable is that in most of the cases the heart is affected during the illness, but without producing permanent valvular changes like those of rheumatic fever; the impulse becomes diffuse, the heart dilates, there is often a local systolic mitral or aortic bruit, or both, and the pulse may be both feeble and intermittent. There is seldom albuminuria, but the general toxæmia in severe cases may be enough to cause delirium and temporary coma. There are no definite joint pains, though the patient aches all over. Convalescence is generally slow at first, two or three weeks elapsing before the patient begins to feel anything like well; but complete recovery soon takes place after this.

Gumma, *squamous-celled carcinoma*, and *tuberculous affections* of the fauces, soft palate, and uvula are relatively uncommon; they are differentiated in the same way as has been described in connection with tonsillitis.

Affections of the Pharynx.

Acute pharyngitis may occur by itself, or in association with acute tonsillitis or acute laryngitis, and in either case it may be due to any of the micro-organisms mentioned above. The differential diagnosis of the micro-organisms is carried out in the bacteriological laboratory. The fact of acute pharyngitis is determined by careful inspection of the structures at the back of the mouth.

Chronic pharyngitis is generally the result of excessive smoking, or of the undue use of the voice, in which latter case it may be associated with hoarseness or ready tiring of the voice, as in those who have to declaim loud and long—clergymen, stockbrokers, costermongers, public orators, and others. The history will generally point to the nature of such a case, but one should examine the sputum and the lungs for evidence of tuberculosis, and also look for signs of syphilis or new growth, in order to exclude these possible alternatives.

Post-pharyngeal abscess is a cause of acute dyspnœa rather than of soreness of the throat; it is almost confined to infancy; after three or four years of age the disease becomes almost unknown. It might be simulated by spinal caries, in which, long before there is soreness of the throat, there is severe pain in the cervical region of the spine, especially on movement.

Chicken-pox and *small-pox* may each produce its characteristic eruption in the mouth, pharynx, and œsophagus, and thus give rise to sore throat; but the latter symptom will never be present by itself, and the presence of the cutaneous papules, vesicles, or pustules, will indicate the diagnosis, especially if there have been other similar cases in the neighbourhood.

Laryngeal Conditions.

Acute laryngitis may be due to the same micro-organisms as acute tonsillitis; soreness of the throat is generally less complained of than is huskiness, or weakness, or loss of voice. The nature of the inflammation is determined bacteriologically. One variety of acute non-diphtheritic laryngitis that merits special mention is that due to pyogenic cocci—pneumococci, streptococci, staphylococci—which in a few cases, in addition to producing acute superficial inflammation, also lead to rapid and extreme œdema of the larynx, with death from asphyxia unless tracheotomy be performed speedily. These cases have been spoken of as *acute suffocative œdema* of the larynx.

Tuberculous, syphilitic, and *carcinomatous* lesions of the larynx are less acute, though they may have relatively acute exacerbations or become secondarily infected with pyogenic cocci; their diagnosis depends partly upon laryngoscopic inspection—tuberculosis and syphilis being bilateral, whilst new growth is generally unilateral; upon examination of the sputum for tubercle bacilli, and of the lungs for apical physical signs of phthisis; upon the beneficial influence of potassium iodide, mercury, or salvarsan; upon Wassermann's syphilitic serum test; upon microscopic examination of small excised portions; or upon the course of the disease.

Sore Throats the Result of the Swallowing or Inhalation of Irritants and Corrosives are diagnosed as a rule by the history; inquiry will generally suffice to indicate that some irritant has been taken, or there may be direct evidence of it in the form of eschars on the lips or the buccal mucosa; there may be vomiting and hæmatemesis; analysis of the gastric contents may indicate the nature of the poison taken; ammonia may be detected by the smell.

Mumps and **Acute Adenitis of the Cervical Lymphatic Glands** may each produce marked soreness of the throat in addition to dysphagia, stiffness, discomfort, and pain. Mumps is not difficult to diagnose unless its possibility is forgotten, in which case it might be mistaken for acute œdema of the neck or other similar lesions. The way in which the swelling is located in the salivary glands, starting on one side and spreading to both, is often pathognomonic. Cervical adenitis might simulate mumps, but careful palpation will generally enable one to determine that the swelling is not in the salivary but in the lymphatic glands, and it will only remain to decide what has been the source of the infection. This will probably have been from some inflammatory, ulcerative, or malignant focus in connection with the shoulders, neck, head, face, lips, cheeks, gums, teeth, tongue, fauces, uvula, palate, tonsils, pharynx, or nares; the differential diagnosis will be based upon inspection and palpation of the parts, together with bacteriological examination.

It only remains to add that *scarlet fever* is at the present time so atypical that acute cervical adenitis may really be of scarlatinal origin without any scarlatiniform rash having been observed upon the skin. One would naturally look for evidence of desquamation, sore throat, bald tongue, albuminuria, nephritis, perhaps otitis media; but there is no doubt that some cases of acute cervical adenitis are really scarlatinal, without there having been any other sign of this disease except pyrexia and sore throat. A few such cases prove rapidly fatal, and they have recently been recorded as examples of acute and fatal sore throat corresponding to one form of the *angina maligna* of the eighteenth century.

Herbert French.

SORES, PENILE.—Sores on the penis may be present on the thin mucous covering of the glans or prepuce, or on the cutaneous surface of the body of the penis; they are more common in the former situation.

Ulceration in the neighbourhood of the glans penis may be due to:—

1. Balanitis
2. Herpes progenitalis
3. Soft sore
4. Chancre
5. Epithelioma
6. Gummatous ulceration
7. Tuberculous ulceration.

Balanitis.—If inflammatory processes have been allowed to continue beneath the prepuce, ulceration and excoriation of the mucous membrane covering the glans penis or lining the prepuce will occur. The surface of the glans will be denuded of epithelium, and the process will be accompanied by a stinking, purulent discharge. Multiple shallow ulcers are formed, rapidly coalescing and causing considerable discomfort. The prepuce often becomes swollen and œdematous, preventing retraction, so that a condition of phimosis occurs; care must then be exercised in diagnosing a simple balanitis from one accompanying acute gonorrhœal urethritis or an underlying syphilitic or soft chancre. With an acute urethritis, there will be a history of infection, pain along the course of the urethra during micturition, and perhaps chordee; further, the intracellular gonococcus may be identified in a stained smear of the discharge (*Plate XXVIII, Fig. K*, p. 614).

If a chancre exist under the swollen phimosed prepuce, there is often a tender spot about the corona or at the frænum. With a soft sore, consecutive sores may appear about the orifice of the prepuce, whilst the inguinal glands are much more likely to be inflamed or to suppurate than with simple balanitis. A syphilitic chancre obscured by a phimosis can usually be felt distinctly under the skin, and causes a comparatively small amount of discharge, whilst the inguinal glands become enlarged but do not suppurate. The history of the date of infection, Wassermann's reaction, or the subsequent appearance of secondary symptoms, will help materially in the diagnosis. Spirochætes may be found in the discharge.

A form of balanitis which is frequently very obstinate to treatment may occur in patients the subjects of gout or diabetes mellitus.

Herpes Progenitalis.—Herpes may attack the genital organs as part of a herpes zoster in which the cutaneous eruption depends upon some lesion of the central nervous system, or as a local affection, the so-called catarrhal herpes. The disease begins as a patch of erythema on the inner surface of the prepuce or on the glans penis, followed by the appearance of vesicles and pustules; the latter become rubbed by the clothes, and form small ulcers. Herpes of the genital organs tends to recur, so that a previous history of a similar attack is often forthcoming. If seen during the vesicular stage, no difficulty will be met with in the diagnosis; but if suppuration has followed, it must be diagnosed from a venereal sore. Soft chancres are usually deeper, with marked edges; their base is sloughing, and they are usually accompanied by a bubo, which is exceptional with herpes. A syphilitic chancre is usually single, indurated and raised, and is accompanied by the typical, multiple, discrete, amygdaloid glands in the inguinal region. It should be remembered that syphilis may become inoculated upon a herpetic patch, or that herpes may appear in an area previously inoculated with the syphilitic virus.

Soft Sores or **Chancroids** of the penis occur almost invariably from infection during sexual connection. The incubation period is short, a vesicle occurs in two days, and this breaks down rapidly to form a rounded or oval ulcer with sharply defined edges, and a yellowish sloughing base. The ulcers appear usually on the mucous surface of the glans, frænum, or corona, and are most often multiple, direct inoculation occurring from an ulcer to the contiguous part. They may cause rapid destruction of tissue, perforating the frænum or spreading over the surface of the glans. The soft sore must be differentiated from others occurring on the glans, and above all from a syphilitic chancre. At the same time it must be remembered that besides the infection with chancroid, a simultaneous infection with syphilis may have taken place, so that a soft sore may ultimately become indurated and assume the character of a primary syphilitic lesion. The chancroids are multiple, are accompanied by a good deal of thin, purulent discharge, and by a painful swelling of the inguinal glands, usually of one side, which have a marked tendency to suppurate. On the other hand, a syphilitic chancre is nearly always single, is raised and indurated, has little discharge, and is accompanied by enlarged, but firm and indolent, glands in both inguinal regions; the incubation period of a syphilitic chancre is from twenty-one to twenty-eight days.

The multiple ulcerations caused by herpes are more superficial, and rarely cause a bubo.

Chancre—the initial lesion of syphilis—generally appears on the penis, and is most common in the neighbourhood of the frænum or coronary sulcus. A chancre appears about twenty-five days after infection, as a reddened patch, which becomes raised above the surface of the mucous membrane, with distinctly indurated margins. The central part breaks down into an ulcer, discharging a thin, purulent fluid, and at the same time the inguinal glands of both sides become palpable, slightly enlarged, but discrete, and with no tendency to suppurate. The chancre increases but slowly in size, or may occasionally become smaller without any treatment, and after a further lapse of from four to six weeks the typical secondary symptoms make their appearance; namely, a roseolar rash on the chest, abdomen, face, and thighs, general adenitis, and mucous patches about the faucial pillars and tonsils. The diagnosis of the primary lesion of syphilis frequently presents no difficulties, the indurated character of the sore, the date of its appearance after infection, and the presence of firm, indurated glands in the inguinal region, being distinctive. In other cases the character of the sore is by no means distinctive, and it is necessary to differentiate it from other lesions of the penis. If the sore be syphilitic, the secondary manifestations of the disease will follow, provided that the doubtful ulcer is not treated as a chancre. Thus, in any case in which syphilis is suspected, but not wholly certain, it is advisable to withhold any specific treatment for syphilis until such time as secondary symptoms appear, so that a patient may not be condemned to the lengthy process of treatment for syphilis until the diagnosis is absolutely certain. The *Spirochæta pallida* should be looked for in scrapings from the affected parts, but too much reliance should not be placed upon a negative Wassermann blood-reaction in the early stages of the disease.

A chancre may be simulated by an inflamed soft sore, especially if the latter has undergone cauterization. Soft sores are, however, frequently multiple, appear within a few days of infection, and are accompanied by painful enlargement of the inguinal lymphatic glands, which are particularly prone to suppurate. It must not be forgotten that a double infection may have occurred, so that a soft sore may show little inclination to heal or, becoming indurated, may present the features of a chancre after about three weeks, and later, the symptoms of constitutional syphilis.

Epithelioma of the penis in the early stage may be confused with syphilitic chancre. In epithelioma there is no history of infection ; it occurs only in elderly patients, and there is frequently a greater destruction of tissue than in syphilis. The inguinal glands are not enlarged until the sore has been present for some weeks, and there are no secondary lesions such as the faucial ulceration and cutaneous rash. If any doubt exists, a small piece may be removed from the edge of the ulcer for microscopical examination.

Perhaps the greatest difficulty in the diagnosis of a chancre is experienced when the latter is hidden beneath an inflamed and phimosed prepuce. There is a purulent and foul discharge from beneath the œdematous and swollen prepuce : the inguinal glands are enlarged from the associated sepsis. If a chancre is present, it can frequently be felt as an indurated area under the prepuce, whilst if it has been present for some time, the secondary lesions of syphilis may be present. If any doubt exists in an elderly patient as to whether an indurated subpreputial area be an early epithelioma or a syphilitic sore, the prepuce should be split up along the dorsal aspect under anæsthesia, the ulceration inspected, and a small piece submitted to microscopical examination if necessary.

Epithelioma (squamous-celled carcinoma) is the most common form of malignant growth of the penis. It arises most frequently from the inner aspect of the prepuce, or from the mucous membrane of the glans, as a small, raised ulcer, with friable, irregular edges. It is rarely present before the age of forty, and frequently occurs on the site of previous ulceration or long-standing irritation. An epitheliomatous ulcer increases gradually in size, in spite of various forms of treatment, and with it is frequently associated glandular enlargement in the inguinal area. At first the glands may be enlarged from septic infection, but later from malignant infiltration. An epitheliomatous ulcer may in some cases be confused with a chancre ; but the friable, irregular edges of the former, the liability to bleed, and the gradual progressive increase in size in spite of treatment in an elderly patient, should give rise to grave suspicion of malignant disease. Microscopical examination of a small piece removed from the edge of the ulcer will give direct evidence of epithelioma.

Gummatous Ulceration of the penis occurs occasionally, resulting from the disintegration of a small gumma of the glans or prepuce, frequently in the position of an old scar. A gumma commences as a small, elevated nodule, which, if left untreated, softens and discharges its contents, leaving an ulcer bounded by thin edges and with a yellowish, sloughy base. A gummatous ulcer has been mistaken for a primary lesion of syphilis ; but the absence of induration, the history of the onset and of a previous infection with syphilis, would be points against a chancre. A second infection with syphilis is by no means unknown, especially in those who have had salvarsan alone in the treatment of the first attack, but it is rare. Occasionally the base of a gummatous ulcer proliferates into a papillary tumour and has given rise to a suspicion of carcinoma ; the diagnosis will be confirmed by the behaviour of the lesion under potassium iodide, when a tertiary syphilitic affection will clear up rapidly.

Tuberculous or **Lupoid Ulceration** of the penis is rare, and is generally associated with advanced tuberculous infiltration elsewhere. Tuberculous ulcers are usually shallow, with thin overhanging edges, painful and multiple. The infection has resulted from the rite of infantile circumcision by the Jewish method.

R. H. Jocelyn Swan.

SORES, PERINEAL.—Ulceration may be present in the perineum as the result of:—

1. Cutaneous inflammations or injury
2. Urethral fistulæ or suppuration
3. Prostatic suppuration
4. Syphilis
5. Epithelioma.

Cutaneous Inflammation or Injury.—An ulcer in the perineum may result from *direct injury* to the area, or from inflammatory *infection of the sebaceous* or *hair follicles* of the cutaneous covering. An ulcer from these causes may be placed at the centre or to one side of the perineum, is movable on the deeper parts, and shows no track into which a probe can be passed. In women, ulceration of the perineal area may be associated with *gonorrhœal* or *septic vaginal discharge.*

Urethral Suppurations or Fistulæ.—During the progress of an acute urethritis, a glandular follicle frequently becomes infected. The suppurative process leading from this in the bulbous urethra may extend towards the perineum and open externally, leaving a small fistula which may or may nor discharge urine during the act of micturition. In a similar manner, urinary fistulæ may result from inflammatory processes behind a urethral stricture, and in an old-standing case it is not uncommon to find a urinary calculus in the dilated portion of the urethra behind the stricture. When the urethral suppuration is acute and an abscess bursts in the perineum, the diagnosis will be quite obvious, and the ordinary treatment for an abscess, in addition to that of the acute urethritis, will usually suffice to cure the condition.

If, however, the perineal wound discharges urine, it will be found that this occurs as a rule only during the act of micturition, as there is no interference with the vesical sphincter. In nearly all cases, however, a stricture of the urethra will be found, though not necessarily one of sufficient degree to cause severe interference with micturition. Endoscopic examination will show the presence of a urethral stricture, whilst behind it can be seen frequently the sloughy granulations denoting the position of the urethral opening of the fistula. Occasionally urine drains from a perineal fistula continuously, and not only during the act of micturition. In these cases there is constant soaking of the perineal skin, and frequently excoriation. That urine should leak constantly from the fistula denotes interference with the vesical sphincter, either by dilatation behind a tight urethral stricture, by the presence of a calculus in the prostatic or membranous urethra, or by actual division of the vesical sphincter following some operation, such as perineal prostatectomy or perineal lithotomy.

Diseases of the Prostate.—An abscess or tuberculous focus in the prostate may occasionally discharge in the perineum, and remain as a sinus. An abscess in the prostate arises practically always from some infection in the posterior urethra, from venereal causes, or after septic instrumentation. It is accompanied by urethral discharge, or there is a history of a recent infection, whilst per rectum the prostate may be felt to be inflamed, or scarred from the shrinkage of the abscess cavity.

When a tuberculous cavity in the prostate opens in the perineum there is advanced tuberculous disease, so that little difficulty will be found in arriving at a diagnosis. A tuberculous prostate is very rarely a primary condition, but in most cases is secondary to disease in the testis or bladder, so that examination of these organs will in nearly all cases give evidence of tuberculous disease and indicate the nature of the perineal fistula. Palpation of the prostate per rectum may reveal the rounded nodular deposit of tubercle in the gland.

Syphilis may cause ulceration on the perineum either as a chancre or as mucous tubercles. A *chancre* on the perineum is rare. It forms a small ulcer with slightly indurated borders, indolent in character, and accompanied by slight enlargement of the inguinal lymphatic glands. A chancre of the skin does not possess the usual features of a genital chancre, and is not usually diagnosed with certainty until the secondary lesions of syphilis become apparent ; but an ulcer with raised, infiltrated edges, which shows no tendency to heal under aseptic precautions, should always give rise to a suspicion of syphilis. The *Spirochœta pallida* may be looked for, and Wassermann's serum test tried.

Condylomata may be present about the perineum in association with active syphilis, They may extend from the anal or vulval orifice, and form oval or rounded, flat-topped, sessile masses, covered by macerated, greyish epithelium, or they may be ulcerated on the surface. The accompanying signs of syphilis will indicate the diagnosis.

Epitheliomatous Ulceration of the perineum is practically only seen as a direct spread of a growth of the anus or vulval area, when the diagnosis presents no difficulty. An epithelioma may develop in the scar of some former cutaneous affection, in which case an ulceration may exist, showing the usual characteristics of a cutaneous epithelioma namely, gradual progressive increase in size, raised, friable edges, and tendency to slight hæmorrhages. The inguinal glands may be enlarged early from inflammatory absorption, or later by infection with malignant disease. In case of doubt a fragment may be removed for microscopical examination.

R. H. Jocelyn Swan.

SORES, SCROTAL.—Ulceration of the scrotum occurs in association with :—

1. New growth :
   - Epithelioma
   - Papilloma.
2. Fistulæ.
3. Syphilis.
4. Testicular disease :
   - Inflammatory
   - Tuberculous
   - Syphilitic.
5. Suppurating cysts.

Epithelioma of the Scrotum, commonly known as 'chimney-sweep's cancer,' is by no means limited to this avocation, but is certainly more common in men engaged in work in which they are exposed to much irritation from solid particles or from noxious fumes. Hence the disease is most commonly seen amongst chimney-sweeps, employés in gas-works, paraffin and chemical works, and coal-mines. It often begins as a small subcutaneous nodule, over which the skin is thinned and adherent ; the nodule enlarges slowly, and the thinned covering gives way, to form an ulcer with thickened, irregular edges and a tendency to bleed on slight injury. The ulcerated area extends both radially and into the tissues of the scrotum, later involving the testes. The inguinal lymphatic glands become enlarged soon after active ulceration commences, at first from inflammatory causes, later from malignant infiltration. In other cases a scrotal epithelioma begins in a *wart* or *papilloma*, which may have been present for years with only slight increase in growth. These soft papillomata are not unusually the starting-point of malignant change, when they become more vascular, whilst the surface epithelium becomes thinned and easily excoriated. A small amount of foul discharge is present, often encrusted into a scab, which on removal leaves an ulcer with indurated, everted edges, with the gradual progress of a cutaneous epithelioma. Any ulcer on the scrotum, especially if indurated or readily caused to bleed, must be looked upon with extreme suspicion, and when it does not improve with ordinary antiseptic medication, should be widely removed without waiting for glandular enlargement.

Epithelioma may occur in the scrotal area as a localized recurrence after removal of a malignant growth of the penis or testicle. Knowledge of the previous condition for which operation has been performed would give the diagnosis.

Fistulæ may occur in the scrotum and cause ulceration. They are most common in association with tuberculous or syphilitic disease of the testes (see below), but occasionally they occur from urethral extravasation, or burrowing from rectal suppuration. An abscess may form and open through the scrotal skin from a peri-urethral abscess accompanying an acute urethritis or formed by septic infection behind a urethral stricture. In either case a small amount of urine may leak through the opening during micturition, whilst the history of urethral discharge, or of difficulty in micturition and other symptoms of stricture, will point to the diagnosis.

Syphilis of the Scrotum may be present either as a primary chancre or as a mucous tubercle. A *primary chancre* in this situation is by no means easy to recognize unless other signs of syphilis are present ; but the presence of a cutaneous sore which does not show much inclination to heal under non-mercurial antiseptic dressings should always give a suspicion of syphilis. There is often only slight induration of the ulcer compared with that of a penile chancre, but the edge is raised and of a rolled appearance. The inguinal lymphatic glands are enlarged and discrete, and some five to six weeks after the commencement of the ulcer the usual secondary symptoms of syphilis become manifest.

Mucous tubercles may be present on the scrotum, usually on the femoral aspect. They may extend directly from the anal area. No difficulty will be met with in the diagnosis, as other signs of syphilis are obvious.

Testicular Disease.—In some cases extension of disease in the testicle may involve the coverings of the scrotum, and may even perforate them to form a scrotal sore. This sequence occasionally occurs with : (1) A testicular abscess ; (2) Tuberculosis of the testis ; (3) Gumma of the testis.

A *testicular abscess* is somewhat uncommon, but may arise from direct extension from the urethra via the vesiculæ seminales and vasa deferentia or by a hæmatogenous infection during the course of a specific fever, such as scarlet fever, mumps, or enterica. It may also follow chronic torsio testis. With urethral disease, the primary trouble may be due to gonorrhœa, or more frequently to a septic urethritis from the introduction of infected instruments, and is thus not infrequent in cases of prostatic enlargement in which the patient is passing his own catheter. In cases in which the infective process extends from the urethra, the epididymis is affected first, whilst in the metastatic cases the body of the testis usually shows the first sign of enlargement. These acute inflammations of the testis occasionally suppurate, when the scrotal tunics become inflamed and adherent, whilst softening occurs later, and unless surgically relieved the abscess opens through the skin, leaving an ulcer, and a sinus discharging pus.

Tuberculosis of the testicle may occur as a primary disease or as a secondary deposit in association with tuberculosis elsewhere in the genito-urinary tract. Testicular tubercle almost always begins as a nodule in the epididymis, but in the later progress of the disease may extend into the testicle proper. If the tuberculous nodule progresses rather than undergoes cure, the scrotal skin becomes adherent, thinned, and finally perforated, leaving a shallow ulcer with thin, undermined edges, and discharging thin pus. Occasionally the necrotic tubules of the epididymis fungate through the opening in the scrotum, appearing as a greyish, sloughy projection from the cutaneous opening—the so-called 'hernia testis.'

A *gumma of the testis* causes a swelling in the body of the testis rather than in the epididymis. A gumma which remains unrecognized or untreated may soften and ulcerate through the scrotal skin in a manner similar to tuberculous disease, leaving a clearly-defined ulcerated area with sharply-cut margins, and a wash-leather-like sloughy base. The gummatous granulation tissue may fungate through the scrotal aperture, forming a yellowish necrotic mass.

The diagnosis of these three conditions may produce some difficulty in the earlier stages (see SWELLING, SCROTAL, p. 696), but in the advanced stage now under consideration, when an open scrotal sore is present, the diagnosis is easier. The *opening of a testicular abscess* on the scrotum leaves a small sinus discharging pus and accompanied by a general enlargement of the organ. Preceding the rupture of the abscess there is acute pain in the testicle, with rise of temperature, rigors, and general signs of suppuration, which are much diminished as soon as the abscess is allowed to burst. There is often a urethral discharge, which, however, is often much lessened with the onset of the acute epididymitis, with distinct thickening of the cord and aching pain in the neighbourhood of the external abdominal ring, or in metastatic cases the abscess occurs during the progress of an acute fever. The general history is one of acute pain commencing in the testicle, with rapid and extremely tender swelling of the organ, followed by abscess-formation.

In *tuberculosis of the testis* the progress is much more gradual. A nodule may have been present in the epididymis for some time, gradually enlarging, but causing very little pain; in some cases a nodule may have been present for months without any apparent change, and then it may enlarge suddenly, involve the scrotal tunics, and discharge its contents. By the time the disease has reached this stage, it is probable that evidence of tuberculous trouble will be found in other organs, particularly the other testis, prostate, seminal vesicles, or bladder. The affected testicle usually presents several nodules in the epididymis, tender on pressure, whilst small nodules may also be felt in the vas deferens.

The opening remaining from the discharge of a *gummatous orchitis* is usually a rounded ulcer with sharply-cut edges and yellowish base. The whole testis is enlarged, practically painless, and feels heavy. The cord is not thickened, and there is no evidence of disease in the other testicle, prostate, or seminal vesicles. There is probably a history of syphilis, and other tertiary syphilitic lesions may be present elsewhere, such as gummatous periostitis. Strong evidence of the syphilitic nature of the disease is often obtained by the result of treatment with large doses of potassium iodide, alone or in combination with mercury or with salvarsan, when a gumma diminishes in size with marked rapidity. It should be remarked, however, that, as in two cases under the writer's care, testes which are subsequently removed and found to contain large gummata, may show no improvement before operation, even under large doses of iodides, though the Wassermann test may be positive.

A *hernial protrusion of necrotic testicular tissue* may be present either with tuberculous disease or from a gumma. In tuberculosis the mass is greyish and necrotic, discharging thin pus, and there will be sufficient evidence of tuberculous disease in the underlying testis and other genital organs. Tubercle bacilli may be found in the discharge. A distinctive feature of the gummatous hernia testis is found in the appearance of the cutaneous opening ; if the fungating mass be pushed aside, the opening in the scrotal skin will be seen to be cleanly cut and to encircle the protruding tissue tightly. The fungating hernia testis of tubercle or syphilis must also be diagnosed from other conditions producing a raised tumour on the scrotum. An epithelioma of the scrotum has raised borders, but the centre is excavated, and there is rarely any enlargement of the testis. A sloughing papilloma of the scrotum may more nearly reproduce the appearance, but the tumour and the skin are freely movable on the underlying testis, whilst in hernia testis the mass is connected with the testicle, and the tubular structure of the latter is often apparent on picking up a small fragment of the fungating tumour.

Cysts of the Scrotum.—As an exceptional occurrence, a sebaceous cyst may develop in the scrotal skin, suppurate, and leave an open sore. The areas remaining present raised borders, and are easily mistaken for an early epithelioma. An accurate history of the previous swelling in the skin is of little assistance in these cases, but microscopical examination of a piece removed from the margin of the ulcer will exclude malignancy. A suppurating cyst in the scrotum is more uncommon than epithelioma.

R. H. Jocelyn Swan.

SPASTICITY.—(See Gait, Abnormalities of, p. 251.)

SPEECH, ABNORMALITIES OF.—Abnormalities of speech are numerous, varying from complete mutism to slight defects in articulation, and dependent on disturbances, functional or organic, in some part of the complex mechanism which is responsible for the production of intelligible language. This article is intended to expose the broad principles by which various abnormalities of speech can be detected and used for the purposes of diagnosis ; it does not embrace a discussion of the controversial views which are held concerning their exact production.

The amount of investigation required for making a diagnosis in cases of speech abnormality varies within wide limits. Great care is called for in examining cases of aphasia which result from disturbance in the function of the cerebral speech centres or their dependent paths of communication ; the defective articulation of a patient suffering from cleft palate needs only a comparatively superficial examination in order to arrive at a correct diagnosis.

It will be convenient to consider the various abnormalities of speech under the following heads : (1) *Mental defects* ; (2) *Aphasia* ; (3) *Deaf mutism* ; (4) *Dysarthria* ; (5) *Functional disorders—stammering, lalling, idioglossia.*

Mental Defects.—The acquirement of the power of speech may be delayed in children who are mentally defective, and in some forms of idiocy may be suspended altogether. Before making a diagnosis of mental deficiency in a child who appears to be dilatory in talking, it is well to remember that the age at which speech is acquired is very variable, and that the delay may be considerable where no mental impairment is present. In such cases the diagnosis must depend on a consideration of other points in the child's development. Inquiry should be made as to whether he is clean in his habits, whether he is destructive, whether he plays with toys or with other children in a natural manner, and whether he displays abnormally bad temper or irritability. In some cases the delay in speaking may be due to a defect in hearing which has been unsuspected by the parents. This point is especially apt to arise in respect to children who have begun to talk at the normal age, and who have lost what little they had learned of the art in the sequel of some acute illness.

In adults, loss of speech may be due to many forms of mental deficiency of a temporary or permanent nature. A familiar example of temporary loss of speech is that degree of alcoholic intoxication to which the term ' speechless ' is vulgarly applied. Similarly, the intoxication of the higher mental faculties associated with organic poisons, such as those of pneumonia or typhoid fever, may be responsible for temporary loss of speech. Complete mutism due to disease of the higher intellectual centres is common in various forms of dementia, and is proved to be no aphasic defect by the sudden and complete restoration of speech which may take place after months or even years of silence. The speechlessness of a melancholic patient or of one who is suffering from paralytic dementia is further differentiated from true aphasia by the fact that the latter is associated with attempts at communication, while the former is not. On the other hand, general paralysis of the insane is a disease in which temporary aphasia is by no means uncommon, especially in connection with the transient hemiplegia following 'congestive' attacks.

Aphasia.—A definition of aphasia is difficult to supply in a few words. The term is used to denote that loss of speech which does not depend on mental deficiency, nor upon paralysis of the motor mechanism of articulation. Such a negative description requires, however, some modification, because aphasia is frequently associated with some impairment of intelligence resulting from disturbance of internal language, which plays an important part in all intellectual processes, and any lesion of the cerebral centres connected with it must necessarily interfere with the higher mental activities. This is particularly the case in what is called sensory aphasia, that variety which depends upon a lesion of the auditory and visual word centres situated in the cortex near the posterior part of the left Sylvian fissure of the brain.

In right-handed persons the chief speech centres are placed in the left cerebral hemisphere (*Fig.* 266), and it is customary to consider them as being three in number. The posterior part of the first temporal convolution is regarded as the area in which the auditory memories of spoken words are stored and recalled. It plays an important part in the development of speech, because it is largely through the sense of hearing that the child first learns to associate objects with their names and expressions with their meaning. The cortex in the angular gyrus has a similar special function in regard to the storage of visual word memories, a function which bears the same relationship to written language as the auditory word centre has to spoken language. These two portions of the cortex constitute the sensory speech centres. A third important centre is called the motor, or, better, the higher kinæsthetic centre, and this is located in Broca's area, or the posterior part of the third frontal convolution. In this situation are stored the memories of afferent impulses excited by the motor activities employed in speech. Unless this centre is intact, the conversion of internal into external language is imperfect or impossible. In the opinion of some authorities, there is a similar kinæsthetic centre in the posterior part of the left second frontal convolution, which plays a part in connection with written language comparable to the part played by Broca's area in relation to spoken language.

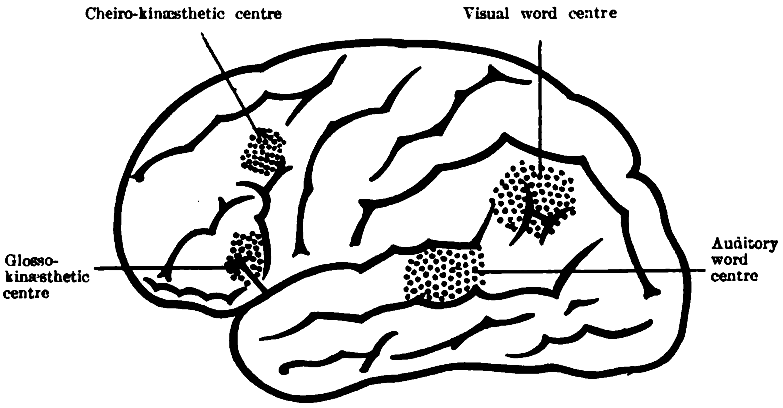


Fig. 266.—Left cerebral hemisphere, with speech centres.

With these physiological and anatomical data as a basis, we can proceed to consider the chief varieties of aphasia and the points in their differential diagnosis. Before doing so, it is well to sound a note of warning with regard to the complications which are constantly being met with by the clinician in attempting to analyse cases of aphasia. In the first place, a diagrammatic anatomical definition of the cerebral centres is liable to give a wrong impression. These centres are more diffuse in their function than they appear to be on a map of the brain, and they are much more interdependent than their topography would suggest. Communicating nervous tracts bind them together in such a way that a destructive lesion of one must necessarily upset the function of another, and so modify profoundly the clinical picture of any particular case. In the second place, aphasia is, in most instances, the result of a vascular lesion, and all the centres referred to lie in the area supplied by one artery—the middle cerebral. Consequently even when the main brunt of a vascular disturbance falls on one of the special speech centres, the others may also suffer more or less, temporarily or permanently, from disturbances of nutrition. In any case of aphasia, therefore, we may have to be satisfied if we can arrive at a conclusion as to the site of the chief defect, without being able to define the exact limits of the loss or impairment of cerebral function. In the third place, due allowance must be made for the fact that the right cerebral hemisphere may gradually acquire some degree of speech activity, especially in cases of aphasia occurring during the earlier years of life, and may tend to replace the loss caused by the defective action of the left.

Word-deafness is the result either of a lesion of the auditory word centre in the temporal cortex, or of one which isolates that centre from the periphery: that is to say, of a sub-cortical lesion cutting off the centre from auditory impulses. In either case the patient who is word-deaf is unable to recognize the meaning of spoken language, although he may hear perfectly the sounds by which it is conveyed. He fails to understand anything which is said to him, and does not obey simple commands so long as they are not accompanied by gestures suggestive of their meaning. If the visual word centre has not been affected at the same time, he will still be able to read and to understand what is written. He will depend upon writing and reading for his means of communication with others. The amount of interference with spontaneous speech will depend upon whether the lesion is cortical or sub-cortical. If the latter the integrity of the auditory word centre preserves internal speech, and so permits the patient to speak spontaneously with fluency and probably with accuracy, and his power of writing will be equally unimpaired. When the cortical centre is itself destroyed, internal language is thoroughly disorganized, and although some spontaneous speech may be possible, it is certain to be more or less unintelligible. According to the extent of the lesion, it will vary between a speech containing inaccuracies of minor importance, and one which is a jargon incapable of interpretation. Characteristic of this defect is the fact that the patient himself does not appreciate the mistakes he makes. His written language is likely to be more accurate and more intelligible than his spoken language, but it will probably not reach a very high standard. He may copy with accuracy, but is quite unable to write from dictation. Such are the usual chief attributes of word-deafness in its pure form. Clinically, word-deafness is usually accompanied by word-blindness, to a greater or less extent.

Word-blindness, or *Alexia,* is produced by a lesion of the left angular gyrus, and may or may not be accompanied by defective vision. As in the case of word-deafness, it may result from a cortical or from a sub-cortical lesion, and it is in association with the latter class of case that HEMIANOPSIA (p. 300) is most common. In cortical word-blindness the patient is unable to read, although he sees the letters clearly and may even be able to copy them in the same way as a child copies letters when learning the alphabet. Writing conveys no meaning to his mind, although in the less severe cases the patient may still recognize familiar words, such as his name. There are, in fact, varying degrees of word-blindness, some of which are difficult to understand and to analyze. The word-blind patient suffers in his spontaneous speech to a greater or less extent according to whether he uses his visual or his auditory memories chiefly in the process of internal language. Should he be a 'visual' his spontaneous speech will suffer much more than if he is an 'auditive.' The terms 'visual' and 'auditive' are used to distinguish two classes of persons, the first depending more on their visual memories of words, and the second more on their auditory memories of words in the course of reviving them for the purposes of internal thought and speech. Spontaneous writing is likely to be lost completely, but writing from dictation may be carried out with more or less accuracy. In word-blindness due to a sub-cortical lesion, although hemianopia is almost certain to be present, spontaneous speech and spontaneous writing are preserved perfectly, although the power of reading and of copying hand-written sentences into printed capitals is entirely in abeyance.

When word-blindness and word-deafness coexist, the condition is called *sensory aphasia*, and is one to which some authorities believe that the term aphasia should be limited. It is, however, usual to describe a motor aphasia which may be dependent upon a cortical or sub-cortical lesion.

Cortical motor aphasia results from a destructive lesion of Broca's area, the part of the cortex which stores memories of the afferent impulses excited by speech, and in which such memories must be revived if spontaneous speech is to be carried out perfectly. This form of motor aphasia may be present without any paralysis, but it is usually accompanied by some disturbances of internal speech, and perhaps even by some defective understanding of spoken and written language, which, however, never amounts to true sensory aphasia.

Much more common is the *sub-cortical motor aphasia*, which is due to a lesion cutting off Broca's cortical area from the motor mechanism connected with articulation. In this form intellectual processes and internal language may be perfectly intact, but in most cases the inability to speak is associated with right hemiplegia in right-handed persons, or with left hemiplegia in left-handed individuals. The imperfect speech of the patient who is partly aphasic from a sub-cortical motor lesion may resemble to some extent that of the patient who is word-deaf; but the former is conscious of his mistakes and the latter is not. Sub-cortical motor aphasia may perhaps be described better as an articulatory rather than a speech defect: as an anarthria rather than an aphasia. All the attributes for speech are preserved, but its emission is impossible.

Agraphia results usually from a lesion of the visual word-centre, or perhaps in some cases from a lesion of the posterior part of the left second frontal convolution. In the former case the power of writing may be lost, although there is no paralysis of the arm or hand. In the latter case the agraphia is usually associated with right hemiplegia, and in order to test whether the power of communicating thoughts by written language is preserved, the patient must be asked to use the left hand for the purpose. There is some doubt as to whether pure motor agraphia occurs, and some doubt as to the lesion upon which it may depend. I have had experience of the clinical occurrence of pure motor agraphia without being able to correlate the phenomenon with its anatomical basis.

We have now considered the various forms of aphasia and have indicated their points of distinction. This will serve as a basis for diagnosing the site of the lesion responsible for the speech defect, but the nature of the lesion must be determined from other considerations. Vascular lesions, for instance, are usually acute in their onset, sudden in the case of *embolism*, less precipitate as a rule in cases of *hæmorrhage* or *thrombosis*. In *cerebral tumour* or *abscess* the onset of symptoms is more gradual, and local troubles such as that of aphasia are generally accompanied or preceded by the symptoms of increased intracranial pressure in the form of headache, vomiting, and optic neuritis. But aphasia is not always the result of a gross and permanent lesion. Transitory aphasia may be observed in the sequel of epileptiform convulsions, or may be in itself an *epileptic equivalent* —a form of *petit mal* in an epileptic subject. Temporary aphasia occurs also in connection with *migraine*, and I have known it to occur at intervals during a period of thirty years in a woman who was perfectly healthy in every respect, and who showed no other symptoms suggestive of either epilepsy or migraine.

Dysarthria, or in its extreme form '*anarthria*,' is the term used to describe defective articulation as opposed to defective speech. Articulation is carried on by certain muscles of the larynx, pharynx, palate, tongue, and lips which are innervated by the bulbar nuclei, and the latter are set into action by voluntary impulses coming from the motor cortex of both cerebral hemispheres viâ the pyramidal tracts. The articulatory movements, therefore, are represented bilaterally in the brain, and, like other bilaterally represented movements of the body, are not disorganized by unilateral lesions of the pyramidal system. Thus, in cases of hemiplegia without aphasia, there is little or no defect in articulation, and the examination of such a patient shows that both vocal cords, both sides of the palate, and the tongue, retain their power of voluntary movement almost, if not quite, to perfection.

Suprabulbar dysarthria is induced, however, in cases of *double hemiplegia*, when the fibres from both hemispheres to the bulbar nuclei are interfered with by destructive lesions. When a right-sided stroke is followed by a left-sided stroke, or when double hemiplegia results from a lesion in the pons, dysarthria results. In such cases the power of speech may be perfect, but the ability to articulate naturally and clearly is disturbed. The patient is not aphasic but dysarthric. Articulation is usually slow, spastic, and indistinct, if it is not altogether unintelligible. These cases are differentiated from cases of dysarthria due to lesions of the bulbar nuclei or of the cranial nerves, not only by the presence of other hemiplegic signs in the limbs and trunk, but by the fact that the tongue retains its shape, nutrition, and normal electrical reactions, and the palate its natural reflex. This condition of *pseudo-bulbar palsy*, as it is sometimes called, is further distinguished by facial starchiness or spasticity, and by the patient's inability to control the expression of his emotions.

Dysarthria of similar origin, but generally of less degree, may be observed in cases of *general paralysis of the insane*, in *cerebral diplegia*, and in *disseminated sclerosis*. In the latter disease the terms 'staccato' or 'scanning' are applied to describe the articulatory defect. Some cases of *Friedreich's ataxy* exhibit a form of articulation which is slow and jerky, not unlike that of disseminated sclerosis. Probably some degree of inco-ordination enters into the production of this peculiar utterance. In all these diseases the diagnosis of the condition depends upon the presence of other symptoms and physical signs, and can rarely be deduced from the articulation alone.

Dysarthria also arises from disease of the bulbar nuclei, or of the nerves arising from the latter which supply the muscles of the larynx, pharynx, tongue, and lips. In true *bulbar palsy*, which is a disease depending on a slowly progressive degeneration of these motor nuclei, articulatory defects are often among the earliest symptoms. The diagnosis of these cases is based on the fact that the symptoms begin insidiously and progress gradually, that the paresis affects the muscles of both sides more or less symmetrically, and that it is an atrophic form of paralysis. The atrophy is seen best in the tongue muscles, and is usually associated with a certain amount of fibrillation, and with diminution of their electrical excitability. The palatal reflex is also impaired, and examination of the vocal cords shows that they too are the seat of a progressive palsy. The dysarthria is always accompanied, sooner or later, by some degree of dysphagia, and also by some weakness and atrophy of the muscles of mastication. Atrophic palsy may also be observed in the small muscles of the hands, and there is a tendency to exaggeration of all the tendon reflexes in the limbs. A similar clinical picture may develop in cases of gross disease of the bulb, due either to local softening or hæmorrhage, or in rarer cases to the gradual growth of a tumour in that region. Such cases can be differentiated from true bulbar palsy, partly by the more acute onset of symptoms in the case of the *vascular lesions*, and partly by the asymmetrical distribution of the muscular atrophy and paresis when a *tumour* forms the basis of the disease. *Gummatous meningitis* at the base of the brain may involve the cranial nerves close to their exit from the bulb, and so produce a dysarthria of a somewhat similar character. When tumour or meningitis is the cause of dysarthria, symptoms of increased intracranial pressure are likely to be observed.

Another form of bulbar palsy is seen in cases of *myasthenia gravis*. In this condition there is little or no atrophy of the articulatory muscles, although some thinning of the tongue is observed sometimes. The distinguishing characteristics of this dysarthria are first of all its marked variability, and secondly the effect produced by fatigue. A myasthenic patient may begin a conversation, or may begin to read aloud from a book, without showing much difficulty in his utterance, but, as he progresses, his articulation becomes more and more defective and more difficult to understand. Usually the palate fails quickly, and a nasal quality is given to the voice. If he is asked to repeat the word 'rub' many times, the terminal 'b' becomes an 'm,' and he ends by saying 'rum' instead of 'rub.' Most cases of myasthenia gravis exhibit similar fatigue phenomena in relation to other parts of the musculature (see *Fig.* 111, p. 235), and in particular show varying degrees of ocular palsy, which, like the dysarthria, is at one time more marked than at another, and is much influenced by rest and exercise.

Some articulatory defect is produced by bilateral peripheral palsy of the palate, which most often results from the effects of the *diphtheria poison*. The voice is nasal, and the pronunciation of certain consonants becomes impossible. 'B' becomes 'm,' 'd' becomes 'n,' and 'k' sounds like 'ng.'

Bilateral facial palsy interferes with that part of articulation which depends upon the labial muscles, and so renders speech indistinct, although not unintelligible. Facial palsy of this kind (*Fig.* 201, p. 493) is met with in some cases of peripheral neuritis and also in some cases of myopathy, especially that form to which the name Landouzy-Dejerine is applied.

Unilateral bulbar palsy may exist without much interference with articulation or phonation. There may be considerable palsy of one vocal cord due to a lesion of one recurrent laryngeal nerve, without a recognizable alteration in the character of the voice. A bilateral laryngeal palsy, when complete, leads to aphonia. Similarly, unilateral palsy of the palate or of one-half of the tongue may exist without articulatory defect, especially after the patient has become accustomed to the altered conditions.

Functional Disorders of Speech.—In cases of hysteria, a *functional aphonia* is by no means uncommon, and in many cases can be distinguished from aphonia due to organic disease only by an examination of the larynx. Hysterical aphonia may be complete ; in other cases the voice is reduced to a whisper, and yet the patient is able to adduct the cords properly in coughing. This is sometimes a recurrent malady, and the suddenness of its onset, as well as the suddenness with which it is often cured, are characteristic.

Stammering is another type of functional dysarthria and presents a large variety of forms. There is little difficulty in their recognition, because in all cases when once the articulatory flow is established the utterance is perfectly normal. The difficulty generally arises either in commencing a word or a sentence, or in connection with certain consonants. Some of the cases depend on an initial spasm of the articulatory muscles, and others upon an inco-ordination between the action of the respiratory muscles and those which have to do with phonation and articulation. The complete absence of physical signs of disease, and the history of the case, make the diagnosis easy. On the other hand, stammering is occasionally an early symptom of progressive degenerative conditions of the central nervous system, especially of general paralysis of the insane.

The term '*lalling*' is applied to a defective form of articulation met with chiefly in persons who are more or less mentally feeble. It is characterized by what appears to be an imperfect pronunciation of certain consonants. In more severe cases one consonant is consistently replaced by another, such as 'r' by 'w.' These mistakes in pronunciation are common enough in normal children when learning to speak, but the endurance of the defect after the learning age is passed generally indicates some permanent mental deficiency. A temporary perversion of speech is seen occasionally in children before they learn the proper use of language. They may talk glibly and fluently in a language which they appear to understand themselves but which is unintelligible to their neighbours. In this condition, to which the term '*idioglossia*' is generally given, the prognosis may be regarded as favourable. *E. Farquhar Buzzard.*

SPINAL CURVATURE.—(See CURVATURE, SPINAL, p. 153.)

SPLEEN, ENLARGEMENT OF THE.

The Physical Signs of Enlargement of the Spleen.—If the organ is only slightly or moderately enlarged, there is no alteration in the size or shape of the abdomen; if it is considerably or enormously enlarged, the abdomen may be much distended, and at a first glance this distention may appear to be uniform, as though due to ascites. Closer inspection may show that it is by no means uniform, there being distinct bulging of the left side, especially in the left hypochondrium, the left lumbar, and the left half of the umbilical regions. The inner border of the spleen may be tilted forward in some cases, so that a distinct edge or ridge may be seen pushing the abdominal wall forward, this ridge running downwards and inwards from the left costal margin near the anterior axillary line towards the umbilicus ; in a few cases a distinct notch can be seen in this edge or ridge. When the patient takes a deep breath, the prominence may be seen to move distinctly downwards, though occasionally the spleen may be so enormously enlarged that its lower end becomes impacted in the pelvis, when no downward movement is possible.

Palpation is the best means of detecting splenic enlargement. If the organ is but little enlarged, it may not be felt until the observer, standing upon the left-hand side of the recumbent patient, and supporting the lower left ribs posteriorly with his right hand, steadily but firmly presses the fingers of his left hand under the left costal margin just in front of the anterior axillary line ; when the patient now takes a deep breath, a definite sense of increased resistance may reveal splenic enlargement when the organ is comparatively soft, as in many cases of typhoid fever for example, or a hard mass with a distinct edge may be felt in more obvious cases. When the enlargement is moderate or considerable, the splenic tumour will be felt coming down from beneath the left ribs close behind the abdominal wall; and unless there is a very large liver at the same time, or some other cause preventing the viscus from following its natural direction as it enlarges, it tends to reach and ultimately cross the middle line at or just below the level of the umbilicus. It is generally smooth and firm, and the characteristic notch or notches can be felt in its anterior border. Except in those rare cases in which the whole spleen is dislocated, it will not be possible to insert a hand between it and the left costal margin, or to define its upper limit by palpation. The lower pole can be felt to move decidedly downwards on inspiration, unless the enlargement is very great. On bimanual palpation, the loin is not filled out as it would be by a renal tumour, and the mass cannot be pushed back into the loin so as to be felt by the posterior hand as readily as it is by the hand on the anterior abdominal wall.

Percussion yields a dull note over the mass, the dullness being directly continuous with an increased area of dullness in the thorax extending upwards as high as the seventh rib in the mid-axillary line, the sixth rib in the nipple line, or even higher, and including the ordinary area of splenic impairment of resonance behind. Percussion of the left loin may elicit resonance here, indicating that the colon is not displaced as it would have been by a renal tumour; no intestines can be felt or percussed over the front of the spleen.

Auscultation seldom affords much evidence of value in these cases, but sometimes when the splenic enlargement is associated with local peritonitis, as in cases of infarction for example, a loud rub may be heard over the mass when the patient takes a particularly deep breath; and sometimes, especially if the enlargement is associated with venous engorgement, a well-marked continuous humming bruit may be heard.

Distinction between an Enlarged Spleen and other Tumours which may simulate it.—An enlarged spleen has to be distinguished from other tumours which may arise in the left hypochondriac region, especially from:—(1) Kidney tumours or perinephric inflammation or abscess; (2) Suprarenal tumours; (3) Carcinoma of the splenic flexure of the colon; (4) Pancreatic tumours, especially cyst or carcinoma; (5) Malignant growth of the stomach; (6) Ovarian tumour; (7) Tuberculous peritonitis; (8) Fæcal accumulation in the colon.

Distinction from a Renal Tumour.—It may be difficult to distinguish an enlarged spleen from a kidney in some cases. Both conditions may cause local prominence or bulging of the left side of the abdomen; in the case of splenic enlargement the bulging is more forward and inward, whereas in a kidney enlargement the loin is more likely to be bulged. No distinct edge or notch can be seen or felt in the case of most renal enlargements, a point the significance of which cannot be over-estimated. Either tumour may move downwards when the patient takes a deep breath; but the spleen, being in closer contact with the under surface of the diaphragm, moves the more markedly of the two. A renal tumour being more deeply situated in the abdomen seldom approximates closely to the anterior abdominal wall unless the enlargement is very great, in which case the loin will be filled out and feel very firm and resistant on bimanual examination. A renal tumour generally slopes away as it approaches the ribs, so that it is less difficult to get one's hand between its upper pole and the costal margin than is the case with the undislocated spleen. The colon may be seen or felt over the anterior surface of a renal tumour, which is never the case with splenic enlargement; and percussion may yield a resonant note in front, or in typical cases a vertical band of colonic resonance down the centre of an otherwise dull mass, the loin posteriorly being dull; whereas with a splenic tumour the loin may be resonant, and the anterior aspect of the mass quite dull. The presence of a local bruit or rub would make renal tumour unlikely. The occurrence of HÆMATURIA (p. 275), PYURIA (p. 574), or ALBUMINURIA (p. 4), would suggest renal enlargement, whilst the conditions of the blood might be such as to suggest splenic.

Notwithstanding all these points, to distinguish between splenic and renal masses is sometimes by no means easy; and it is only by paying careful attention to the history and the patient's own sensations, as well as to the physical signs and the changes in the blood and urine, that a correct diagnosis can be made.

Malignant Disease of the Left Suprarenal Gland may cause a large mass which is sometimes particularly difficult to distinguish, either from a splenic or from a renal enlargement. Owing to the close proximity of the suprarenal capsule to the kidney, and the liability for the capsule of the latter to become infiltrated by growth of the former, the physical signs of a suprarenal are practically the same as those of a renal tumour, except that it may be more difficult to pass the hand between the mass and the costal margin. Hæmaturia and other urinary changes may result from spread of the disease to the kidney; affection of one suprarenal gland alone does not produce Addison's disease, and it may be impossible to arrive at a correct diagnosis without laparotomy. A peculiar affection of children deserves special mention: at a comparatively early age there may be an abnormal development of the pubic and axillary hair and of the genital organs (see *Figs.* 174. 175, p. 408), with premature puberty, associated with overgrowth of suprarenal rests in the kidney, the resultant tumour being spoken of as a hypernephroma.

Carcinoma of the Splenic Flexure of the colon is usually annular, giving rise to no definite tumour, but rather to symptoms of chronic, followed by acute, intestinal obstruction. Occasionally, however, the growth may be more voluminous, or it may have caused leakage and inflammatory matting from local perforation through or above the growth, with the result that a fairly large tumour may be felt in and below the left hypochondrium. This mass is generally resonant to percussion, has no well-defined edge or notch, and may vary somewhat in position from day to day: it will usually be associated with intestinal symptoms, especially constipation alternating with diarrhœa, and the passage of mucus, and occasionally blood, per rectum. Sometimes there are obvious secondary deposits in the liver or in the left supraclavicular glands.

Pancreatic Tumours are usually situated more in the median line of the abdomen than is a spleen, between the ensiform cartilage and the umbilicus; sometimes, however, a very large cyst, such as may nearly fill the abdominal cavity, may cause considerable difficulty in the diagnosis. One very important point is that no definite edge and no notch can be felt. The stomach generally lies in front of a pancreatic cyst; or, if the latter pushes its way forward so as to displace the stomach upwards and the transverse colon downwards, it may be possible to define its relationship to the stomach by inflating the latter with gas. A splenic tumour rarely extends to the right of the middle line unless the enlargement is great, and then it crosses at or below the umbilicus, whereas a pancreatic cyst reaches across to the right of the middle line above the navel. Pancreatic new growth has a similar position; but the outline of the mass, if any can be felt at all, is more nodular; there will generally be jaundice and a palpable gall-bladder, and the urine may yield Cammidge's pancreatic reaction (p. 100).

Malignant Growth of the Stomach may be mistaken for enlargement of the spleen, especially gastric sarcoma, which, though very much rarer than carcinoma, is more likely to involve the whole of the stomach and give rise to a very large tumour occupying chiefly the upper part of the left side of the abdomen. The following changes will serve to distinguish a gastric new growth from enlargement of the spleen: the mass is apt to shift its position during the course of an examination or from day to day; it does not present a well-defined edge with definite notch or notches; it may extend a considerable distance to the right of the middle line, although its lower limit may not be below the level of the umbilicus; it is likely to be resonant in front, though the percussion note over it may be impaired; there may be anæmia and leucocytosis, but the blood-changes would not be characteristic of any positive blood disease; the taking of food may cause an increase in the gastric pain; vomiting will generally be a prominent symptom; the vomit may contain blood, obvious or occult; free hydrochloric acid may be deficient or absent; sarcinæ ventriculi may be found (*Fig.* 121, p. 241); and there may be secondary deposits, especially in the liver or in the left supra-clavicular glands. Examination of the stomach with the *x*-rays after a bismuth or barium meal may also assist the diagnosis (*Fig.* 131, p. 270).

Ovarian Tumours have been mistaken for enlargement of the spleen, and vice versa, the differential diagnosis being particularly difficult in cases in which the spleen has become dislocated, or is so large as to reach down as far as the uterus. The organ has sometimes been found so dislocated as to lie wholly within the pelvis. The differential diagnosis depends in most cases on the following points: an ovarian tumour rarely extends upwards to such an extent that its upper limit comes into actual contact with the left costal margin so that the hand cannot be placed between it and the ribs; it does not move much downwards during deep inspiration; it extends upwards from the pelvis, whence it may be felt definitely to arise, the lower part of the abdomen being more prominent than the upper; it is usually more globular than a splenic tumour, and has no sharp, well-defined edge with notches in it, even when covered with projecting bosses of simple or malignant new growth; it usually extends more to the right of the middle line than an enlarged spleen; and it is more apt to transmit aortic pulsations; a vaginal examination may determine that the mass is attached to one or other of the broad ligaments, and that the cervix and the body of the uterus are drawn upwards; there will probably be no distinctive blood-changes, but very likely amenorrhœa.

Tuberculous Peritonitis may cause various abdominal tumours (see p. 48), and sometimes gives rise to a mass occupying the left hypochondriac region, the result of matting together of the intestines, thickening of the omentum, or thickening and infiltration of the peritoneum attached to the abdominal wall here. The tumour does not generally extend close up under the ribs, so that the hand may be placed between it and the costal margin, and although it may feel somewhat rounded, with a more or less well-defined edge, there is no definite notch to be felt; sometimes, however, when there are two, three, or more separate masses united together, a notch may be simulated to some extent. The mass itself may be dull, but there is generally resonance between it and the normal splenic dullness. Ascites is often present, and there may be palpable lumps in other parts of the abdomen, or perhaps redness and œdema of the abdominal wall, or a purulent or fæcal discharge from the umbilicus. Indeed, tuberculous peritonitis is the commonest cause of acquired umbilical fistula, the next commonest cause of the latter being pneumococcal peritonitis which has recovered slowly, either without or with operation. There may be signs of tuberculosis elsewhere, for instance in joints, or lymphatic glands. Calmette's or von Pirquet's tuberculin reactions may be positive. The patient will generally be young, and have consumed unsterilized cow's milk. Pyrexia may be present or absent, either with tuberculous peritonitis or with splenic affections, so that its occurrence does not assist the diagnosis much, except perhaps that if the chart exhibits marked evening pyrexia in a young subject, with a sub-normal temperature in the morning, it is an additional argument in favour of tubercle. The reverse type of pyrexia—morning rise and evening fall—has been spoken of as characteristic of tubercle, but it is seldom met with.

Fæcal Accumulation in the Splenic Flexure or adjacent parts of the transverse or descending colon may be mistaken for an enlarged spleen upon a first examination; but this source of error is usually removed when the patient is re-examined after an action of the bowels has taken place. The condition is found most frequently in women if the age is not great, or in elderly people of either sex. There is generally a history of severe obstipation, and possibly attacks of temporary obstruction. The mass is generally irregular, more or less cylindrical, and in thin persons it may be possible actually to alter its shape by manipulation with the hand. The best test of the condition, however, is the effect of copious enemata upon the mass.

Hæmatoma due to Leakage from an Abdominal Aneurysm is by no means always easily recognized, and it may be mistaken for an enlargement either of the spleen or of the kidney, unless the aneurysm itself can be felt pulsating; or unless there is a history or an acute exacerbation of intra-abdominal pain, accompanied by blanching due to the amount of blood lost.

CAUSES OF SPLENIC ENLARGEMENT.

Having concluded that the spleen is enlarged, the next step is to decide the cause of the enlargement. There are various ways in which the different causes may be classified, but from a diagnostic point of view the following is serviceable:—

I. Chronic Enlargement of the Spleen.

(*a*). *Very great enlargement*:

- Splenomedullary leukæmia
- Lymphatic leukæmia
- Mixed leukæmia
- Chronic malaria
- Kala-azar
- Splenomegalic polycythæmia
- Splenomegalic cirrhosis
- Splenic anæmia
- Pseudo-leukæmia infantum
- Gaucher's disease
- Still's disease
- Familial acholuric jaundice.
- Egyptian splenomegaly

(*b*). *Moderate enlargement.*—All conditions mentioned in group (*a*) will at some stage exhibit a spleen that has not yet become enormous ; and besides these, chronic and moderate enlargement of the spleen may be exhibited in cases of :—

Pernicious anæmia
Rickets
Congenital syphilis
Hodgkin's disease
Cirrhosis of the liver
Lardaceous disease
Thrombosis of the portal vein
Pressure on the portal vein by enlarged lymphatic glands or by adjacent tumour of the gall-bladder, liver, pancreas, stomach, etc.

II. Acute Enlargement of the Spleen, the enlargement as a rule being slight.

(*a*). *Acute infective fevers* ;

Especially—
- Typhoid fever
- Paratyphoid fever
- Relapsing fever
- Typhus fever
- Malaria
- Malta fever
- Erysipelas
- Septicæmia.

Less often in—
- Pneumonia
- Diphtheria
- Scarlet fever
- Small-pox
- Rheumatic fever
- Influenza
- General tuberculosis.

(*b*). *Embolism*, especially in cases of fungating endocarditis.

(*c*). *Injury.*

(*d*). *Strangulation* by twisting of the pedicle.

It will be noted that no mention is made of abscess, gumma, carcinoma (whether primary or secondary), sarcoma (primary or secondary), or hydatid cyst of the spleen, for these are all so exceedingly rare they are very unlikely to be met with. It will also be noted that no mention is made of backward pressure, whether due to chronic valvular disease of the heart with failing compensation, or to obstruction to the inferior vena cava above the hepatic veins, such as may result from thrombosis or from pressure upon the veins by mediastinal fibrosis or new growth ; these conditions are omitted purposely, for it is quite exceptional for ordinary backward pressure to produce enlargement of the spleen. So true is this that in a case of chronic valvular heart-disease with failing compensation the existence of a definitely palpable spleen is evidence of there being more than mere mechanical heart-failure—probably superposed fungating endocarditis. The chief exceptions to this occur in childhood, where the spleen becomes palpable more easily than in adults, so that with heart-failure in a child enlargement of the spleen is less good evidence of fungating endocarditis than it is in a grown-up person.

I. CHRONIC ENLARGEMENT OF THE SPLEEN.

Chronic and very great Enlargement of the Spleen.—When the spleen is so large as to occupy half the abdomen or more, the diagnosis is generally easy. The largest of all spleens are those due to *splenomedullary leukæmia.* The first step is to make a full examination of the blood, including particularly total and differential leucocyte counts. If there is an extreme degree of leucocytosis, up to anything between 50,000 and 1,500,000 per c.mm. for example, the diagnosis is almost certainly leukæmia, and if in the differential leucocyte count there are from 20 per cent to 50 per cent of myelocytes, it is of the splenomedullary type, whilst if the lymphocytes amount to 90 per cent or more, the disease is of the *lymphatic* form, in which the lymphatic glands are almost certain to be enlarged as well as the spleen ; in some cases of lymphatic leukæmia the latter may be scarcely enlarged at all, but in others it may be almost if not quite as large as in the splenomedullary type of the disease. For mixed leukæmia, see ANÆMIA (p. 25). In the absence of any marked leucocytosis, or of characteristic differential leucocyte counts (see also ANÆMIA), the diagnosis of the nature of a very large spleen will depend in the first place upon whether there has or has not been residence in a *malarial* region—the fen districts of Great Britain, the tropics, or certain parts of Europe, particularly Italy. The *ague-cake* spleen of the fens is now very rare ; it is more often found in chronic cases of tropical malaria, when the history may indicate its nature, and if the patient is having febrile attacks, the parasites

(*Plate XXVIII, Figs. A, B, C, D, E*, p. 614) may be found in the blood. Recent investigations have shown that some, at least, of the enlarged spleens formerly attributed to malaria, are due to other infections. One of these has been differentiated clearly from the rest, namely ***Kala-azar***, which occurs in India, particularly in Assam, in Africa, and in Sicily, and is diagnosed chiefly by the discovery of the Leishman-Donovan bodies in the fluid obtained by splenic puncture (*Plate XXVIII, Fig. H*, p. 614).

Splenomegalic polycythæmia is a rare affection of adults, characterized by more or less cyanosis (*Plate XXIX*, p. 634) and symptoms which might suggest a cardiac lesion, together with more or less enlargement of the spleen, and polycythæmia amounting perhaps to six, seven, ten, or even twelve million red corpuscles per c.mm. The malady is generally chronic, extending over years; with an insidious onset and slow progress. The first symptoms are vague, with progressive loss of working power, and some shortness of breath on exertion. Hæmorrhages may occur early, or at any stage of the malady; especially bleeding from the mouth, epistaxis, hæmoptysis, hæmatemesis, hæmaturia, or melæna. Purpura is not common. The patient's heart is generally hypertrophied and dilated to some extent, with or without a systolic apical bruit; and the blood-pressure is above the normal—it may be anything between 150 and 250 mm. Hg. The patient does not usually waste. He may develop effusion into any of the serous cavities. The diagnosis is arrived at when polycythæmia and enlargement of the spleen occur in the absence of any definite cause.

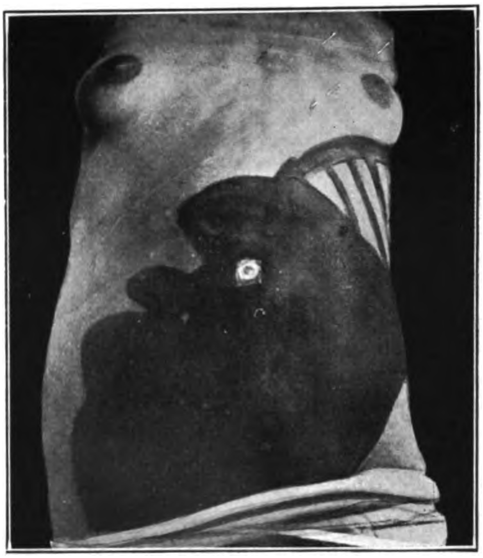


Fig. 267.—Splenic anæmia: photograph showing the outline of the spleen. There was severe chlorotic anæmia without leucocytosis; the patient died, and at the autopsy there was no cirrhosis of the liver.

Splenomegalic cirrhosis is an affection of children and young adults, in whom there are likely to be more or less jaundice, anæmia, lack of development, and ultimately ascites, as well as considerable enlargement of the spleen. There is a tendency for this malady to affect more than one member of a family, and this sometimes gives the clue to the diagnosis. When death ultimately ensues, in addition to the great enlargement of the spleen these cases exhibit more or less fibrosis or cirrhosis of the liver, and sometimes the liver is indistinguishable from that of ordinary alcoholic cirrhosis. What relationship this malady has to ordinary alcoholic cirrhosis of the liver on the one hand, and to splenic anæmia or Banti's disease upon the other, is not clear; but owing to the enlargement of the spleen, it is differentiated as splenomegalic cirrhosis. Hæmorrhages, particularly hæmatemesis, are not infrequent in this as in other forms of cirrhosis of the liver. The blood-changes are merely those of a simple chlorotic anæmia. The diagnosis is afforded chiefly by the age of the patient, by the size of the spleen, and by the absence of any positive blood-changes, particularly if more than one member of the family is affected in the same way. The patient often lives for a number of years, and is able to work in spite of the complaint, until ascites supervenes. The fingers may be clubbed.

Splenic anæmia has been discussed under ANÆMIA (p. 37). The spleen is not as a rule very greatly enlarged, though sometimes it may be enormous (*Fig.* 267). The blood changes are simply those of progressive and severe anæmia of a chlorotic type; even though there may really be a disease meriting the distinctive term, 'splenic anæmia,' not a few cases diagnosed as such on account of the co-existence of splenic enlargement with simple anæmia, ultimately turn out to be cirrhosis of the liver. When that which is really cirrhosis of the liver is diagnosed in its early stages as splenic anæmia, the condition is termed *Banti's disease*. One particular variety of familial splenic anæmia of young persons has been distinguished from the rest as *Gaucher's disease*. It runs a course very similar to that of familial acholuric jaundice (see below), but is distinguished during life by the occurrence of a peculiar fat-like deposit under the exposed part of the conjunctiva external to the cornea; and after death by the presence of special Gaucher cells in the spleen and liver. It is rare.

Pseudo-leukæmia infantum (VON JAKSCH'S DISEASE) was until recently regarded as, apart from true leukæmia, almost the only cause of very great enlargement of the spleen in young children (*Fig.* 13, p. 37). It is diagnosed by the severity of the anæmia, which is of the indeterminate chlorotic type without great leucocytosis, but with all the changes that are to be expected in any severe anæmia (pp. 21, 22) developing in an infant of a year old or less, running a chronic course, but sometimes resulting in complete recovery. It is probable that von Jaksch's disease (splenomegaly and anæmia in a child) is not a disease but a syndrome, and that it includes cases in which these symptoms are due to different causes; in some this is congenital syphilis, as proved by Wassermann's test; others with a negative Wassermann reaction are probably related to the splenic anæmia of adults; others are familial acholuric jaundice; others, Gaucher's disease; while some are due to obscure causes not yet elucidated.

One clinical point of interest which may assist some day in separating these cases into more definite groups is that some show undue fragility of the red corpuscles and others do not; this fragility is tested by putting a drop or two of the patient's blood into successively weaker solutions of salt until the point at which laking occurs is found; the nearer to normal saline this point is, the greater the fragility of the cells. In one group, with normal fragility undue destruction by some toxic agent in the patient is taking place; in another, the fragility of the corpuscles is a main cause of the anæmia.

Still's Disease is really subacute generalized rheumatoid arthritis of children (*Fig.* 169, p. 377) associated with wasting, moderate enlargement of most of the superficial lymphatic glands, and in some cases considerable enlargement of the spleen. The latter is not essential, however, and in any case it is the affection of the joints that attracts chief attention. The patient may be completely crippled.

Familial Acholuric Jaundice is in many respects similar to splenomegalic cirrhosis of children, described above; without laparotomy, or post-mortem examination, it may be impossible to be certain whether the enlarged spleen is accompanied by cirrhosis or not. The disease affects several children in the same family as a rule, runs a chronic course, beginning soon after birth but permitting of survival for many years. The patient might be described as delicate rather than ill, with more or less anæmia of chlorotic type, a facies reminiscent of that of pernicious anæmia, no great wasting, a distinctly icteric tinge of the skin and conjunctivæ, without any bile-staining of the urine, which remains of a normal colour; considerable enlargement of the spleen and often of the liver also: some of these cases give a positive Wassermann reaction, others do not; the congenital syphilitic type is held by some to be quite distinct from true familial acholuric jaundice. Splenectomy has cured a certain number of these cases (*Plate XVIII*, p. 332).

Egyptian Splenomegaly is a disease affecting natives in Egypt, clinically very similar to splenic anæmia, but thought to be due to infection by a protozoon not yet discovered, though perhaps related to either malaria on the one hand or to trypanosomiasis upon the other. It runs a chronic course for some years, but ends like cirrhosis of the liver with ascites, pyrexia, and cachexia. The spleen may be enormous.

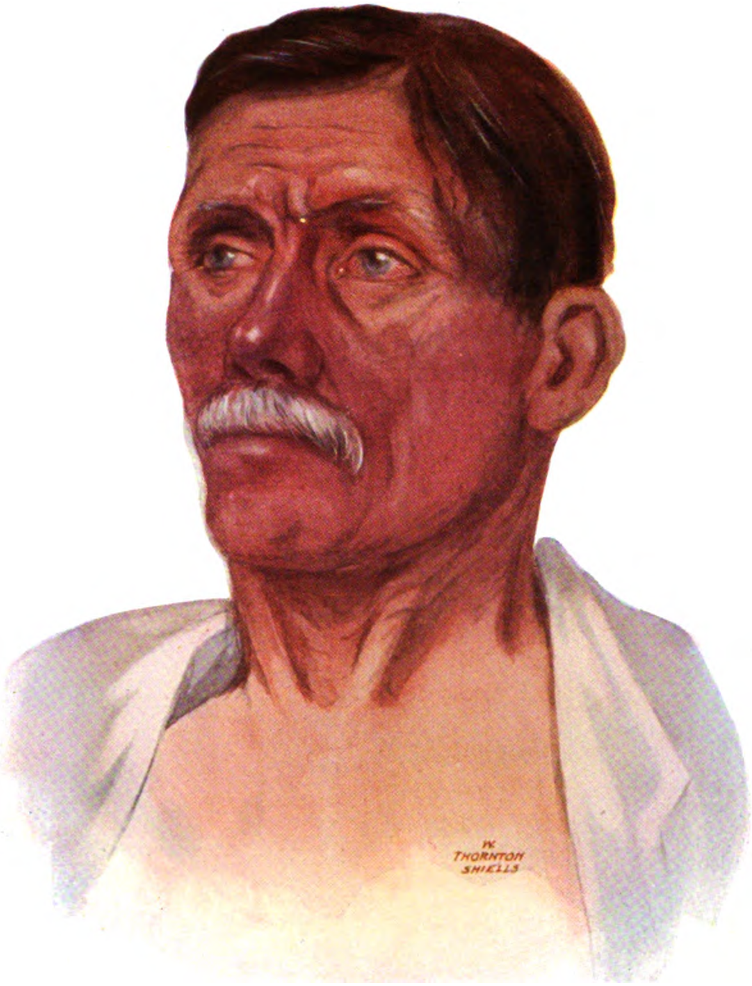
Chronic Enlargement of the Spleen, the enlargement being of moderate size.

It is clear that conditions which may sometimes produce great enlargement of the spleen must go through a phase in which the spleen is not yet enormous, and at this stage all those diseases that have just been discussed will come into the present group. The remarks already made need not be repeated here, however, for the diagnosis at the stage in which the spleen is yet only moderately big is arrived at in the way already described. A blood-count is essential in order to exclude or diagnose leukæmia; parasites may be discovered to account for malaria or kala-azar; and so on. The spleen is palpable in a considerable proportion of cases of *pernicious anæmia*, but it is seldom greatly enlarged, and the diagnosis is arrived at by finding the blood-changes described under ANÆMIA (p. 24). In none of the other diseases mentioned in the list above are the blood-changes themselves pathognomonic.

The spleen of a small child is often just palpable without there being any disease at

PLATE XXIX

SPLENOMEGALIC POLYCYTHÆMIA



The characteristic facies of splenomegalic polycythæmia.

all; if it is more decidedly enlarged, the first suspicion will be that it is due to *rickets* or *congenital syphilis*. The bony changes, quadrate head, beaded ribs, large epiphyses, exaggerated curves of the long bones, particularly of the legs, delay in the closure of the fontanelles, and the pot-belly, will suggest rickets; it should be added, however, that owing to the eversion of the lower ribs along a line corresponding with the attachment of the diaphragm, and known as Harrison's sulcus, the spleen often becomes unduly palpable in rickety children without being necessarily enlarged. Congenital syphilis may be suggested by a knowledge of the family history, by the occurrence of snuffles, of specific skin eruptions, and so forth; but in many cases the diagnosis will be one of surmise only, unless it can be confirmed by the specific serum reaction of Wassermann. *Pseudo-leukæmia infantum* has been discussed above; by some it has been regarded as in some way associated either or with rickets, with congenital syphilis, or both; but the most recent view is that it is due to some cause other than these, of a nature not yet known.

Hodgkin's Disease, when it is typical, is associated with considerable and progressive LYMPHATIC GLAND ENLARGEMENT (p. 376), especially those of the neck, and later those of the axillæ and groins, thorax and abdomen, together with moderate but seldom very great enlargement of the spleen; without any anæmia to begin with, but later with a progressive and ultimately severe anæmia of the chlorotic type, with all the changes mentioned on pp. 21, 22; without leucocytosis, and with nothing characteristic about the differential leucocyte count, except that an occasional basophile cell or myelocyte may be seen. Hodgkin himself laid particular stress upon the changes in the spleen in this disease, but there can be little doubt that there are cases of a precisely similar nature in which there is much lymphatic glandular enlargement without enlargement of the spleen. An attempt is sometimes made to distinguish this type from that with splenic enlargement, by styling it lymphadenoma; but where lymphadenoma ends and Hodgkin's disease begins, and vice versa, is by no means settled. It would seem much more likely that there is every degree of acuteness and severity between extremes that are wide apart, those cases which have lymphatic glandular enlargement and a rapidly fatal ending without leucocytosis as their most prominent feature being styled lymphosarcoma; similar cases with the addition of enlargement of the spleen, but a fairly rapid fatal ending, being termed acute Hodgkin's disease; others again, with enlargement of the glands without enlargement of the spleen and with rather greater duration, being termed lymphadenoma; whilst precisely similar cases with enlargement of both spleen and glands, and a duration of anything between several months and several years, are termed ordinary Hodgkin's disease. One very important point to be realized about this disease is that the blood-changes in it are not pathognomonic even when they are severe.

Cirrhosis of the Liver, by the time it has ended fatally, is nearly always associated with a spleen that is bigger than normal as judged by post-mortem weights. Clinically, however, this enlargement can be made out only in a small proportion of the cases, and even in these the enlargement is seldom great. When, however, there is doubt as to the diagnosis, and cirrhosis of the liver seems to be a possible cause for other symptoms, such as HÆMATEMESIS (p. 265), ASCITES (p. 43), JAUNDICE (p. 324), the presence of chronic but not very great enlargement of the spleen, without affection of the lymphatic glands and without pathognomonic blood-changes, is an additional argument in favour of the diagnosis. On the other hand, splenic enlargement is a very prominent and relatively early feature in a few cases, as in the splenomegalic cirrhosis of children (p. 372) and young adults; whilst in some older patients, long before the hepatic changes themselves attract attention, the case may come under observation for anæmia, with or without hæmorrhage, such as purpura, epistaxis, hæmatemesis, or the passage of blood per rectum; and a considerable enlargement of the spleen may be found. The blood-changes may be merely chlorotic, and in the absence of other definite signs or symptoms the diagnosis of splenic anæmia, that is to say of simple anæmia associated with an enlarged spleen, may be made; many such cases ultimately turn out to be examples of cirrhosis of the liver—Banti's disease.

Lardaceous Disease.—A lardaceous spleen is not always large, but is frequently big enough to be palpable, and the liver is generally enlarged at the same time. The condition results from long-continued suppuration, discharging sinuses from empyema or spinal caries, purulent cavities in phthisis or bronchiectasis, or from tertiary syphilis. If considerable enlargement of the spleen is associated with any of these it is probably due to lardaceous disease. There is generally diarrhœa on account of affection of the intestines; and polyuria and albuminuria owing to renal changes. The patient is weak, frail-looking, and bloodless. Blood-counts exclude leukæmia, and indicate more or less anæmia of the chlorotic type. The disease is very much less common than formerly, because modern surgical methods have done away with much of the persistent suppuration that was formerly common; for the most part it is diagnosed by reason of there being obvious cause for it, especially prolonged sepsis or tertiary syphilis.

Thrombosis of the Portal Vein as a cause for splenic enlargement can seldom be more than guessed at (see Ascites, p. 51).

Pressure on the Portal Vein by enlarged lymphatic glands or by adjacent tumours, will almost certainly be associated with obstruction to the bile-duct at the same time, so that there will be jaundice, and probably also ascites, in addition to any splenic enlargement; the latter will be slight.

II. ACUTE ENLARGEMENT OF THE SPLEEN.

Acute Infectious Fevers.

Typhoid Fever is the best-known febrile disease in which moderate enlargement of the spleen occurs. The organ is usually soft, so that in many cases only an increased sense of resistance is noticed on palpating close under the left ribs. The enlargement may be so slight that the organ may be felt only when the patient takes a deep breath, so as to push it down from under the ribs; or it may be so big that its lower border reaches to the level of the umbilicus. If, in a case of obscure fever in which a continued pyrexia (*Fig.* 241, p. 565) is associated with a relatively slow pulse-rate, the spleen is found to be enlarged, the diagnosis of typhoid fever is very likely; especially if there is a history of gradual onset with anorexia and lassitude, accompanied by headache and sometimes epistaxis, a gradual rise of temperature which, if it has been observed from the first, is seen to go up about two degrees every night, with a fall of one degree the following morning, until step by step it reaches 103° F. or 104° F., or even higher; and perhaps no definite abnormal physical signs whatever except as regards the spleen, or a few rhonchi in the chest. The characteristic rash does not appear until the sixth day or later, when it comes out on the abdomen, sometimes also upon the chest and back, in the form of small, rosy-red, flattened papules which fade on pressure, come out in successive crops, and are seldom present to the extent of more than half a dozen or a dozen at a time. Widal's agglutinating serum reaction should ultimately be positive in a dilution of 1 in 200 in half an hour, but it is generally the second week before this test is positive. Earlier confirmation of the nature of the fever may be obtained by the blood-count, there being no leucocytosis—indeed, sometimes Leucopenia (p. 361)—whilst, unlike many febrile illnesses, typhoid fever produces a relative increase, not in the polymorphonuclear cells, but in the small lymphocytes. Such blood-changes are in themselves almost pathognomonic, and they are obtainable before Widal's reaction is to be expected, though the latter is the ultimate test of the fever. Typhoid bacilli may be recovered from the blood on special cultivation quite early in the attack, but this method of diagnosis is not resorted to often. When neither blood-count nor serum-test is possible, the diagnosis may not be cleared up until the third week or later, when sloughs from Peyer's patches can be discovered in the stools. The ratio of the pulse-rate and temperature is of considerable value in the diagnosis, for in most cases the pulse-ratio is very low; for instance, with a temperature of 104° F., the pulse-rate may be only 90 or 100 per minute, when the physiological ratio for this temperature is 120. Pneumonia in its earlier stages may also produce a low pulse-ratio; but the respiration-ratio is here increased, which is not the case in typhoid fever. The following figures illustrate these points:—

	T.	P.	R.
Physiological ratio	104° F.	125	32
Typhoid fever	104° F.	90	30
Pneumonia	104° F.	100	40

General tuberculosis may simulate typhoid fever in this respect also, and sometimes it is not possible to decide between the two until the case has been watched for some time.

Paratyphoid Fever is closely related to typhoid fever, and the clinical symptoms are very similar; the importance of distinguishing between the two lies chiefly in the carrying out of Widal's agglutinating serum reaction. It sometimes happens that in a case which, from a clinical point of view, is almost certainly typhoid fever, the serum will not cause clumping of Eberth's typhoid bacilli; and so far as the bacteriological test goes, the diagnosis might remain altogether obscure unless the serum were tested also against the *Bacillus paratyphosus A* and the *B. paratyphosus B.* In a certain proportion of cases clumping will be obtained with one or other of these, the diagnosis of paratyphoid fever being based upon bacteriological rather than upon clinical conditions. The spleen is enlarged in paratyphoid fever to about the same extent as in typhoid.

Relapsing Fever is associated with considerable enlargement of the spleen. The disease is contagious, but nowadays rare, developing only under conditions of filth and famine. It is characterized by an acute onset, with chills, pains in the back, and a sudden rise of temperature. The latter remains high for six or seven days, and then falls by crisis. For about a week the temperature remains normal, and then it rises again as before, several such remissions and relapses succeeding each other and being pathognomonic of the disease (*Fig.* 5, p. 27). The pulse is rapid, and there is profuse sweating. Enlargement of the spleen is detected early. It is most conclusively distinguished from other diseases by examination of blood-films in which the *Spirochæta obermeieri* (*Plate XXVIII, Fig. I*, p. 614), will be found.

Malta Fever is discussed on p. 565; the splenic enlargement is similar to that of typhoid fever.

Malaria.—Apart from the chronic enlargement of the spleen due to recurrent attacks of malaria, the spleen becomes enlarged and soft as the result of active hyperæmia during acute attacks. Even when no splenic enlargement can be detected in the intervals, during the paroxysms the viscus can usually be felt projecting below the costal margin, presenting a soft and indefinite lower border. When the patient has more or less chronic enlargement of the spleen as the result of preceding attacks, each acute febrile paroxysm is associated as a rule with an additional swelling which passes off after the attack. For the characters of the fever, see pp. 29–32. The nature of the malady will be suggested by geographical considerations, or by the influence of quinine; but the only conclusive proof is the discovery in stained blood-films of the malaria parasites (*Plate XXVIII, Figs. A, B, C, D*, p. 614). There is often marked anæmia, especially in cases of recurrent malaria, the red corpuscles and hæmoglobin becoming reduced as in chlorosis; the leucocytes are also diminished, and the differential leucocyte count shows a relative increase in the large hyaline lymphocytes up to even 15 or 20 per cent.

Erysipelas is often associated with moderate enlargement of the spleen; but the fever, rigors (*Fig.* 245, p. 568), and slightly-raised red spreading infection of the skin are sufficiently characteristic to indicate the diagnosis.

Septicæmia may be less easy to diagnose unless there is some obvious source of sepsis in the first instance, such as infection of the uterus after childbirth, sepsis in connection with the general peritoneal cavity, joints, wounds, and so forth. The chief difficulty arises in those cases in which the source of the sepsis is not obvious, being due to absorption from such lesions as pyorrhœa alveolaris, whitlows, acne, or other comparatively small superficial affections; or to deep-seated suppuration, such as a hidden empyema, infective pylephlebitis, infective cholangitis, pyosalpinx, and so forth. In some cases of chronic or subacute septicæmia, enlargement of the spleen may be considerable, and the diagnosis of infective endocarditis will very likely suggest itself. Whether or not the heart valves are affected in these cases, the ultimate diagnosis will depend upon discovery of infective organisms in cultures obtained by venepuncture.

Diphtheria, Pneumonia, Scarlet Fever, and *Small-pox* seldom give rise to very prominent splenic enlargement, and the only importance of it is that in the early stages of the malady detection of a spleen that is just palpable may temporarily arouse a suspicion that the patient may be suffering from typhoid fever. The course of the disease, bacteriological examination of swabbings from the throat, the physical signs in the lungs, and characters of the sputum and the skin rash, will serve to point to the correct diagnosis.

Typhus Fever is fortunately very rare now, although there are small outbreaks of it in the poorer parts of large cities from time to time; the spleen becomes soft and moderately enlarged, but less constantly so than in typhoid fever. The disease sets in more acutely than enteric, with chills, early prostration, and a high temperature which ends by less

marked lysis (*Fig.* 268) than does that of typhoid fever (*Fig.* 241, p. 565); and sometimes almost by crisis at the end of the second week (*Fig.* 269). The rash differs from that of typhoid fever, in that it appears on the fifth day, and consists of petechiæ and of dark-red groups of subcutaneous macules in addition to rosy-red papules on the surface. Nervous symptoms become very marked, especially at the end of the first week, the so-called typhoid state being an expression used to denote, not the condition that occurs in typhoid fever, but that which develops in typhus. There may be severe vomiting, and retention of urine, important symptoms that are rare in typhoid fever. There should be no positive Widal's reaction, and no sloughs in the stools.

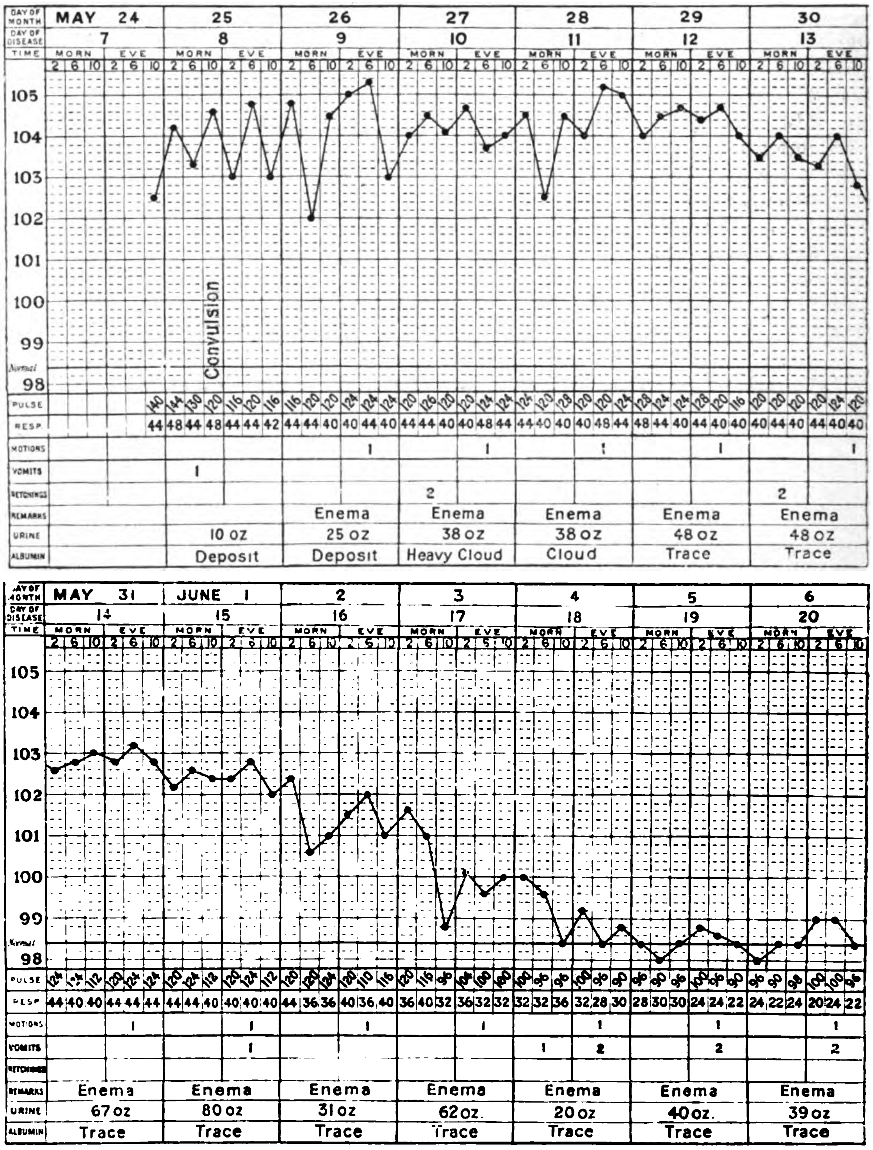


Fig. 268.—Temperature chart from a typical case of typhus fever ending by lysis rather than by crisis.
(*Chart kindly supplied by Dr. Turner, Med. Supt. of the South Eastern Fever Hosp., London.*)

Influenza **is a diagnosis which should never be made except with very good cause, for many febrile illnesses in which the real cause escapes recognition receive the label influenza.**

It is easiest to diagnose correctly in times of severe epidemic, and then slight enlargement of the spleen may occur in a few cases. This in itself is not important if influenza can be diagnosed with certainty on other grounds; but until the nature of the fever becomes obvious, it is important in that it may suggest typhoid when none exists. The sudden onset, extreme prostration, high pulse-rate as well as temperature, initial chill, profuse sweating which comes on when the patient begins to improve, and the fall of the temperature after an illness lasting from twenty-four hours to three or four days or a week, would all point to influenza. It may, however, be impossible to distinguish influenza from

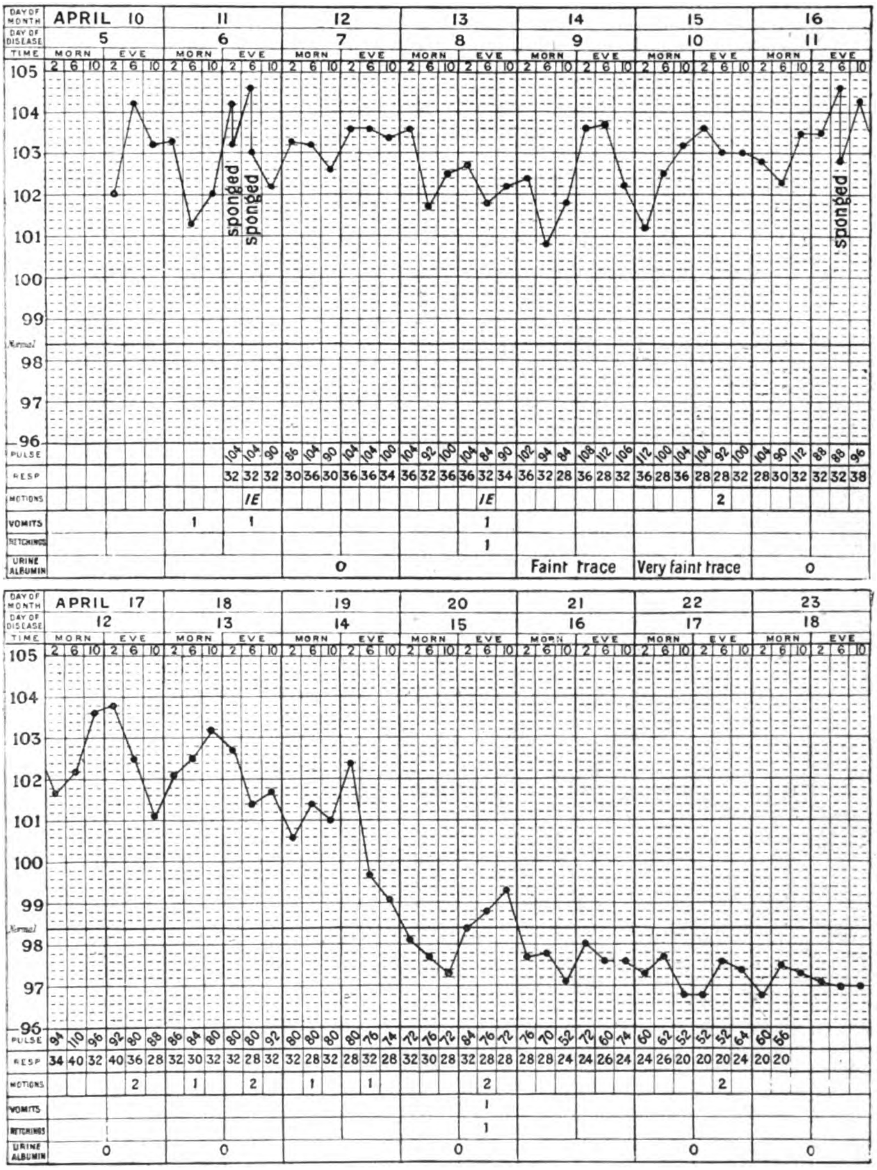


Fig. 269.—Case of typhus fever, showing termination by crisis at the end of the second week. The majority of the cases exhibit a less abrupt ending to the pyrexia perhaps, but the above type is characteristic in some epidemics. (*Chart kindly supplied by Dr. Turner, Med. Supt. of the South Eastern Fever Hospital, London.*)

typhoid fever until the course of the pyrexia has been watched, or unless typical *Bacilli influenzæ* are recovered in nearly pure culture from some secretion, such as the sputum. It is worthy of note that in influenza as well as typhoid there is no leucocytosis.

General Tuberculosis may also simulate typhoid fever in certain cases, and enlargement of the spleen may result from the development of tubercles in it. When cerebral symptoms predominate, the diagnosis is relatively easy ; the headache may be equally severe in both, but with tuberculous meningitis there is more vomiting and more retraction of the head, whilst it is probable that optic neuritis, and perhaps choroidal tubercles, can be detected. Widal's test will remain persistently negative ; there will be no rosy rash, probably no leucocytosis, and no sloughs will be found in the stools. In some cases, however, general tuberculosis produces a clinical picture that may be very difficult to distinguish from typhoid fever. Lumbar puncture may decide the diagnosis.

Embolism.—Fungating endocarditis is nearly always associated with palpable enlargement of the spleen, and sometimes the organ attains a considerable size (*Fig.* 270). As stated above, ordinary heart-disease with failure of compensation does not give rise to splenic enlargement that can be recognized clinically, except perhaps in children, notwithstanding the fact that one might have expected the backward pressure to cause the spleen to be big by being dilated with blood. Except in children, enlargement of the spleen in a heart case should always arouse serious suspicion of infective endocarditis. The enlargement may be due to embolism and infarction, in which case there may have been a history of acute pain low down on the left side of the chest, accompanied by a definite rub due to perisplenitis over the infarct. The splenic enlargement in some cases, however, is due less to actual infarction than to the general toxæmia ; even when there has been an infarct it is not always easy to be sure of it. Fungating endocarditis sometimes develops without there being any bruit at all ; the diagnosis is then exceedingly difficult unless the patient suffers from multiple emboli—cerebral, renal, intestinal, splenic, peripheral. Sometimes such an embolus may be followed by the development of an acute aneurysm—femoral, popliteal, cerebral and so forth. A cerebral embolism of this kind has sometimes resulted in sudden transient coma and hemiplegia ; the patient has seemed to be recovering ; then in a day or two has relapsed into coma again, and died, the cause of the relapse and fatal ending being the development of an acute cerebral aneurysm at the site of the embolus, rupture of this aneurysm, and death from the resultant hæmorrhage. Progressive anæmia of the chlorotic type, without much leucocytosis, is another feature of these cases. The diagnosis must always be difficult when there is no cardiac bruit ; when there is a bruit, the difficulty is to determine whether the patient is suffering merely from mechanical heart-failure, or from fungating endocarditis superposed upon the chronic heart lesions (p. 34).

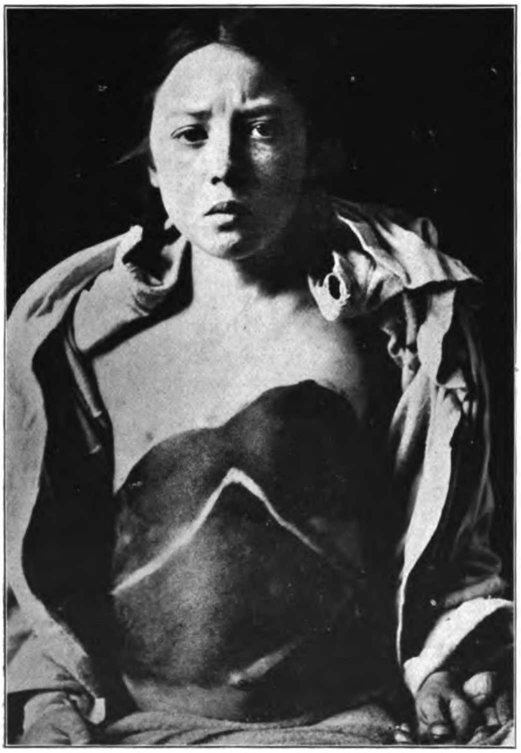


Fig. 270.—Photograph showing great enlargement of both the spleen and liver in a case of fungating endocarditis (the same patient as in *Fig.* 236, p. 555, illustrating purpura). Note also clubbing of the thumb and fingers just discernible in the left hand. The diagnosis was verified at autopsy.

Thrombotic infarction may cause acute splenic enlargement in almost any of the blood diseases, particularly in lymphadenoma and leukæmia.

Neither *Injury* nor *Strangulation of the spleen by its becoming twisted upon its own hilum* is a very common event, and the latter is nearly always the result of injury. A blow in the splenic region may cause a rupture in the pulp of the spleen without bursting its capsule, and without obviously injuring the chest wall or abdomen. The bleeding that occurs within the capsule of the spleen itself causes great pain in the part and enlargement of the organ ; the diagnosis can seldom be more than guessed at unless laparotomy is performed. Strangulation of the spleen seldom occurs if the organ is in its natural position ; but when there has been previous dislocation, an abdominal injury, or sometimes a sudden spontaneous effort, has led to its becoming twisted on its own hilum, the symptoms being such as to suggest an acute intra-abdominal condition requiring immediate laparotomy, but seldom pointing to the actual diagnosis until the laparotomy has been performed.

Herbert French.

SPONGY GUMS.—(See BLEEDING GUMS, p. 72.)

SPUTA vary enormously as to their amount, consistence, colour, and so forth ; but by far the most important point about them in diagnosis is the determination of whether they contain tubercle bacilli or not. There is no particular variety of sputum which can be said to be characteristic of pulmonary tuberculosis, although stress is generally laid upon the fact that phthisis with cavitation produces a nummular sputum—that is to say, sputum of which the individual portions expectorated tend, not to coalesce but to flatten out as separate round portions, if they are spat on to a flat, dry surface ; if expectorated into antiseptic fluid, they remain as more or less globular, separate masses. As a matter of fact, however, ordinary bronchitis may produce sputum possessing a typical appearance of nummularity, and it is most unwise to rely on the naked-eye appearances of sputum for any diagnosis except that of lobar pneumonia, when it may be typically viscid and rusty. It is in almost all cases essential to make films of the sputum, and to stain these for tubercle bacilli by the Ziehl-Neelsen method with carbol-fuchsin.

The carbol-fuchsin solution is made up of 1 gm. of fuchsin, 10 c.c. of absolute alcohol, and 100 c.c. of 5 per cent solution of carbolic acid in distilled water. The slide is covered by the stain in a suitable receiver, and held over a small Bunsen burner or spirit flame until the fluid steams briskly but does not actually boil. After immersion in this for five minutes at least, and it does not much matter if for longer, the excess of stain is poured off, the film washed in water, the excess of the latter drained off, and the slide immersed in 25 per cent sulphuric acid for about half a minute ; it is then transferred to water again, and recovers more or less of the red tint of the fuchsin ; if too little of this has been discharged, the slide is returned to the sulphuric acid for another period, and so on ; when well decolorized, only the thickest parts of the film retain obvious red ; it is then counterstained by five minutes' immersion in carbol-methylene blue, the excess of this stain being washed off with water, the film dried in the air, and either mounted in Canada balsam or else examined directly through cedar-wood oil ; the tubercle bacilli (*Plate XXVIII. Fig. K*, p. 614) show up as bright-red rods in a blue field under the oil-immersion lens.

The presence of acid-fast bacilli in an ordinary sputum film is very nearly proof positive of tuberculosis of the lung, the only source of fallacy being the possibility of non-pathogenic acid-fast bacilli being derived from the mouth. It is very unlikely that this source of fallacy will persist from day to day, especially if care be taken to make the films from the interior of the sputum pellets. It should be remembered, of course, that the absence of tubercle bacilli, or rather their non-detection, is no proof of the absence of pulmonary phthisis, and if there is doubt, successive sputa should be tested in the same way. It should also be remembered that a lesion which may have been tuberculous originally, may in time lose its tuberculous character, the tubercle bacilli may die out, though the cavities produced by them still persist and become occupied by pyogenic organisms and their products. Many of the symptoms of phthisis itself are not due to tubercle bacilli directly, but rather to secondary infection by streptococci, staphylococci, pneumococci, micrococci catarrhales, and so forth ; and the degree of this secondary infection may be gauged from the films at the same time as one looks for tubercle bacilli.

It is also important to realize that a person may expectorate sputum containing tubercle bacilli in abundance every day for months, and yet may have precisely as much lung tissue left by the end of that time as at the beginning ; this is due to the fact that when cavities have been produced they are lined by granulation tissue, and it is possible for the discharge from the surface of these granulations to produce a continuous supply of sputum without the erosion of the lung tissue progressing. One sees a precisely similar state of affairs in cases of some skin ulcers, which may discharge abundantly and yet remain much the same size for months. The best evidence of lung destruction is afforded by the discovery of elastic fibres (*Fig.* 271) in the sputum ; if these are present there must be something which is eroding the lung tissue, and if tubercle bacilli are present at the same time, the two together indicate advancing phthisis. The elastic fibres may be obvious when ordinary sputum is examined fresh after it has been pressed out between cover-slip and slide, but oftener they are more easily detected when a quantity of sputum has been boiled with strong caustic soda to destroy pus cells, mucus, and so forth, leaving the very resistant elastic fibres unaffected. Tubercle bacilli are also very resistant to the effect of strong alkali, and when they are suspected to be present but cannot be found without in some way concentrating them, it is a useful plan to boil the sputum with an equal amount of 5 per cent caustic potash, or with antiformin, to dilute the result with water, to centrifugalize it well, and make films from the deposit. There are various other methods of obtaining concentrated bacilli from the sputum, but this is one of the simplest. It should be borne in mind that tubercle bacilli may be found even when the sputum is exceedingly small in amount and apparently insignificant and mucoid.

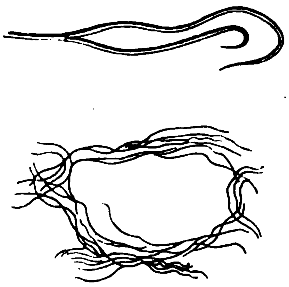


Fig. 271.—Elastic fibres from sputum. The lower figure is less magnified than the upper, and shows alveolar arrangement of the fibres. (From French's *Medical Laboratory Methods.*)

For sputa containing blood, *see* HÆMOPTYSIS (p. 285).

Viscid, Rusty Sputum is almost pathognomonic of pneumonia. As a rule the diagnosis of lobar pneumonia is fairly clear owing to the sudden onset of an acute pulmonary complaint associated with fine crepitations confined to one or more lobes, followed by dullness, with bronchial breathing, bronchophony, and pectoriloquy without râles ; these being succeeded by redux crepitations, with a diminution in the bronchophony, pectoriloquy, and bronchial breathing until normal voice and breath sounds are restored. Herpes labialis is common. The patient's temperature, after maintaining a high level such as 103° F. or 104° F. for from five to ten or more days—usually about seven—falls by crisis (*Fig.* 272). The respiration rate is very rapid—for example, 40 per minute—during the height of the fever, and the skin is flushed, dry, pungent before the crisis, moist from profuse perspiration after it. The diagnosis is much more difficult in some cases, however ; there are not a few patients in whom the consolidation is deep-seated, so that it does not come to the surface at all, and lobar pneumonia has to be diagnosed when there are no abnormal physical signs to be detected in either side of the chest. In such cases the general symptoms may suggest the diagnosis, and the sticky, viscid sputum, the colour of which is generally that of iron rust—but which may be any of the colours that a bruise may have, from bright-red or brown to greenish-brown, greenish,

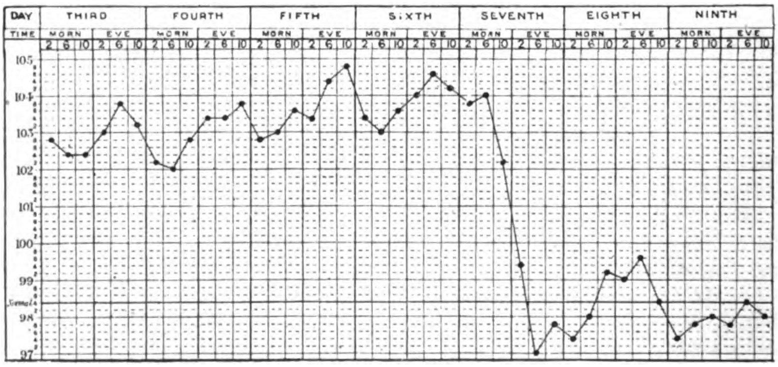


Fig. 272.—Temperature chart of an ordinary case of lobar pneumonia, showing a crisis on the seventh day of the illness, and a slight post-critical rise on the following day.

yellowish, or even bluish-yellow—confirms it even when the lung signs remain normal. The viscidity of the sputum in these cases is of as much importance as the colour. Films of it usually contain numbers of pneumococci (*Plate XXVIII, Fig. O*, p. 614); in exceptional instances pneumo-bacilli. When lobar pneumonia is due to the influenza bacillus, the sputum has not the viscid, rusty character as a rule, but is more like that of ordinary mucopurulent bronchitis. The presence of large numbers of pneumococci, however, or of any other micro-organism than the tubercle bacillus, is by itself no proof that these are the cause of the lung lesion, for even in the sputum of perfectly normal persons pneumococci and other bacteria are frequently abundant. It is quite possible for a patient who is dying of general miliary tuberculosis of the lung to have no tubercle bacilli in the sputum, but an abundance of capsulated pneumococci which may readily, when they are discovered, lead to an erroneous diagnosis.

Influenza bacilli are exceedingly small; but it is important that they should be looked for, both directly and by cultural methods, in all cases thought to be influenzal, before this diagnosis is regarded as established: even when influenza bacilli are found there is still the possibility that they may be an intercurrent infection in some other malady; but it is so tempting to think of influenza when no other obvious cause for a febrile illness can be discovered, that it should not be diagnosed until influenza bacilli have been shown to be present.

Abundance of Foul Sputum, especially when expectorated much at a time at comparatively long intervals, is sometimes by itself a striking symptom, and it suggests that the patient is suffering from one or other of the following:—

Bronchiectasis
Phthisis with cavitation
Fœtid bronchitis
An empyema ruptured into the lung
Gangrene of the lung.

It is sometimes very easy to distinguish between these; with *fibroid lung and bronchiectasis* the patient is likely to have had symptoms periodically for a long while; there will generally be CLUBBED FINGERS (p. 111); the abnormal physical signs are confined to one lung as a rule, and especially to the lower lobe, with displacement of the heart towards that side; there will be deficiency of movement, resonance, and vesicular murmur in the affected lower lobe, together with either absence of voice sounds and of râles, or scattered foci of crackling râles, especially when the patient coughs, with bronchophony, pectoriloquy, and bronchial breathing. If, on the other hand, the abundant and foul sputum is associated with abnormal physical signs in both lungs, and if the upper lobes are obviously more affected than the lower, if the patient has a strong tuberculous family history, and if tubercle bacilli are either now present in the sputum, or are known to have been present formerly,—the diagnosis of *chronic phthisis with extensive cavitation and secondary infection* of the cavities with pyogenic organisms is obvious.

Fœtid bronchitis is always a dangerous diagnosis to make, and the probability is that many cases so diagnosed have been examples either of deep-seated bronchiectasis, of phthisis with cavitation and secondary pyococcic infection, or of *empyema ruptured into the lung*. The latter is generally associated with hardly any abnormal physical signs, because if the original empyema had given rise to the ordinary signs it would have been diagnosed and relieved by operation; an empyema may develop either between the lobes, or between the pericardium and the lung, or between the diaphragm and the lung, in such a way as to leave normal lung tissue all round the surface next the chest wall, so that the usual evidence of pus in the chest is entirely wanting. Even if abnormal physical signs are produced when the pus is kept in an abnormal position in this way, the needle may have to pass through so much tissue before the empyema cavity is entered, that the pus cannot be located: in either of these cases the empyema will, in the course of time, tend to ulcerate its way through the pleura and lead to the expectoration of large quantities of foul sputum at intervals as the empyema cavity re-fills. The diagnosis depends largely upon the exclusion of other causes of abundant foul expectoration, and perhaps upon the history of a preceding illness predisposing to empyema, for example lobar pneumonia.

Gangrene of the lung may be simulated to some extent by bronchiectasis or by empyema rupturing into the lung; but generally speaking nothing but gangrene will produce so much stench. Foul though the sputum in bad bronchiectasis may become, it seldom approaches the awful fœtor of pulmonary gangrene. The history, moreover, is acute; there may be some obvious cause for gangrene, particularly lobar pneumonia in a patient debilitated from some other cause, such as diabetes, or inhalation of foul particles after immersion in a dirty river, or as the result of disease of the mouth, throat, or œsophagus, or septic embolism of the lung from lateral sinus thrombosis. If any doubt remains as to whether lung tissue is being destroyed or not, elastic fibres can be sought for, their presence at once distinguishing between bronchiectasis or deep-seated empyema on the one hand and gangrene on the other.

When a large quantity of pus is expectorated through the lung in a person who, having been in the tropics and having possibly suffered from dysentery, has since had symptoms pointing to hepatic trouble, the possibility that an amœbic abscess of the liver may have opened its way through the diaphragm into the lung will immediately occur to one, especially if the expectorated pus is tinged the colour of anchovy sauce. It might be thought that the *Amœba coli* would be found in it; but this is not the case, for this protozoon is not present in the pus of a hepatic abscess as a rule, but only in the granulations of the abscess wall. The sputum in these cases is not generally foul.

The other abnormal features that may be exhibited by sputum are relatively uncommon, and are of diagnostic significance only in exceptional cases. The serous, mucoid, muco-purulent, or purulent sputum of the various stages of acute and chronic *bronchitis* may arouse a doubt as to whether the patient has not a tuberculous focus; repeated examination will fail to reveal either tubercle bacilli or elastic fibres, but it is to be remembered that a considerable minority of phthisical subjects seem not to expectorate the bacilli. *Black sputum* is common in those who live in smoky atmospheres, particularly in towns, colliery districts, and manufacturing centres. Other changes in colour may be due to hæmoptysis, pneumonia, or hepatic abscess, which are all discussed above; sometimes infection by the *Bacillus pyocyaneus* may produce greenish or bluish sputa which may alarm the patient, but which need not have any serious import, and red sputum simulating the hæmoptysis of phthisis may be due to infection by pigment-producing bacilli such as the *Bacillus prodigiosus*; phthisis may be diagnosed wrongly in such a case unless the most careful investigations of the sputa are made by cultural methods.

Curschmann's spirals (p. 153) and *Charcot-Leyden crystals* (p. 102) have been discussed elsewhere.

Casts of the bronchial tubes are met with in very exceptional cases, and they are of two main types—namely, diphtheritic, and non-diphtheritic. The distinction depends on bacteriological examination; histologically they consist of ill-defined exudate containing cells irregularly embedded in it. Non-diphtheritic casts are due to plastic or fibrinous bronchitis, a very rare disease of which the sputum is the diagnostic point. Two other rare causes for the expectoration of casts of the bronchi are lobar pneumonia, and the inhalation of blood from some other part of the lung in a case of hæmoptysis, and its subsequent expectoration after it has clotted.

Fig. 273.—Calcareous concretions from phthisical sputum.

Now and again a cretaceous pellet (*Fig.* 273) or a small caseous mass may be found in the sputum of a patient who either has pulmonary phthisis, as evidenced by the abnormal apical physical signs, and by the detection of elastic fibres as well as tubercle bacilli in the sputum, or in children as the result of the ulceration of a caseous bronchial gland into the trachea or a main bronchus, and then expectoration of its caseous or cretaceous contents.

Another rarity which has occasionally been found in the sputum is a recognizable particle of new growth, the detection of which may be of material assistance in diagnosis.

Chemical analyses are relied on by some observers in distinguishing tuberculous from non-tuberculous sputum, it being stated that expectoration containing coagulable proteid is more likely to be the result of tuberculous infection than is sputum which does not coagulate with heat. This distinction, however, is not universally accepted, and microscopical examination for tubercle bacilli is certainly a more reliable test in the great majority of cases.

The rarer bacteria and moulds that may be detected in the sputum by special bacteriological methods generally require very special investigation, including cultural tests by skilled bacteriologists; one need not, therefore, enter into details here, though it may be well to enumerate certain micro-organisms which may be pathogenic in the lung in comparatively rare instances—*B. mallei*, generally amongst workers in stables or otherwise in connection with horses; *Aspergillus flavus*, *A. niger*, *A. fumigatus*, generally amongst those who have to do with the artificial feeding of pigeons and other birds; *Actinomyces* or the ray fungus, in those who have had to do with barley in some way or another, or, as has recently been demonstrated, in those who are in the habit of holding cotton in their mouths, such as tailors and seamstresses. Besides these pathogenic micro-organisms, not a few others which are not actually pathogenic are to be recognized in the sputum when it has become secondarily infected in chronic cases. *Penicillium glaucum*, for instance, or *Oidium albicans*; yeast and other moulds; *Micrococcus tetragenus*; or *Oidium tropicale*, a micro-organism similar to but culturally different from *Oidium albicans*, which has recently been reported to be a cause of lung lesions both in Europeans and natives in Ceylon, the symptoms suggesting phthisis, but the latter being excluded by the persistent absence of tubercle bacilli from the sputum and by the absence of reaction to tuberculin.

Whooping-cough is sometimes difficult to distinguish from other colds and from bronchitis, and if the recent statements to the effect that it is due to the minute Bordet-Gengou bacillus prove true, bacteriological examination of the sputum may be of use in diagnosing this condition in doubtful cases; the patient's blood-serum may also give a positive clumping reaction with cultures of this organism.

The lung fluke, *Paragonimus Westermani*, which causes hæmoptysis in Korea, Japan, and parts of China, is to be diagnosed by the discovery of its oval, capsulated eggs in the sputum.

Herbert French.

SQUINT.—(See STRABISMUS, p. 649; and DIPLOPIA, p. 174.)

STAMMERING.—(See SPEECH, ABNORMALITIES OF, p. 623.)

STERILITY.—The differential diagnosis of the causes of sterility is often difficult, and although there are many well-defined conditions which give rise to it, there are numbers of cases in which no definite cause can be found. Further, we must not overlook the fact that the husband is responsible for a sterile marriage in one-fourth to one-third of the cases. This is a fact shown by many observers, and too often forgotten when investigating cases. Therefore, we must not consider a case to be complete unless the husband *and his semen* have been investigated. Many a woman has her married life made miserable, and is taken from doctor to doctor on account of sterility, when the husband really is to blame.

The causes of sterility are shown in the table on the next page. From a study of it, it is clear that some of the causes of sterility are primary, whilst others are secondary. Thus absence of the uterus or infantile uterus means primary sterility, whilst hyperinvolution, carcinoma of the cervix, etc., may occur in women who have had children, and only secondarily become sterile on account of these lesions. Further, some of these causes are common, or may be remedied; others, on the other hand, are rare or absolutely incurable. Diagnosis is therefore of great importance, for it is far better to discover and remedy a defect early in married life, than to wait until the best years are soured and embittered by the longing for a child. Unfortunately, many patients, from various motives, put off the investigation too long.

Congenital Lesions.—Some of the congenital lesions are diagnosed easily, such as *closure of the hymen*, *absence of the vagina*, or *closure of the cervix*, whilst absence of the essential organs often requires an anæsthetic in order that a bimanual examination may be made satisfactorily. The *infantile uterus* and *small adult type* are very difficult to differentiate; but in the former the body forms only one-third of the total length of the organ, whilst in the latter it forms two-thirds, both types of uterus being small in the antero-posterior and lateral dimensions, and only slightly shortened in the vertical. Of all the congenital lesions, the '*cochleate*' *uterus* of Pozzi is the commonest cause of sterility, and is the most hopeful as regards treatment. The uterus is felt to be unusually curved anteriorly, has a long conical cervix, and a small external os. In such an externally malformed uterus as this it is possible that the internal structure is abnormal; the endometrium, perhaps, is unduly thin and fibrous, the muscle layers badly developed. The hypothetical *change in the endometrium* may be the essential cause of sterility: hence curettage for its complete removal forms an essential part of the treatment. The peculiar shape and curvature have long been considered a possible cause, preventing the entrance of spermatozoa into the cavity. *Pseudo-hermaphroditism* usually shows itself by shortness of the vagina, elongation of the clitoris, and the presence of glandular masses in the groins, which are almost always testes, proving that the subjects of it are really undeveloped males.

LESIONS OF THE GENERATIVE ORGANS.		GENERAL CONDITIONS.
Congenital Lesions—	*Acquired Lesions—*	
Absence of uterus, tubes, ovaries	Dyspareunia	Old age
Closure of hymen, vagina, cervix	Vaginismus	Obesity
Pseudo-hermaphroditism	Salpingo-oöphoritis	Anæmia
Infantile uterus	Uterine fibroids	Nutritional disturbances
Small adult type of uterus	Endometritis	Incompatibility
'Cochleate' uterus	Cervical catarrh	Absence of sexual feeling.
Displacements	Polypi	
Hypothetical changes in the endometrium.	Carcinoma of cervix	
	Carcinoma of fundus	
	Ovarian tumours	
	Acquired atresia of vagina	
	,, ,, cervix	
	Genital fistulæ	
	Hyperinvolution	
	Astringent douches	
	Deficient ovarian activity.	
	In the male.	
	Azoöspermia	
	Oligospermia	
	Necrospermia.	

Acquired Lesions.—The differential diagnosis of the acquired lesions can only be made by complete examination of the patient by inspection, bimanual examination, and the use of the microscope to elucidate doubtful growths. *Dyspareunia* as a cause is dealt with in the article under this head (p. 193). *Hyperinvolution* is diagnosed easily; it occurs always after a labour, and strictly means a continuance and progressive increase of the normal lactation atrophy of the uterus; the latter is felt to be very small bimanually, and the sound may pass only 1½ inches in a marked case; it is always associated with incurable amenorrhœa.

Deficient ovarian activity, whereby the Graafian follicles do not ripen or rupture, is not to be diagnosed by any of the ordinary methods we can employ, and it is doubtful whether a microscopic examination of the ovaries themselves would reveal the true condition. It is supposed to be associated with scanty menstruation or amenorrhœa for which no other definite cause can be found. Absence of ovarian activity must be the true cause in the general conditions which are outwardly shown by *obesity*, *anæmia*, and *disturbances of nutrition*; and it is a fact that some women have not conceived as long as they remain too fat, whilst loss of weight has in some cases been followed by conception. *Incompatibility* between husband and wife sexually is an ill-defined condition, which, however, is fully believed to be a cause of sterility. It is almost incapable of proof, for in the case of a sterile widow who remarries and conceives for the first time we have no proof that the former husband was capable of procreation. *Absence of sexual feeling* or the sexual orgasm, too, is not always a cause of sterility, for conception has occurred in women who are absolutely devoid of these feelings. On the other hand, most authors quote the case of a woman who conceived as a result of the only coitus at which an orgasm was experienced. The influence of *age* on child-bearing must not be forgotten, the liability to conceive falling rapidly every year over thirty.

Sterility of the Male.—Finally, the examination of the husband and his seminal fluid should never be omitted unless there is some quite well-defined cause to be found in the wife. Assuming that the penis and testes are present, and that erections render the sexual act possible, the seminal fluid must be examined carefully. The fluid should be collected in a condom by means of a normal coitus, and should be examined within twelve hours. It must be spread on a slide and examined with a high power of the microscope. There may be no spermatozoa present at all, the condition known as *azoöspermia*, in which case the husband is incapable of procreation. There may be but few spermatozoa, and those exhibiting only feeble powers of movement: *oligospermia*. There may be plenty of spermatozoa present, but quite devoid of motility: *necrospermia*. It is unnecessary in this article to enter into the causes of these conditions. They are usually incurable, and consequently further investigation is unnecessary. *Thos. G. Stevens.*

STERTOR is really another word for snoring; but it is commonly restricted to the heavy, snoring sound accompanying inspiration, produced not in the nose but by vibrations of the soft palate, generally when the patient is in a state of profound unconsciousness. It differs from stridor in that the latter is produced in the larynx. If, as is generally the case, the patient is comatose, the presence or absence of stertor helps little in the diagnosis, which is discussed under the heading COMA (p. 117). Sometimes, however, without being comatose, the patient may have stertor during sleep, when he is suffering from the effects of drink or from any of the following:—

Adenoids
Hypertrophied tonsils
Quinsy
Paralysis of the soft palate
Post-pharyngeal abscess.

The stertor in these cases is closely akin to snoring. The differential diagnosis generally becomes manifest when the interior of the mouth and the pharynx are examined. Possibly the condition most likely to be overlooked is post-pharyngeal abscess, but this should not be mistaken for anything else if a digital examination of the back of the mouth is made; moreover, except when due to tuberculous caries of the cervical vertebræ, it is commonest in infants and quite small children, becoming rarer with each year of life.

Herbert French.

STIFF NECK.—This occurs in a number of diseases entirely different in character, and its significance may be either grave or trivial. It is rarely that stiffness is the only symptom, but it may be the first thing complained of, or it may be a complication arising in the course of a disease. It is not right to assume that the trouble is trivial, or vaguely to designate it as 'rheumatic,' without a thorough investigation. It is necessary first to inquire into the history, when it may become obvious that it follows, say, an injury, or has arisen during the course of some disease, and is not primary. Next examine the patient with the head and shoulders bared, and see whether there is any swelling or abnormality present, also the extent of possible movement, and whether or not it is the movement that causes pain; if possible, locate the seat of the pain. Many further investigations may be necessary, e.g., examination of the throat for tonsillitis, the ear for suppurative otitis media, etc., according to the circumstances of the case.

Exposure to Cold or *Sleeping in a Cramped Position* may give rise to a transient stiff neck associated with no other symptoms. There is generally a distinct history of the patient waking up in the morning with a stiff neck, and the diagnosis is made by exclusion.

Inflammation of the Lymphatic Glands and the cellular tissues of the neck may cause local stiffness, whether the infecting focus be a boil or carbuncle, or a carious tooth, an inflamed tonsil, pediculosis capitis, or other similar cause. There is no spasm or rigidity of the muscles here, the neck can be moved quite well; but it hurts to do so, and therefore it is held stiffly. The diagnosis is easy as a rule.

Torticollis or *Wry-neck* is due to contraction of the sternomastoid muscle on one side, usually the result of an injury to the muscle caused by pulling on the aftercoming head in breech presentations. The muscle stands out as a tight band in the neck, and its contraction leads to a characteristic deformity. The head is pulled down towards the affected side, and the face and chin are tilted towards the opposite shoulder. The movements of the head are necessarily restricted owing to the shortening of the one muscle, and in long-standing cases this leads to a marked asymmetry of the face. The consequences are not limited to the head and neck, for the spine shares in the general obliquity, and shows marked lateral curvature in old cases.

Spasmodic Torticollis is an unusual form due to spasms of the sternomastoid and other muscles of the neck. The spasms are intermittent, coming on suddenly with great pain, the affected muscles relaxing after a variable time.

Cervical Caries.—The greatest care must be taken not to confound muscular rigidity with tuberculous disease of the cervical vertebræ. In the latter, pain and rigidity are among the earliest signs; the pain is increased by the least movement, and the child—for it is generally a child that is affected—takes the greatest precaution to avoid any movement, even holding the head between the two hands. The position of the head varies; it is most often held very stiff and straight, the natural backward curve of the neck being lost. In the late stages there may be an angular or lateral curve. The distaste for movement is very well brought out when the patient is asked to look round—the eyes only are moved, or the whole body is rotated. Bearing the possibility of this condition in mind, there is not much difficulty in diagnosis, but in doubtful cases a skiagram should be taken.

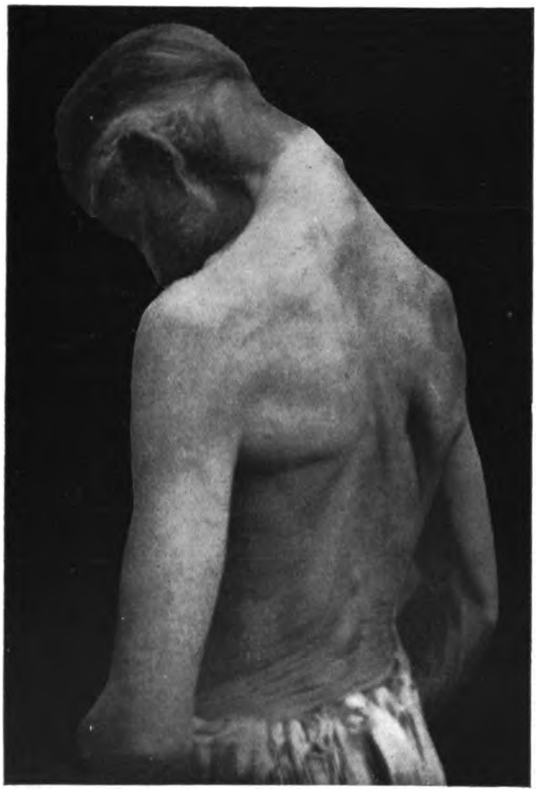


Fig. 274.—Spondylitis deformans. The patient was unable to raise the head, though he could just nod and shake it slightly. Except for the atlas and axis, the whole vertebral column was fixed. There was no affection of other joints in the body, and the general health was good.

New Growth in one of the cervical vertebræ may cause progressive stiff neck, and generally much local pain on movement; the diagnosis may suggest itself when the patient is known to have had a new growth elsewhere, especially a carcinoma of the breast or of the thyroid gland; cases of primary new growth of the vertebræ are fortunately rare, but myeloid sarcoma does occur; it can generally be diagnosed only after the patient has been observed for a long time. The symptoms simulate those of caries.

Infective Arthritis of the Cervical Vertebræ.—Following infective diseases such as scarlet fever, diphtheria, and tonsillitis, especially in children, there may ensue a very chronic form of suppurative arthritis affecting one or several of the cervical vertebræ, and going on sometimes to complete bony ankylosis. Similar stiffness of the back of the neck, especially its lower part, with pain on movement, is apt to occur in sufferers from rheumatoid arthritis (p. 341); it is due to infective periarthritis of the vertebral joints, secondary to toxic absorption from such sources of chronic infection as pyorrhœa alveolaris.

Spondylitis Deformans causes fixation of the neck (*Fig.* 274), though the movements of nodding 'yes' and 'no' remain; the nature of the case is at once indicated, however, by the fixation of the other regions of the spinal column also.

Injuries to the Neck.—A stiff neck may arise from some slight injury, such as a blow or a sudden twist. This will be clear from the history. Severe injuries involving fracture or dislocation are almost always fatal: if not immediately, then in a few days. A rare injury that may not be fatal is unilateral dislocation of one of the cervical vertebræ. This may result from a fall on to the head. From the start there are great pain and stiffness in the neck, the head being fixed immovably and turned to the opposite side to that of the displacement.

Burns.—A self-evident cause of stiffness is the cicatricial contraction following a burn on the neck.

Stiffness and retraction of the head are important indications of *meningitis*, but they are by no means constant; when present they are generally accompanied by other well-marked signs of meningitis.

Stiffness of the neck is one of the earliest signs of *tetanus*; it is rarely the only one, however. The trouble soon spreads to the jaw, causing trismus and other characteristic symptoms (p. 730).

Geo. E. Gask.

STOMACH, DILATATION OF.—(See DILATATION OF THE STOMACH, p. 173.)

STRABISMUS—Squints may be classified, according to their *direction*, into convergent, divergent, or attitudinal; according to their *cause*, into paralytic and non-paralytic (concomitant). The diagnosis between paralytic and non-paralytic strabismus is, as a rule, easy. In a paralytic strabismus the convergence or divergence of the two eyes is not constant in amount in all directions, as the farther the eyes are moved over in the direction of the action of the paralyzed muscle the greater will be the deviation from parallelism. In a concomitant squint the eyes always bear the same relative position to each other in whatever direction they are turned.

The diagnosis of the cause of a paralytic strabismus, which is associated with DIPLOPIA, is discussed under that heading (p. 174). The causes of concomitant strabismus are usually —*Error of refraction*; *Failure of binocular vision*; *Defective vision in one eye*; or the association of one or more of these conditions. The cause cannot be determined accurately without a careful examination of the ocular refraction under a mydriatic. In general terms it may be stated that convergent squints are, as a rule, due to hypermetropia, and divergent squints to myopia.

Herbert L. Eason.

STRANGURY differs somewhat from mere pain on micturition, in that, in addition to severe pain before, during, or after the act, the patient is troubled constantly by urgent and repeated necessity to discharge his urine, sometimes as often as every few minutes, yet without any satisfactory relief to his discomfort. The condition is also spoken of as vesical tenesmus. Very little urine is passed each time; sometimes the desire and the necessity are urgent when there is no urine in the bladder at all. The causes resolve themselves into five groups, as follows:—

1. **Nervous Conditions,** especially:—

Hysteria
Neurasthenia
Irritable bladder
Tabes dorsalis
 (vesical crises).

2. **Obstruction to the Urine Outflow, leading to Retention with Overflow:**—

Urethral stricture
Enlarged prostate
Carcinoma of the prostate
Retroverted gravid uterus
Uterine fibroid }
Ovarian cyst } Impacted in the pelvis
Ovarian carcinoma }
Extreme prolapse of the uterus and bladder
Calculus impacted in the urethra
Inflamed urethral caruncle
Gonorrhœa
Urethritis other than gonococcal
Periprostatic abscess
Periproctal abscess.

3. **Local Affections of the Bladder Wall:**—

Injury
Acute cystitis
Chronic cystitis
Tuberculous cystitis
Papilloma vesicæ
Carcinoma vesicæ
Infiltration by—
 Carcinoma of the uterus
 Carcinoma of the rectum.

4. **Reflex Conditions:**—

Inflamed hæmorrhoids
Injury to the back
Tuberculous kidney, before the bladder is involved
Acute myelitis } Without infection of the
Coli bacilluria } bladder wall.

5. **The effects of certain Drugs,** especially:—

Cantharides
Oxalic acid
Turpentine
Urotropine
Helmitol
Cystopurin
Hexamethylenetetramine.

Most of the conditions mentioned above, and the methods of distinguishing between them, are discussed in the article on MICTURITION, ABNORMALITIES OF (p. 393). Irritable bladder is discussed under the heading of OXALURIA (p. 423). Two points of importance deserve stress, however, and chief amongst these are the vesical crises of *tabes dorsalis*. The patient's sole complaint may be that he can never be far from a lavatory because of acute and painful calls to empty his bladder at frequent intervals; sometimes he has no sooner passed what is in his bladder than he has to run back and again do it, though there is no urine whatever to pass; and his vesical pains may be extreme. From loss of sleep his general health suffers, and he becomes anæmic and wasted to such an extent that carcinoma of the bladder or genito-urinary tuberculosis are simulated closely, and in some cases acute cystitis is diagnosed erroneously upon the symptoms alone. The true diagnosis will be suggested when it is discovered that the knee-jerks are absent and the pupils give the Argyll Robertson reaction; in some cases, however, the nature of the malady may be difficult to decide for a time, because crises of all kinds, like the lightning pains, are apt to develop in the earlier stages of tabes, when the knee-jerks are not yet absent. Both jerks should be tested, for there are a good many cases in which one knee-jerk is still present when the other has disappeared. A thorough examination of the urine and bladder should be carried out even if the patient is known to have tabes dorsalis, for he may have a gross lesion of the bladder in addition to his nerve disease; but this is unusual. The actual cystitis resulting from retention of urine with overflow is generally a late symptom, and not a relatively early one like the vesical crises, which are quite distinct phenomena. As time goes on the bladder crises may cease spontaneously just as the lightning pains, the rectal crises, and the other painful phenomena of tabes are apt to do.

The other point that merits attention is the strangury that certain drugs produce. *Cantharides* is familiar in this respect, but more from its prominence in text-books upon forensic medicine than from its occurrence in actual practice. The same applies to *oxalic acid* and to *turpentine*. It is less recognized that certain drugs in common use may be responsible for very similar symptoms, in which respect *urotropine*, *helmitol*, *cystopurin*, and *hexamethylenetetramine* are important. These are all employed in the treatment of pyuria, as well as for gall-stones and other conditions. If given for pyuria, when there may have been frequent and painful micturition already, before any treatment is begun, the increased frequency and pain that sometimes ensue when any of the above drugs are administered are very apt to be attributed to an increase in the cystitis or other genito-urinary lesion, and the dose of the drug is increased instead of diminished. It may be only after the patient deliberately ceases to take the medicine that the fact of the increase in the symptom being due to the drug becomes obvious; some cases develop strangury every time they take urotropine, and lose the symptoms a day or two after they have stopped the medicine. In some instances transient hæmaturia accompanies the strangury, and the danger always is lest these symptoms be attributed to the disease and not to the drug. Speaking generally, it is in cases in which the urine is concentrated, or at least in small amount, that urotropine and its allies are most liable to cause strangury and hæmaturia. If the patient drinks excess of water, so as to dilute his urine, these symptoms often disappear. Sometimes the patient can take helmitol with ease when he cannot bear urotropine, and vice versa. The important point is that urotropine and other drugs of like nature may be responsible for such strangury as may simulate local disease of the bladder, and unless this is borne in mind an erroneous diagnosis is liable to be made. *Herbert French.*

STRIDOR is a term used to denote a harsh, vibrating noise produced as the air passes in or out of a partially obstructed larynx or trachea. It may be due to many different causes, which may be classified as follows :—

1. **Partial Obstruction Inside the Larynx or Trachea** :—

   Mucus or muco-pus
   Foreign body
   Caseous gland bulging or rupturing into the trachea.

2. **Affections of the Wall of the Larynx or Trachea** :—

   Diphtheria
   Acute œdema due to—
      Bright's disease
      Potassium iodide
   Irritant vapours such as ammonia or chlorine
   Acute laryngitis—streptococcal, pneumococcal, staphylococcal
   Secondary infection in cases of tuberculous, syphilitic, malignant, traumatic, or post-typhoidal ulceration
   Stenosis after tracheotomy or cut throat
   Epithelioma of a vocal cord
   Fibroma of the vocal cords
   Epithelioma of the trachea
   Syphilitic stenosis.

3. **Swellings Outside Compressing the Larynx or Trachea** :—

Enlargement of the thyroid gland
Enlargement of the thymus gland
Thoracic aneurysm
Mediastinal new growth
Post-pharyngeal abscess
Epithelioma of the œsophagus invading the trachea
Malignant glands in the neck
Cellulitis of the neck
Erysipelas of the throat
Angina Ludovici.

4. **Bilateral Abductor Paralysis of the Vocal Cords,** generally due to syphilitic degeneration of the vagal-nuclear nerve cells.

5. **Unilateral Vocal Cord Paralysis** in a few cases ; generally if one cord remains normal there is no stridor. The diagnosis of the causes is given on p. 494.

Distinction is sometimes drawn between inspiratory and expiratory stridor, and stridor which is both inspiratory and expiratory ; but in practice such a distinction is not helpful. The main value of stridor as a symptom is that it indicates stenosis of the main air-passages by one or other of the above causes, except in those rare cases in which it is functional ; *hysterical stridor* ceases during sleep, is nearly always confined to the female sex, as a rule between the ages of fifteen and thirty, and is often associated with other functional nervous symptoms, such as globus hystericus and functional aphonia (p. 494). Stridor should never be diagnosed as functional, however, until every possible organic cause has been excluded. The differential diagnosis of the causes of obstruction to the main air-passages will be found discussed on page 418. *Herbert French.*

STUPOR.—(See Coma, p. 117.)

STUTTERING.—(See Speech, Abnormalities of, p. 628.)

SUCCUSSION SOUNDS may be heard when a part that contains any considerable bulk of both fluid and gas is shaken whilst the ear or the stethoscope is applied over the part. Sometimes the sounds are so loud that they can be heard at a considerable distance from the patient. A very good example of succussion sound is often afforded by the normal stomach after a quantity of fluid has just been swallowed. It is a mistake to suppose that gastric succussion sounds are evidence of abnormality ; they merely prove that the viscus contains fluid and gas at the same time ; the gas may be due to fermentation, but it is often nothing but air that has been swallowed during drinking. The chief value of gastric succussion sounds is that, according to the position in the abdomen at which they can be heard, they afford some clue as to the position, and perhaps as to the size, of the stomach. They should not be heard lower than the umbilicus ; if they are, the stomach is either displaced downwards, or dilated, or both.

Another variety of succussion sounds may sometimes be heard in the chest, especially in cases of *hydropneumothorax* ; when the patient deliberately oscillates his trunk to and fro, and then stops, the fluid and air can be heard making noises like those produced when a partly-filled barrel is moved about. Sometimes the fluid splashes up on to the collapsed lung and then drips off again into the pool at the bottom of the pleural cavity, each drop echoing in the cavity and producing a metallic clink like a *bruit d'airain* or coin sound. Similar succussion sounds may be produced by a pyopneumothorax or a hæmopneumothorax, the difference between these being decided, as a rule, by exploratory needling.

Succussion sounds other than those due to the stomach, or to gas and fluid in the pleural cavity, are uncommon, but the following is a list of the chief possible causes :—

1. **Causes of Succussion Sounds in the Thorax** :—

Hydropneumothorax
Pyopneumothorax
Hæmopneumothorax
Diaphragmatic hernia
Subdiaphragmatic abscess communicating with stomach or duodenum, and so containing air and pus ; or else infected with the *Bacillus coli communis,* and containing gas and pus
Hydropneumopericardium
Pyopneumopericardium
A huge phthisical cavity beneath a thin chest wall.

2. Causes of Succussion Sounds in the Abdomen :—

The normal stomach
Dilatation of the stomach
Enormous dilatation of the cæcum
Enormous dilatation of the sigmoid colon
Enormous dilatation of some other part of the colon
Pneumoperitoneum, due to : (i) Perforated gastric ulcer ; (ii) Perforated duodenal ulcer ; (iii) Perforated typhoid ulcer of the intestine ; (iv) Perforated tuberculous ulcer of the intestine ; (v) Perforated malignant ulcer of the colon; (vi) Production of gas by the *Bacillus coli communis*, either in a local abscess (e.g. appendicular or subdiaphragmatic) or in the general peritoneum
Subdiaphragmatic abscess communicating with the interior of stomach
Air and urine in the bladder (see PNEUMATURIA, p. 529)
Infection of an ovarian cyst or other collection of fluid by a gas-producing micro-organism.

Succussion Sounds in the Chest.—The diagnosis is not as a rule difficult. It is very rare indeed for a *phthisical cavity* to give succussion sounds ; but should it do so, the phenomenon would be apical rather than basal, and thus distinguishable from most cases of hydro- or pyo-pneumothorax. It is possible for the latter to be apical, however, if old adhesions prevent the parietal and visceral layers of pleura from separating in the lower part of the chest, and then, if tubercle bacilli were found in the sputum, it would become a matter of opinion as to whether the sounds were produced in the pleural cavity or in a huge vomica. *Hydro-* and *pyo-pneumopericardium* are very rare, and they are at once distinguished by the extraordinary churning sounds made by the heart beating within the mixture of air and fluid. Survival is improbable. The cause is generally either an epithelioma of the œsophagus opening the pericardium from behind, a foreign body, such as a tooth-plate, ulcerating through from the œsophagus, or the opening of an air-containing subdiaphragmatic abscesst hrough the diaphragm into the pericardium, or infection of the pericardial sac by a gas-producing organism such as the *Bacillus coli communis.*

A *subdiaphragmatic abscess* containing air owing to communication with a hole in a gastric or duodenal ulcer, sometimes pushes the diaphragm up so high that the condition may be mistaken for hydro- or pyo-pneumothorax ; it may be possible to distinguish the two by knowing that the trouble began with gastric ulceration ; on the other hand, it may be impossible to tell which it is until the position of the diaphragm is ascertained, either by the use of the *x*-rays, or by operation. When the trouble is subdiaphragmatic, the tendency is to displace the heart upwards rather than towards the opposite side of the chest, whereas the contrary is true of pneumothorax.

Diaphragmatic hernia is very rare ; it may be congenital, or it may be the result of severe injury to the abdomen and chest. In neither case are the patient's prospects of survival good. The exact diagnosis may not be arrived at without operation or post-mortem examination ; if the stomach is herniated into the thorax, however, the effects of eating and drinking upon the physical signs may point to the diagnosis, or the *x*-rays may be used to demonstrate the gastric shadow after the administration of bismuth or barium salts by the mouth.

In most cases of *hydropneumothorax* there is little difficulty as to the diagnosis of the condition itself ; it may be less easy to decide what the hydropneumothorax is due to. If the onset has been sudden, with acute pain in the affected side of the chest, cyanosis, and dyspnœa, the commonest cause is *phthisis.* The sputum should be examined for tubercle bacilli. In some instances an injury may have been the immediate cause, but injury will very seldom produce hydropneumothorax unless there was already a tuberculous or other lesion in the lung at the time of the accident. Hydropneumothorax may result temporarily after *paracentesis thoracis.* If there has been bleeding at the same time, *hæmopneumothorax* may be found, and it is common after bullet wounds of the chest. Either a hydro- or a hæmo-pneumothorax may become infected with pyogenetic organisms and converted into a *pyopneumothorax.* The diagnosis will be confirmed by needling the chest. Pyopneumothorax is apt to escape detection, however, because it arises when the patient is too ill to be shaken—in cases of gangrene of the lung for instance, resulting perhaps from lobar pneumonia, obstruction of a bronchus by a foreign body or a new growth, or the breaking down of an infective bronchopneumonia or pulmonary infarct. Generally speaking one may say that the existence of well-marked succussion sounds in the pleural cavity of a patient who has sufficient vigour to shake his own body to and fro indicates hydropneumothorax of phthisical origin.

Succussion Sounds in the Abdomen.—The first point in the differential diagnosis of succussion sounds in the abdomen is to decide whether the sounds are *gastric* or not. Generally this is obvious ; if there is doubt, the effect of putting more gas or more fluid into the stomach by taking a seidlitz powder in two halves, or by drinking a quantity of water, will usually so change the character and distribution of the sounds if they are gastric, that little doubt will remain ; or the *x*-rays and bismuth method of demarcating the stomach may be employed. As has been mentioned, the existence of gastric succussion is no proof of gastrectasis ; if, however, the succussion sounds are audible over a larger area than the normal stomach should occupy, they afford valuable evidence of *gastrectasis*, and the next step will be to determine the cause of the latter. Dilatation of the stomach has three main causes, namely, atony, non-malignant pyloric obstruction, especially by a healed simple ulcer, and malignant pyloric obstruction by primary gastric carcinoma.

The presence of visible peristaltic waves, or the occurrence of vomiting, will exclude simple atony, which can never be diagnosed with certainty until it is known that there is no pyloric obstruction. The latter will be indicated by the periodicity of the vomiting ; by the abundance of the fluid vomited being greater than the amount taken at the last meal ; by the presence in it of particles of food eaten a day or more previously—ham, for instance, vomited on Tuesday when last partaken of on Sunday ;—by the visible peristaltic waves corresponding with the stomach ; and by the presence of sarcinæ in the vomit (see *Fig.* 121, p. 241). The most certain method of detecting pyloric stenosis, however, is by means of bismuth or barium and the *x*-rays, especially in those cases, not infrequent, in which vomiting does not take place in spite of great gastrectasis.

There may also be evidence of delay in the absorption of substances that are not dissolved until they reach the pancreatic juice in the duodenum, tested for instance by giving methylene blue in keratin-coated capsules, and observing when the urine first begins to be blue. Keratin is not dissolved by gastric juice, but is by pancreatic ; if there is no evidence of pancreatic disease, delay of more than one to two hours in the first sign of blueness of the urine, after giving the capsules, indicates marked delay in their transit from stomach to duodenum.

It is often a matter of extreme difficulty to decide whether pyloric stenosis is simple or malignant, though upon the whole the shorter the history, the older the patient, and the more definite the pyloric thickening or lump the more likely is the lesion to be carcinomatous. The latter may occur in quite young subjects, however, even between 20 and 30 ; and a long history does not exclude carcinoma, since some cases of simple ulcer ultimately become malignant. Even when laparatomy is performed for the relief of the condition, its nature may not be obvious ; sometimes, indeed, post-mortem examination has failed to decide whether the stenosed pylorus was carcinomatous or not, until microscopical examinations have been made. It has been stated that if the gastric juice after a test meal contains a normal amount of HCl, the diagnosis is unlikely to be carcinoma, and vice versa ; but even this general rule has many exceptions (p. 270).

The diagnosis of pyloric stenosis due to other causes than adhesions, a healed ulcer, or a carcinoma is seldom possible without a laparotomy ; occasionally such out-of-the-way things as a calcified retroperitoneal cyst adherent to the pylorus and thought to have been a carcinoma pylori may be found.

If there are well-marked abdominal succussion sounds that can be shown to be definitely not gastric, there are generally other well-marked signs and symptoms which materially assist the diagnosis.

Succussion sounds in the general peritoneal cavity are excessively rare, for even though this cavity should contain both gas and fluid, for instance after perforation of a typhoid ulcer, the coils of bowel prevent the sounds from being produced readily. The list of causes given above indicates the conditions that may be present. It would clearly be next to impossible to diagnose most of them unless the previous state of the patient was known accurately, or unless exploratory laparotomy were resorted to. It is important to remember that the *Bacillus coli communis* produces gas, so that intra-abdominal abscesses, appendicular and otherwise, are not infrequently resonant. The occurrence, however, of marked non-gastric succussion sounds in the abdomen of a patient who is not acutely ill will generally arouse a suspicion that there is distention with gas and fluid of some part of the large bowel, especially the cæcum or the sigmoid colon. This distention will generally be the result either of chronic constipation (see p. 121) or of some cause of intestinal stenosis.

In some cases that were formerly described as idiopathic dilatation of the colon, but which are now regarded as chronic volvulus of the sigmoid colon, the result of persistent constipation, the sigmoid dilatation may be so extreme that this part of the intestine bulges up as far as the diaphragm (Hirschsprung's disease, see *Fig.* 55, p. 127, and *Fig.* 171, p. 389); the occurrence of succussion sounds in such a dilated colon might readily lead to the erroneous diagnosis of gastrectasis; the pear-shaped outline of the dilated viscus, and the fact that it is known to have come upwards from the pelvis, may indicate the true nature of the case, but sometimes the fact that succussion sounds are colonic and not gastric can only be determined by giving large doses of bismuth by the mouth and then outlining the stomach by the dark shadow cast by the bismuth under the x-rays. *Herbert French.*

SUGAR IN THE URINE.—(See GLYCOSURIA, p. 260.)

SUPPRESSION OF URINE.—(See ANURIA, p. 39.)

SWEATING, ABNORMALITIES OF.—The functional disorders of the sweat-glands, *sudamen* (*miliaria*) and *hidrocystoma*, are dealt with from the diagnostic point of view in the article on VESICLES (p. 753). The other abnormalities require but the briefest notice in a work on diagnosis, for it is hardly possible to confuse them with each other or with any other conditions. In *hyperidrosis* the secretion of sweat is excessive, either over the whole skin or in some particular region, e.g., the palms and soles, and especially covered parts furnished with large sweat-glands, such as the axillæ and genital regions. Occasionally hyperidrosis is limited to the area of distribution of a particular nerve—the fifth, for example. In some cases a peculiar pink tint of the inner side of the palm and the ball of the little finger and thumb has been noticed. In rare instances hyperidrosis in delicate children is associated with *granulosis rubra nasi*, a condition in which the skin of the nose becomes intensely red, and is dotted over with minute deep-red specks and papules, the papules gradually developing into pustules which soon dry up. The cells around the sweat-ducts are infiltrated, and both ducts and coils, and also the blood-vessels and the lymphatic spaces of the corium, are dilated. This complication is distinguishable from rosacea by the age of the patient and the absence of telangiectases and of change in the sebaceous glands; from eczema, by the absence of vesiculation and weeping, and its obduracy to local treatment; from lupus erythematosus, by the absence of scales; and from lupus vulgaris, by the absence of apple-jelly nodules. The night sweats of *phthisis*, and those associated with *rickets* and with *infantile scurvy* (Barlow's disease, p. 556), are not, as a rule, difficult to attribute to their cause.

In *anidrosis* the secretion may be merely diminished or totally suppressed, and either the whole skin, or only some particular area, may be affected. The abnormality is rarely idiopathic, but is usually associated with ichthyosis, psoriasis, eczema, sclerodermia, belladonna poisoning, with malnutrition or with disordered innervation; under the latter heading one may mention in particular the unilateral sweating of the face and head that accompanies irritation of the cervical sympathetic by an aneurysm, thoracic cyst, or new growth; the outbursts of local perspirations, such as a band of sweating round the body, that constitute a rare symptom of tabes dorsalis—a sweating crisis; and the sweating of half the body—hemidrosis—that may be a purely functional or hysterical phenomenon.

Bromidrosis, or foul-smelling sweat, sometimes associated with hyperidrosis, may occur in connection with such general affections as acute rheumatism, uræmia, and scurvy, or following a serious illness like pneumonia, or may be idiopathic. Occasionally generalized, it is much more frequently limited to particular parts, such as the feet, the axillæ, and the perineum. The foul smell is due to the growth of the *Bacillus fœtidus* upon the sweat after exudation.

In *chromidrosis* both sweat and sebum may be coloured, generally some shade of blue, but occasionally red, green, yellow, violet, and even black. The pigmentation is usually localized, the most frequent situations being the eyelids, cheeks, forehead, and side of the nose ; but occasionally the whole of the face and large parts of the trunk and limbs, and especially the axillæ and groins, are affected. The condition is often a neurosis, but it may be due to the ingestion of copper (green sweat), or of iron (blue sweat), or to the action of cocci or the *Bacillus pyocyaneus* upon the sweat after secretion. The chief point in diagnosis is the exclusion of imposture.

Hæmatidrosis, or bloody sweat, generally limited to particular parts, the face, hands, feet, navel, etc., may be a form of so-called vicarious menstruation, or an expression of emotional stress in highly-strung persons ; it is sometimes simulated by the presence in the sweat of bacteria producing a red pigment, such as the *Bacillus prodigiosus*. *Uridrosis*, in which urinary constituents are present in the sweat in abnormal quantity, is not an idiopathic affection, but an accompaniment of such grave conditions as cholera and uræmia. It is quite unmistakable ; the sweat has a urinous odour, and white crystals will be seen on the skin.

It is worthy of note that the sweat may have peculiar effects when the patient is taking certain drugs ; for example, persons whose occupation it is to make polished steel implements, may be discharged from their employment if they are taking mercury and iodide of potassium, because the articles they have polished go dull and spotty almost at once, in a way which does not result from ordinary perspiration. *Malcolm Morris.*

SWELLING, ABDOMINAL.—This may be acute or chronic, general or local, and caused by abdominal accumulations that are mainly either gaseous, fluid, or solid. The *position*, *physical consistency*, and *duration* of abdominal swellings are their three outstanding clinical features for purposes of diagnosis.

CLASSIFICATION.

I.—Swellings in the Abdominal Wall.

II.—General Abdominal Swellings :—

A. Mainly Gaseous—
- Surgical emphysema
- Meteorism (p. 338)

B. Mainly Fluid—
- Ascites (p. 43)
- Large cystic tumours
- Distention of hollow viscera
- Hydatid disease

C. Mainly Solid—
- Obesity (p. 408)
- Constipation (p. 121)
- Inflammatory deposits
- New growths

III.—Local Abdominal Swellings :—

A. Due to General Causes—
- Encysted ascites
- Tuberculous peritonitis
- Hydatid disease
- Subphrenic abscess
- Phantom tumours
- Enteroptosis

B. Due to Enlargement of Particular Organs.

I. SWELLINGS IN THE ABDOMINAL WALL.

Swellings situated in the abdominal wall itself can be recognized by their superficial position ; by their adherence to the skin, muscles, or fascia ; or by their not following the movements of the viscera immediately underlying the wall of the abdomen, to which they must therefore be superficial. But it may be impossible to distinguish between a fatty tumour in the deeper part of the wall, for example, and a fatty omental mass that has become adherent to the parietal peritoneum and so has practically incorporated itself with the abdominal wall.

Inflammatory swelling of the wall may occur by infection from without or, less often, from within. Thus a liver abscess may cause extensive redness and swelling in the right hypochondriac region ; infiltration of the abdominal wall is often met with in operations for appendicular abscess ; in acute cases of *Hodgkin's disease* and *lymphosarcoma*, tumours suggesting a subacute inflammatory process may occur in the abdominal wall, but they are really localized lymphadenomatous or sarcomatous deposits, not due to infection, and are associated with gland enlargement in other parts of the body. *Inflammatory swelling about the umbilicus* is not rare in newly-born infants, due to the entrance of infection by way of the cord; in stout uncleanly adults the umbilical fossa may be the seat of intertrigo, which becomes painful, swells, and suppurates; but a far more serious umbilical inflammation may occur in patients, usually children, with *tuberculous peritonitis*; a tuberculous mass in connection with the round ligament may break down, perforate at the umbilicus, set up a chronic discharge there, and ultimately establish a fæcal umbilical fistula; in rare cases a *subdiaphragmatic* or *perigastric abscess* may cause inflammatory thickening of the round ligament and umbilicus.

Œdema of the abdominal wall may be either local or general (see ŒDEMA, p. 410).

Tumours of the abdominal wall, excluding those due to inflammation, are rare except in certain situations. *Lipomata* and *fibro-lipomata* may occur in any part of it, and in the inguinal or femoral rings closely imitate omental hernias. *Herniæ* are commoner, particularly at the umbilicus and in the groins; there is little likelihood that a definite hernial protrusion in any part of the abdominal wall will be overlooked, but minute hernias into the abdominal wall, such as may occur along the linea alba, especially above the umbilicus, at the femoral or inguinal rings, or along the lineæ semilunares, may suffice to produce complete intestinal obstruction and yet be small enough to demand very careful palpation for their discovery.

In *malignant disease* of the stomach, pylorus, or region of the portal fissure generally, small secondary nodules may appear quite early at the umbilicus or in the round ligament just above it; and this may occur before the primary tumour has given rise to any definite signs or symptoms.

II. GENERAL ABDOMINAL SWELLING.

A. **Mainly Gaseous.** — In certain cases of extensive *surgical emphysema* the fascia planes of the abdominal wall are invaded and dissected out by gas, which imparts to them a highly characteristic feathery, crepitant, and crackling feeling on palpation. The gas may have entered from wounds in the neck, thorax, or trachea, or it may have been generated by gas-producing microbes in any abscess or focus of inflammation in the trunk or viscera, and have made its way thence into the abdominal wall.

Distention of the intestines with gas is an event so common as to be familiar to all; its diagnosis is discussed under METEORISM (p. 388). In this condition the whole of the abdomen, or in special cases some part of it only, is distended, and on percussion gives a highly resonant or tympanitic note. It often happens that the outlines of the gas-distended viscera can be seen on the abdominal wall, particularly when it is looked at in an oblique illumination. The increased size of the inflated intestine is apt to produce displacement of the other viscera; the dome of the diaphragm is pushed up into the chest, carrying the heart with it and shifting the apex-beat upwards; the liver is similarly pushed up, and in addition it is often caused to rotate round a transverse axis, its lower anterior edge ascending and its lower posterior edge descending, with the result that the area of liver-dullness in front is much reduced, or even lost altogether; but it is reduced only a little in the mid-axillary line so long as the gas remains in the intestine; and if the liver-dullness in the mid-axillary line disappears, the diagnosis of free gas in the peritoneal cavity is to be made.

B. **Mainly Fluid.**—The diagnosis of the various causes producing accumulations of fluid in the peritoneal cavity is given under the heading ASCITES (p. 43).

C. **Mainly Solid.**—In OBESITY (p. 408) the abdomen may swell either in consequence of the deposit of fat in the abdominal wall itself, or as the result of fatty deposits behind the peritoneum generally, in the mesentery, in the omentum and appendices epiploicæ. In very fat patients it is rarely possible to diagnose the exact nature of an intra-abdominal mass by the usual methods of palpation and percussion, and without having recourse to exploratory laparotomy, because the abdominal walls are so thick. The frequency with which inconveniently large fatty accumulations occur in the abdomens of such persons must not be forgotten when the diagnosis of some vaguely-felt tumour within the abdomen has to be considered.

In severe chronic cases of CONSTIPATION (p. 121), abdominal distention may result from accumulation of fæces in the large intestine, particularly when dilatation of the colon, idiopathic or secondary, is present. The scybala can usually be felt, perhaps soft and plastic in the region of the ascending colon, usually hard and nodular in the descending sigmoid and colon. *Idiopathic dilatation of the colon* (*Hirschsprung's disease*) seems to be congenital, and is associated with much hypertrophy of the colon (*Fig.* 171, p. 389). A description of it will be found on p. 126. In older patients a very similar *dilatation and hypertrophy of the colon* may come on as the result of chronic obstruction about the lower end of the large intestine. As much as 47 lb. of fæces may accumulate in the intestines of such patients.

In rare cases of chronic, particularly tuberculous, peritonitis, semi-solid *inflammatory masses* may bring about a general swelling of the abdomen; the diagnosis is discussed under Ascites (p. 47). General swelling of the abdomen may occur in *malignant disease* of the peritoneum, due in part to the growth of numerous secondary malignant nodules in part to a concomitant ascites. The symptoms are often vague at first—loss of weight, strength, appetite, with indefinite abdominal disorders. The abdomen enlarges, and if there is not much ascites the secondary deposits can be felt obscurely through the abdominal wall. Occasionally they can be palpated in the abdominal wall itself near the umbilicus, or in the round ligament above it, or the urachus below. As the case progresses, emaciation becomes marked, the skin loses its elasticity and often develops a diffuse brownish pigmentation; bedsores are not rare. The primary growth may be in any of the thoracic, abdominal, or pelvic organs, or in the mamma or testis. If any primary growth can be made out, the diagnosis will not be difficult, especially if glandular enlargements are found in the groins or axillæ, if the ascitic fluid is hæmorrhagic, and if it is found to contain multinuclear endothelial cells, and cells with atypical mitotic figures. Tuberculous peritonitis can be excluded by means of the tuberculin test and by the result of injecting several c.c. of the ascitic fluid into guinea-pigs.

Hydatid disease of the peritoneum is discussed on p. 49.

III. LOCAL ABDOMINAL SWELLING.

A. **Due to General Causes.**—It often happens that the causes which produce general swelling of the abdomen fail to do so in particular cases, and give rise only to a local swelling. Thus in *encysted ascites*, left behind after an acute diffuse peritonitis, or accompanying a chronic peritonitis, an accumulation of fluid bounded by fibrinous or fibrous adhesions between the adjacent viscera may be found in any part of the peritoneal cavity, but most often in the flanks and about the pelvis. If a good history of the case can be obtained, the diagnosis of the nature of such a cyst will at least be suggested; the physical signs will be those of a fixed cystic or semi-solid tumour, and the diagnosis will often be obscure until laparotomy has been performed.

Abdominal swellings of the most various size and position may occur in *tuberculous peritonitis* (p. 47, under Ascites). Many of them are composed of the infiltrated and rolled-up omentum, others of enlarged and tuberculous mesenteric glands, others of doughy masses of adherent intestine and fibrin. The amount of ascitic fluid varies widely in different cases. When there is much, and the patient is an adult, the diagnosis of cirrhosis of the liver is likely to be made; when the peritonitis is dry (the obliterative form), the abdominal cavity may be smaller than normal, and occupied by a doughy, rather tender mass that presents areas of alternating resonance and dullness. The physical signs vary from day to day according to the amount and position of flatus in the intestine. The symptoms of tuberculous peritonitis are very variable; as a rule the patient is thin, anæmic, seriously ill, with a drawn aspect; abdominal pain and tenderness are usual, nausea and constipation with chronic intestinal obstruction are frequent; in acute cases there is high fever, in chronic the temperature is not high, but irregular, or may even be subnormal. If there is ulceration of the large intestine, diarrhœa may occur, and blood may be passed in the motions. The discovery of tuberculosis in some other part of the body, or of a family history of tuberculosis, is strong argument for regarding a case with such signs as tuberculous. A general or patchy brown pigmentation of the skin is not uncommon.

Single or multiple *hydatid cysts* may occur in any part of the abdominal cavity. Usually they are single, the large majority occurring in the liver; more rarely they affect the spleen, omentum, mesentery, or peritoneum. The cyst grows slowly, and is spherical except in so far as it is moulded by the pressure of adjacent structures. It contains a clear saline fluid, in which may be found hooklets (*Fig.* 18, p. 49), and secondary cysts completely detached from the walls of the parent cyst. Until it has become large enough to cause mechanical obstruction and pressure-symptoms, the single hydatid cyst gives rise to little pain or complaint. It then produces a bulging of the overlying abdominal wall, smooth, rounded, more or less tense, dull on percussion ; if it is of a certain degree of tenseness it may yield the hydatid thrill—just as any other cyst may. Hydatid cysts in the mesentery, omentum, or peritoneum are often multiple, and may be felt as scattered, rounded tumours generally accompanied by ascites ; the disease runs a slower course than malignant peritonitis, and echinococcal cysts may be found in the ascitic fluid if it is tapped. As a rule, these are secondary after rupture (accidental or by paracentesis) of a primary cyst in the liver. They cause a slowly progressive enlargement of the abdomen, which appears to be filled with a solid or semi-solid mass ; if the individual cysts are large, they can be seen outlined on the abdominal surface, and can be felt. They are freely movable, and not connected with any particular viscus ; they do not, as a rule, give the hydatid thrill on percussion. Peritoneal hydatid disease is rare except in countries (Iceland, Australia, or South America) where the inhabitants live in close company with dogs that are the hosts of *Tænia echinococcus*. Eosinophilia may be found, and when the cysts are living and active the patient's blood-serum may give a specific hydatid precipitin reaction, though the absence of both this and eosinophilia does not exclude hydatid disease ; exploratory laparotomy may be necessary before the diagnosis can be established.

Any part of the abdomen may swell from the formation of an *abscess* ; several forms of which are more or less localized, and are considered below (see *B*) ; others present more generalized abdominal signs and symptoms, and will for that reason be considered here. A *subphrenic abscess* is any abscess in contact with the under surface of the diaphragm except those situated in the liver or in the spleen. It is intraperitoneal in more than half the instances ; it contains gas in about half the cases. The *simple* or *non-gaseous abscesses* are generally the result of appendicitis or of suppuration in the liver, and so are usually on the right side of the body ; less often they are secondary to gastric or duodenal ulcer, or to suppuration spreading from the pancreas, kidney, Fallopian tubes, spleen, or thorax. They are deep-seated, and tend to produce abdominal swelling with signs and symptoms that are indefinite. The onset is insidious, often consisting in nothing more than failure to recover from the primary disorder—appendicitis, hepatic abscess—after it has been treated surgically ; the patient remains seriously ill, with fever and quick pulse, leucocytosis, and often a septic aspect. If the abscess is at the back the signs may point to pleurisy or pleural effusion, with the appropriate pain and friction sounds. If it pushes forwards, the hypochondrium and epigastrium may bulge in front and become tender. The diagnosis of subphrenic abscess may be very difficult when there is no obvious antecedent to suggest its occurrence, especially if the abscess is behind and below the liver, and is complicated by pleurisy or empyema. If it is above the liver, it may be very difficult to say whether the pus is inside the liver or outside it, or both ; enlargement of the liver downwards is in favour of intrahepatic abscess. Examination with the *x*-rays is often of great assistance ; but often it is necessary to give the patient a general anæsthetic and insert a long exploring needle successively into the intercostal spaces (tenth to sixth) in the scapular and mid-axillary lines. It must be thrust in deeply. As viewed by the *x*-rays the diaphragm is depressed by empyema or pleural effusion, elevated by subphrenic abscess, and immobilized by either.

The gas-containing abscess or *subphrenic pyopneumothorax* is commoner in females than in males, and is usually due to the perforation of a gastric, or less often a duodenal, ulcer, or to appendicitis ; in rare instances it is secondary to an ulcer of the colon or even to a suppurating hydatid cyst. It is usually on the left side. When a gastric ulcer perforates, the onset is generally sudden, with acute abdominal pain and collapse ; but both the ulcer and its perforation may be latent, and nothing more than a history of chronic dyspepsia may be obtainable. The abdomen soon becomes distended ; hectic fever, with rigors, rapid pulse, leucocytosis, and shortness of breath, are the symptoms likely to appear. The physical signs, on the whole, resemble those of PNEUMOTHORAX (p. 530) ; the diaphragm is pushed up into the thorax, and the gas in the abscess-cavity below it causes the signs of pneumothorax to develop in the upper part of the abdominal cavity and the lower part of the thorax. The picture is complicated by the fact that the inflammatory process habitually spreads through the diaphragm, so that the signs due to pleurisy, with or without effusion, are added. The diagnosis has to be made between this condition and true pneumothorax. The points that serve to distinguish the two are, that in pneumothorax the gas seems to occupy the whole of one side of the thorax, the heart is pushed or pulled over to the sound side, and the physical signs are limited to the thorax ; whereas in subphrenic pyopneumothorax the signs occur at the base of one or both lungs but not at the apex, the heart is displaced upwards but not to either side, and the upper part of the abdominal cavity is involved as well as the thorax. Examination with the x-rays is of the greatest service, for it shows that the gas-containing cavity is below the diaphragm and not above it ; the readiness with which the level of the fluid in the abscess changes as the patient alters his position can also be noted, and proves that the abscess-cavity contains gas as well as fluid.

The abdomen is not infrequently the seat of *phantom tumours.* These are felt as fixed and more or less rounded smooth swellings, either in or immediately underneath the abdominal wall ; they are dull on percussion, and may be tender on palpation. They are caused by involuntary contractions of the muscles in the area in which they occur ; they persist when the patient's attention is distracted, and also during sleep, but disappear under a general anæsthetic. Phantom tumours are commoner in women than in men, and in the neurotic than in the stolid. They often persist for long periods, but may vanish when the patient believes that they have been cured. A phantom tumour in the region of the liver may simulate cholecystitis, hepatic abscess, or gumma ; in the left hypochondrium, gastric carcinoma ; in the appendix region, an appendicular abscess ; above the pubes, pregnancy (*pseudocyesis*).

In *enteroptosis* (Glénard's disease), or downward displacement of abdominal viscera, any or all of the organs may slip away from their normal position and attachments (*Fig.* 56, p. 127). The diagnosis must be made by the discovery that one or more of the viscera is out of place, and is also abnormally mobile.

B. **The Regional Diagnosis of Local Abdominal Swellings.**—For clinical purposes the abdomen may be subdivided into nine regions, by two vertical lines drawn through the middle of Poupart's ligaments, and by two horizontal lines, one passing through the lowest points of the tenth ribs (the subcostal line), the other drawn at the level of the highest points of the iliac crests (*Fig.* 275). The viscera, or portions of viscera, commonly contained in the areas thus demarcated, are given in the table on page 660.

The abdominal swellings that may be felt in and about these nine regions will now be considered seriatim, excluding the tumours situated in the abdominal wall itself that have been described under heading I. (p. 655).

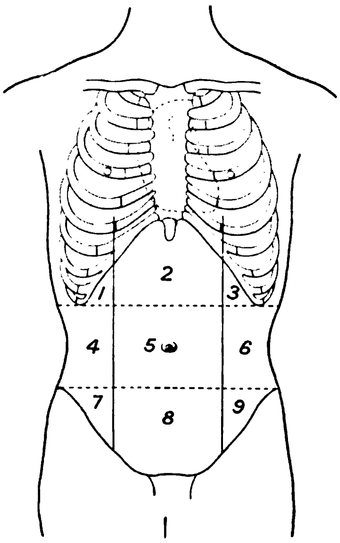


Fig. 275.—The regions of the abdomen : for the significance of the numerals, see the table on page 660.

1. *Right Hypochondriac Region.*

Most tumours in this area are connected with the liver or gall-bladder, and their diagnosis is discussed under LIVER, ENLARGEMENT OF THE (p. 366), and GALL-BLADDER ENLARGEMENT (p. 252).

To regard the firm and rounded swelling produced by the upper segment of the right rectus abdominis muscle as evidence of tumour, enlargement, or induration of the liver or gall-bladder, is a mistake easily and frequently made.

Tumours in connection with the *hepatic flexure of the colon,* excluding scybala, are rare. Scybala may be recognized by their general shape, by the ease with which they can be moulded or indented by the pressure of the fingers, and by their being dispersed by a purgative. Carcinoma or tuberculosis of the colon may produce a palpable tumour here; and so may an intussusception. The diagnosis must rest upon the previous history and the course of the disease.

THE NORMAL CONTENTS OF THE ABDOMINAL REGIONS.

1. **Right Hypochondriac**	2. **Epigastric**	3. **Left Hypochondriac**
Liver Gall-bladder Hepatic flexure of colon Right kidney	Liver Stomach and pylorus Transverse colon Omentum Pancreas Duodenum Kidneys Suprarenal capsules Aorta Lymphatic glands	Liver Stomach Splenic flexure of colon Spleen Tail of pancreas Left kidney
4. **Right Lumbar**	5. **Umbilical**	6. **Left Lumbar**
Riedel's lobe of the liver Ascending colon Small intestine Right kidney	Stomach Duodenum Transverse colon Omentum Urachus Small intestine Kidneys Aorta Lymphatic glands	Descending colon Small intestine Left kidney
7. **Right Inguinal**	8. **Hypogastric**	9. **Left Inguinal**
Cæcum Vermiform appendix Lymphatic glands	Small intestine Sigmoid flexure Distended bladder Urachus Enlarged uterus and adnexa	Sigmoid flexure of colon Lymphatic glands

Tumours of the *kidney* or *suprarenal gland* rarely present themselves in this region of the abdomen.

2. *Epigastric Region.*

Abnormal lobes in the liver, tumours in either of its lobes or in its falciform or round ligament, may be felt here.

In thin people and children the curvatures of the normal *stomach* when it is full may often be seen dimly outlined in the epigastrium, the lower curvature habitually, the upper less often, and the gastric succussion-splash can often be elicited here in healthy persons as well as in those with dilatation of the stomach. An epigastric splash is usually gastric, but may be colonic. In *dilatation of the stomach* due to obstruction at the pylorus (caused in infants by spasm or hypertrophy of the pylorus, in adults by malignant or cicatricial stenosis), waves of peristalsis travelling from left to right may be seen in the epigastrium. Similar waves, but travelling from right to left, occur in the colon of patients with obstruction in the rectum or sigmoid (see below). *Tumours of the stomach,* usually carcinomatous, rarely sarcomatous, or due to inflammatory deposits round a gastric ulcer, may sometimes be felt here, particularly when the patient takes a deep breath and drives the abdominal viscera down and out of the cover of the diaphragmatic dome ; it must not be forgotten that the normal pylorus can sometimes be felt in an infant, child, or thin adult, as a rounded finger-like mass deep in the right side of the epigastrium. The connection of a gastric tumour with the stomach can often be made out more clearly if that organ is inflated with gas ; or by the method, little used in this country, of gastrodiaphany. Examination with the *x*-rays after the administration of a bismuth or a barium sulphate meal is often of great assistance in obscure gastric cases, and should always be employed.

The *transverse colon* goes across the lower part of the epigastrium in some cases, more usually across the upper part of the umbilical area. Its sacculations and peristalsis are often outlined on the abdominal walls of pot-bellied rickety children or of thin adults particularly when they are flatulent or constipated. In acute or chronic obstruction the peristalsis becomes much more marked. Tumours of the transverse colon are very rare, except the common occurrence of scybala in it; a few cases of chronic hyperplastic tuberculosis of this part of the colon have been recorded, with great diffuse thickening of its wall and stenosis of its lumen.

Swellings in connection with the *omentum* lie below the colon and in immediate relation with the anterior abdominal wall, in front of the mass of small intestine. In *tuberculous peritonitis* it often forms an irregular rope or mass composed of inflammatory tissue, cheesy tubercle, or encysted exudate, that may lie in the epigastrium, or extend into any of the regions of the abdomen—when the diagnosis of malignant disease of the intestine or some other viscus may possibly be made. Similar nodular enlargement and deformity of the omentum is common in *chronic peritonitis* of any sort; and it may become the seat of an abscess in cases of perforated gastric ulcer. *Cysts of the omentum*, single or multiple, are not very rare, and are often inflammatory in origin. Tuberculous or inflammatory masses of omentum often adhere freely to the anterior abdominal wall as well as to the neighbouring viscera.

Swellings derived from the *pancreas* push forwards from the depths of the abdominal cavity towards the epigastric and the upper part of the umbilical areas, and present themselves as deeply-seated vaguely-felt masses on palpation. They have the stomach, or the stomach and colon, in front of them, and are fixed to the posterior abdominal wall; they are usually made out best by examination under an anæsthetic; they move little on respiration, and often transmit from the adjacent aorta a non-expansile pulsation. They are separated from the liver and from the spleen by areas of resonance. These swellings may be carcinomatous, in which case wasting, anæmia, and jaundice are likely to be observed, with death in a few months' time; or due to chronic pancreatitis, when the course of the disease will be slower and there will be more epigastric tenderness and pain, with clayey stools and perhaps intermittent jaundice, a positive Cammidge's reaction (p. 100), or glycosuria. In acute pancreatitis the swollen pancreas has only exceptionally been palpated before laparotomy; the main symptoms are acute epigastric pain, vomiting, constipation, fever, and proneness to collapse, simulating in part acute intestinal obstruction and in part acute peritonitis, so that immediate laparotomy is usually resorted to and the diagnosis established by the discovery of opaque yellow patches of acute fat necrosis in the omentum and elsewhere within the abdomen.

Pancreatic cysts, so-called, are often cysts not in the pancreas but in its neighbourhood, and therefore better called peripancreatic cysts. Pancreatic cysts proper are single or multiple retention-cysts, usually the result of chronic pancreatitis; they form deeply-seated, smooth, rounded tumours, possibly giving a feeling of fluctuation. At first they occupy the lower epigastric or hypochondriac regions; but if they enlarge much they may fill the whole upper part of the abdomen, or extend down to the pubic symphysis or flanks. The symptoms of chronic pancreatic disease should be present—chronic indigestion, the passage of pale and bulky stools, glycosuria, perhaps jaundice from time to time if pancreatic calculus is present. *Peripancreatic* or *retroperitoneal cysts*, due to accumulations in the lesser sac of the peritoneum, or to growths originating in residues of the Wolffian body behind the peritoneum, may produce apparently identical cysts; the evidences of chronic pancreatic disease should be absent in these cases, but the diagnosis may be impossible until laparotomy has been performed. Cammidge's urinary reaction (p. 100) is said to be of assistance in diagnosing these cases.

Swellings in connection with the *duodenum* are felt in the right side of the epigastric and umbilical areas, and are usually due to primary malignant disease. In many cases they escape palpation because they are so deeply placed, and they usually have to be diagnosed from such conditions as cancer of the stomach, pylorus, pancreas, bile-ducts, and portal fissure generally, not by their physical signs but by the general symptoms and progress of the disease. A growth in the first part of the duodenum produces symptoms like those of cancer of the pylorus—wasting, anæmia, progressive dilatation of the stomach with visible peristalsis, attacks of copious vomiting, and occasional hæmatemesis perhaps; the motions contain bile, but the vomit does not; jaundice is absent unless there are secondary growths in the portal fissure. Malignant disease of the second part of the duodenum, in or involving the biliary papilla, soon produces obstructive jaundice and distention of the gall-bladder, and often leads to suppurative cholangitis, whereas cancer in the head of the pancreas or bile-ducts produces steady jaundice and is not followed by suppuration in the bile-passages. Cancer in the third part of the duodenum or below the bile papilla produces duodenal stenosis, with dilatation of the duodenum and stomach and frequent vomiting ; but in this case the vomit is habitually bilious and contains the pancreatic ferments. If there is no stenosis, the bilious vomiting will be less, and the case may be indistinguishable from one of cancer of the stomach. In most of these cases the exact diagnosis is more often made post mortem than ante mortem.

Swellings in connection with the *kidneys* and *suprarenal* capsules occur in the epigastrium only after they have reached a considerable size. They rise up out of the loin and flanks, and their diagnosis is considered below.

Enlargement of the *spleen* may bring its blunt anterior end or its notched upper edge into the epigastric area. The splenic swelling always lies in contact with the anterior wall of the abdomen, with the stomach above and behind it (see SPLEEN, ENLARGEMENT OF THE, p. 628).

In every region of the abdomen *lymphatic glands* abound, and any of these may become palpable in cases of Hodgkin's disease, tuberculous peritonitis, or malignant disease. The enlarged glands are felt as nodulated chains or masses, usually hard and rounded, but softer and even cystic if their contents caseate or break down into pus ; they may also calcify, when they become hard and stony. The enlarged glands that will be felt in the epigastric area are those connected with the stomach, liver, and mesentery ; the diagnosis must be made on general and anatomical lines (see LYMPHATIC GLAND ENLARGEMENT, p. 376).

3. *Left Hypochondriac Region.*

An abnormally lobulated *liver* may make a superficial tumour in this area continuous with the main mass of the liver in the epigastric region. In the same way, a tumour in the left lobe of the liver may project superficially into the left hypochondrium.

Part of the *stomach* lies in this region normally ; the diagnosis of gastric swellings has been considered above. A gastric tumour may often be differentiated from a tumour of the adjoining spleen by the fact that while the spleen is anchored at its hilum, and so is capable of but little movement, the stomach is highly mobile, changing its position with the position of the patient, and also in accordance with its fullness and distention.

The diagnosis of a tumour of the splenic flexure of the *colon*—scybalous, tuberculous, or malignant—is considered above (see 1 and 2).

The diagnosis of the various causes of enlargement of the *spleen* is discussed under SPLEEN, ENLARGEMENT OF THE (p. 631). The tumour is usually to be recognized by the fact that it comes down from under the left costal margin in direct contact with the anterior abdominal wall, descends on inspiration, has a smooth surface, and a notched upper and inner margin. In exceptional cases, however, the enlarged spleen seems to adopt a more compact and cubical form in place of its usual elongated prismatic shape, and also to lie back in the loin and left lumbar region instead of occupying the anterior and upper part of the abdominal cavity ; it then simulates a tumour of the left kidney or suprarenal body, and unless the blood and leucocyte count give a definite lead the diagnosis may be settled only by a laparotomy. Conversely, a spleen-shaped hypernephroma or suprarenal tumour, or a calculous and cystic kidney, may easily be mistaken in an anæmic patient for an enlarged spleen, unless the possibility of the error be kept in mind.

Tumours of the *pancreas* and *retroperitoneal cysts* may project into the left hypochondrium (see 2 above).

Tumours of the left *kidney* and *suprarenal body* rarely appear in the left hypochondrium unless they are very large (see 6 below). Unless very large, they have the stomach or the stomach and colon in front of them, and so are variably resonant on percussion—according to the amount of gas in those viscera—and are also less distinctly palpable than tumours arising from the spleen, stomach, colon, or omentum, that may be felt in the same situation.

4. *Right Lumbar Region.*

When the *liver* is abnormally lobulated, either congenitally or as the result of tight lacing, a thin flange of liver-tissue, known as *Riedel's lobe*, may be met with as a superficial tumour, continuous with the liver above it, in this region. Sometimes it is freely movable, and then may be mistaken for a movable kidney or for a dilated gall-bladder.

The *ascending colon* can usually be palpated and rolled under the fingers as a tube-like structure at the confines of this and the umbilical region ; when empty and contracted it may feel almost rod-like. Its contents are usually fluid, but it may contain semi-solid or solid fæcal masses that can be moulded by pressure, in constipated patients. In patients with obstruction lower down, it may be greatly distended, and show sacculation and visible peristalsis. It may become much thickened with inflammatory tissue, or even come to lie in an abscess of its own production, in pericolitis, perityphlitis, typhlitis, appendicitis, and hyperplastic tuberculosis of the colon, forming a thickened and tender mass immediately under the abdominal wall ; the patient will be more or less acutely ill, with local pain and tenderness, constipation, often vomiting. In the more chronic of these cases, the diagnosis of malignant disease of the colon will often be suggested.

General thickening of the ascending colon, with tenderness and characteristic mucous or blood-streaked stools, is common in muco-membranous colitis, in dysentery, and in ulcerative colitis. The first of these is met with in nervous constipated women ; dysentery, amœbic or bacterial, is caught abroad, and is commoner in men than women ; while ulcerative colitis, whether it be dysenteric or no, is a severe and progressive painful diarrhœa, often associated with vomiting and irregular fever, that commonly leads to emaciation and death from exhaustion or intestinal hæmorrhage in a few weeks or months.

The ascending colon can be felt as a sausage-shaped tumour in acute, subacute, and chronic ileocæcal and ileocolic *intussusception* : at first in the right flank, then extending across the abdomen above the umbilicus, and finally down the left flank and into the pelvis. The chief symptoms are spasmodic abdominal pain, vomiting, the passage of blood and mucus by the rectum, and tenesmus ; the palpability and consistency of the elongated tumour vary according to the degree of muscular spasm in it.

The *small intestine* is but rarely the cause of abdominal swelling in this region, excepting when it becomes the seat of enteric intussusception.

Tumours in connection with the *right kidney* and *suprarenal body* usually make their first appearance deep down in this region, having the ascending colon and small intestine in front of them. They can be lifted forwards *en masse* from behind by a hand placed at the back of the loin. For their diagnosis see KIDNEY, ENLARGEMENT OF (p. 352.) The lower pole of the right kidney can be felt in normal persons on deep abdominal palpation ; but when the kidney is abnormally mobile, the whole of it may be felt, and in rare cases it may be found in any of the adjoining abdominal areas. The shape and consistence of the movable kidney are characteristic, and the patient complains of a peculiar sickening sensation when it is grasped bimanually ; in the lesser degrees of mobility it disappears readily into its normal position under cover of the diaphragm, and ceases to be palpable until the patient drives it down again by a deep inspiration. As regards its diagnosis, the movable right kidney will hardly be mistaken for anything else in this region ; on the other hand, Riedel's lobe of the liver, the enlarged gall-bladder, fæcal accumulations or a cancer of the ascending colon, and omental masses, have all been mistaken for it, although they are all superficial to the kidney, and lie in contact with the anterior abdominal wall. Other wandering tumours, e.g., of the ovary, Fallopian tube, mesentery, hydatid disease, may give rise to the same error if reniform.

5. *The Umbilical Region.*

Examination with the x-rays after a bismuth-meal has shown that the normal *stomach* is a far more mobile organ than was formerly supposed, and that in health its lower margin often descends even below the level of the umbilicus ; but if much of it habitually occupies the umbilical region, it is probably dilated to a pathological degree, either from atony or from pyloric obstruction (*Fig.* 276).

Tumours in connection with the *transverse colon* have been considered under the headings 1 and 4 above.

Tumours in connection with the *omentum* are common in this region : those arising from the *small intestine* are rare. Both are superficial, and their diagnosis has been given above (see 2).

Abdominal swellings in connection with the *urachus*, which runs from the umbilicus to the bladder, are considered below (see 8).

Swellings arising from the *duodenum*, *kidneys*, *suprarenals*, *pancreas*, and *mesentery*, may all present themselves in the deeper parts of the umbilical region, usually as more or less fixed masses arising from or connected with some definite part of the posterior wall of the abdomen. Their diagnosis will depend mainly upon the success with which the origin and connections of the tumour can be made out; if the patient is fat, or if relaxation of the abdomen cannot be obtained, palpation under a general anæsthetic may be desirable. Consideration must also be given to any general symptoms such as may point to renal calculus, hydronephrosis, pancreatitis, for example.

The *aorta* bifurcates half an inch below and just to the left of the umbilicus. In thin, nervous, and excited patients, particularly young women, great pulsation of the aorta can often be felt in the umbilical and lower epigastric areas, and may lead to the wrong diagnosis of abdominal aneurysm. Careful examination will almost always show that this pulsation is no more than a throbbing, an up-and-down movement as the patient lies, without lateral expansile pulsation. Aneurysm of the abdominal aorta is very rare; it is seen in patients who have had syphilis, and is commoner in men than in women. The aneurysmal sac is distinctly larger than the normal aorta, and presents diagnostic expansile lateral pulsation met with in no other condition. These abdominal aneurysms often leak into the retroperitoneal tissues; large irregular clots of blood, weighing several pounds and of the most varied extent and distribution, may form gradually in the flanks, pelvis, and back of the abdomen generally, causing the patient great pain by their situation and rendering him anæmic and breathless. The abdominal aneurysm also causes pain and stiffness in the back by eroding the bodies of the vertebræ upon which it presses.

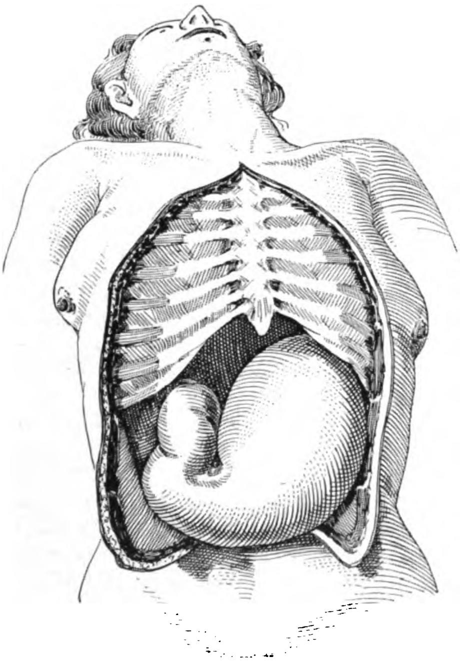


Fig. 276.—Idiopathic dilatation of the stomach. The organ post mortem almost filled the abdominal cavity—*Introduction to Surgery* (Rutherford Morison).

6. *Left Lumbar Region.*

The enlarged *spleen* (see 3) may intrude into this area; it forms a firm mass, dull on percussion, and is in contact with the abdominal wall, driving the splenic flexure of the colon inwards or downwards before it. The spleen, when enlarged, comes down into the abdomen in front of all the other structures in the left side, and its abdominal dullness is continuous with its thoracic dullness, which extends back and up into the axilla along the line of the ninth or tenth ribs. Tumours of the stomach, omentum, suprarenal, kidney, or descending colon, may all be in contact with the anterior abdominal wall, and though usually nodular and irregular, may present a smooth and spleen-like surface on palpation. They may be distinguished from the enlarged spleen by the fact that they produce no such typical area of thoracic dullness in continuity with the dullness of abdominal tumour; while the renal and suprarenal tumours may in addition be shown to occupy the back of the loin, so that they can be tilted forwards by the fingers placed behind just outside the edge of the erector spinæ muscles, and so pushed against the other hand, which is placed on the anterior surface of the loin. A suprarenal tumour may be associated with sexual precocity (see *Figs.* 174, 175, p. 408).

The diagnosis of tumours of the *small intestine*, *kidney*, and *suprarenal gland* in this region has been given already.

7. *Right Inguinal Region and Right Iliac Fossa.*

Abdominal swellings in the right inguinal region are rarely confined to it, and usually extend into the outer part of the hypogastric region, occupying what may be described somewhat indefinitely as the right iliac fossa.

New growths, inflammatory thickenings, and abscesses in connection with the *cæcum* and *appendix* may all extend into this region of the abdomen, giving rise to more or less acute and severe abdominal symptoms—pain, fever, vomiting, constipation, with a tumour in the right iliac fossa. The physical signs are very variable, depending on the extent and acuteness of the process, the degree to which the abdominal wall can be relaxed, the exact position of the tumour—an abscess to the inner side of and behind the cæcum and appendix may lie too deeply to be felt per abdomen. The rare condition of sarcoma or lymphosarcoma of the cæcum may be associated with fever; the tumour is soft, and the diagnosis of some chronic inflammatory condition will probably be made. A cæcal carcinoma is usually a harder mass and of slower growth; it tends to constrict the bowel, with the result that fæcal accumulation occurs behind it, and so the new growth may be overlooked when the hard mass of impacted fæces is discovered. The diagnosis of appendicular abscess has been made in patients with movable right kidney during a Dietl's crisis; fever is usually absent in the latter; careful examination will generally show that the tumour in the right iliac fossa is an enlarged and movable kidney, and a history pointing to intermittent hydronephrosis, with polyuria after the acute attacks, should be obtainable. Inflammation of the right ovary or tube, or ovarian neuralgia occurring with the catamenia, may all give rise to symptoms in nervous patients that closely simulate those of appendicitis; and if scybala are present in the cæcum, vaguely felt as a tumour through the rigid abdominal wall, the mistaken diagnosis of appendicitis may easily be made; but as a rule pelvic symptoms and signs will be found, and pain be felt in the pelvic region and the lower part of the back; the diagnosis will be cleared up by a vaginal or rectal examination—which, indeed, should never be omitted when there is any doubt as to the exact causation of an inflammatory swelling in the iliac fossa—and by the previous history of the case.

Inflammatory swellings and abscesses in the right iliac fossa may arise in connection with *psoas abscess*, *abscess* originating in the *sacro-iliac joint*, *hip-joint*, or *ilium*, and from the swelling or breaking down of *lymphatic glands* (the external iliac) infected from some perhaps trivial wound in the leg or perineum. The symptoms of bony disease about the hip or the pelvic girdle will be present; the leg will be held more or less stiffly in some abnormal attitude of flexion and inversion to relieve the pain, and movement of the leg will be painful. Unless local peritonitis is present, there will be none of the special symptoms that point to appendicular or cæcal disease..

8. *Hypogastric Region.*

In rare instances, tumours arising in the *small intestine*, and more often the sausage-like swelling of an enteric intussusception, may be felt in the hypogastric area (see 4). Tumours extending into it from the iliac fossæ are described under headings 7 above and 9 below.

In infants, the *bladder* reaches half way to the umbilicus when moderately full, and does not fall below the pubic symphysis when empty. In the adult, the distended bladder is a common hypogastric swelling, particularly in females with *retroverted gravid uterus*, or in males of about sixty with *enlargement of the prostate*; it may reach up as an ovoid elastic mass arising from the front of the pelvis almost to the umbilicus under conditions that are in no way pathological, as well as when the retention is due to some pathological cause. Such a distended bladder (*Fig.* 277) has been tapped as ascites, operated upon as ovarian or urachal cyst, and diagnosed as the pregnant uterus: mistakes that are not likely to occur if these possibilities be remembered, and are put out of court by micturition or the use of a catheter before the diagnosis is made.

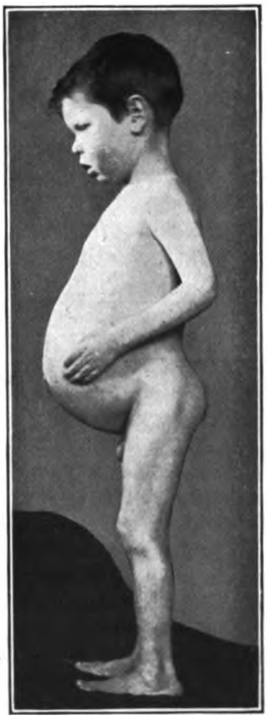


Fig. 277.— Idiopathic dilatation of the bladder. The physical signs were those of a cystic tumour occupying the lower part of the abdomen.—*Introduction to Surgery* (Rutherford Morison).

The *urachus* is a fibrous cord running in front of the peritoneum from the top of the bladder to the umbilicus, in the middle line; it sometimes becomes the seat of cyst-formation, more often in women than in men. The urachal cyst is a rounded tumour lying between the umbilicus and pubes, soft or firm according to the tension of its contents; it may produce hypogastric pain. It must be distinguished from encysted tuberculous peritonitis, from ovarian cystadenoma, and from the distended bladder.

Abdominal swellings arising from the *uterus*, *ovaries*, *tubes*, and *uterine ligaments* may all rise up out of the pelvis and present themselves as swellings in this region, and, as they grow larger, may spread into the whole or any part of the abdomen. While they are comparatively small and manifestly connected with some intrapelvic organ, their origin is not difficult to determine; their diagnosis is considered under SWELLING, PELVIC (p. 688). But when they have grown up into the abdomen, or have acquired a long pedicle, or have become fixed by adhesions to some distant part of the abdominal wall or to some other viscus, perhaps causing it to become inflamed and impairing its functional activity, these pelvic tumours may give rise to signs and symptoms suggesting any disease rather than one that is pelvic, and the true diagnosis may be very difficult to make. The possibility of pregnancy in the female should always be remembered.

9. *Left Inguinal Region and Left Iliac Fossa* (see 7 above).

The *sigmoid flexure of the colon* can be felt normally as a tube-like cord passing from the left lumbar region down into the pelvis, and rolled under the fingers. It very frequently contains hard ovoid scybalous masses. In rare instances it may be uniformly thickened and tender in consequence of chronic inflammation, tuberculous or otherwise. It is occasionally the seat of cancerous new growth, when the patient will complain of chronic intestinal obstruction, with cachexia, tenesmus, and the passage of blood-stained stools, phenomena that may also be met with in hyperplastic or stenotic tuberculosis of the sigmoid.

The left iliac fossa may be the seat of abscess or inflammations similar to those described under 7 above. In addition, suppuration around an exaggerated colonic diverticulum, with symptoms not unlike those of appendicitis on the wrong side, has been known to occur; such a condition has been spoken of as *acute diverticulitis* of the colon

A. J. Jex Blake

SWELLING, AXILLARY.—Swelling in the axilla is due in the great majority of cases to enlargement, from one cause or other, of the lymphatic glands; a subsequent abscess, either acute or chronic, is frequent. Any other form of tumour is distinctly rare. In examining a case, therefore, these two causes should be uppermost in the mind, and indeed, on inspection only, the diagnosis may be obvious, e.g.:—

Acute Abscess may be recognized at once by the well-marked signs of local inflammation and the general febrile disturbance. There is one form of acute abscess that may not be obvious, namely, one situated in the upper part of the axilla and covered by the pectoral muscles. On account of its distance from the surface the local signs of inflammation may not be great, though the general signs are marked. There will be great disinclination to move the arm on account of pain, and there is usually some cause, such as a whitlow on the finger, to account for the trouble. It must be remembered, however, that the abscess may be 'residual'; that is to say, the original source of infection, such as the whitlow, may have healed completely two, three, or even more weeks before the axillary abscess declares itself. Occasionally an empyema points in the axilla; there are generally, but not always, abnormal lung signs to suggest the diagnosis.

Chronic or **Tuberculous Abscess** forms a single fluctuating swelling which, if large, may extend upwards under the pectoralis major. Owing to the fact that few, if any, of the local signs of inflammation may be present, difficulty arises in distinguishing this form of abscess from a soft lipoma. The duration and the rapidity of growth of the swelling are a good guide, for though the duration of a chronic abscess may run into months, it does not exist for years, as does a lipoma.

Enlargement of the Lymphatic Glands.—Next, supposing that examination proves that the swelling is not an abscess, attention should be directed to ascertain whether it is glandular, and it is therefore necessary to recall the anatomical position of the glands. The axillary lymphatic glands are ten to twelve in number, and are arranged in three sets. One chain surrounds the axillary vessels and receives the lymphatics from the arm; a small chain runs along the lower border of the pectoralis major as far as the mammary gland, receiving the lymphatics from the front of the chest and the breast; the third chain is placed along the lower margin of the posterior wall, to receive lymphatics from the integuments of the back. If the glands are affected in any way, all need not necessarily be enlarged, but it would be extremely unusual if only one were picked out, and commonly two or three, or one entire group, are affected. Therefore axillary swellings due to glandular enlargement are almost always multiple, and are situated in the part of the axilla where glands are normally present. This may not be quite accurate when much inflammation has occurred around the glands and they are matted together, as happens with tuberculous infection; but even then the mass may be felt to be made up of many glands. For the differential diagnosis of glandular swellings, see LYMPHATIC GLAND ENLARGEMENT (p. 376).

Primary Tumours of the Axilla are distinctly rare.

Lipoma is the most common. It may attain a large size and extend up under the pectoral muscles. It should be diagnosed by its long history, slow growth, definite outline, and free mobility. When very soft, the tumour may give the feeling of fluctuation, and so be mistaken for a chronic tuberculous abscess. The skin wrinkles when one attempts to raise it away from the tumour.

Cystic Hygroma of the axilla is very rare. It is usually congenital. It forms a soft, fluctuating, painless swelling, which sometimes grows rapidly. It may easily be mistaken for a lipoma.

Primary Malignant Tumours may arise, but are of extreme rarity.

Aneurysm of the Axillary Artery does occur, but is uncommon. It is easily recognized, because it is comparatively superficial and it gives an expansile pulsation, synchronous with the heart's beat; the veins of the forearm may be distended on account of pressure on the axillary vein, and the radial pulse on the affected side is diminished in size and delayed. There may be a definite history of local injury, or in cases of apparently spontaneous aneurysm there may be signs or symptoms of fungating endocarditis (p. 34).

George E. Gask.

SWELLING ON A BONE.—It is presumed that the swelling has been ascertained to be of the bone, immovable apart from it, and that it is not merely some tumour lying close to it.

The following method of examination should be adopted: (1) Inquiry into the clinical history, mode of onset, duration; (2) Search for the signs of inflammation; (3) Evidence as to whether the swelling is a localized projection or involves the whole circumference of the bone; (4) Investigation for involvement of other bones or further signs of disease, e.g., tuberculosis, syphilis, rickets, etc.; (5) A skiagram should always be taken if possible; (6) If a discharge is present, a bacteriological examination should be made.

The various swellings may be classified under the following headings:—

(I.) *Injury*; (II.) *Infective Diseases*; (III.) *General Diseases*, not limited to one bone; (IV.) *Tumours*; (V.) *Cysts*.

I. INJURY.

A blow or kick may give rise to a swelling due to *extravasation of blood* or serous fluid under the periosteum. This disappears rapidly, but may leave a small permanent thickening or node. Such a node is found not infrequently on the shins of football players. A fracture of bone is followed by the formation of *callus*, which forms a large swelling if the broken ends do not lie in accurate apposition, or if there is too much movement between them. After four to six weeks the callus begins to be absorbed, and it may disappear entirely; in most cases a small permanent swelling indicates the site of fracture. A green-stick fracture may not show any swelling at first, and may be overlooked on this account, being discovered only when the formation of callus draws attention to it.

II. INFECTIVE DISEASES.

These give rise to inflammatory changes in bone, the signs of which are more or less obvious, according to the nature and virulence of the infection. These changes have usually been named according to the chief starting-point (periostitis, osteomyelitis, etc.), though they seldom remain confined to one particular part of the bone. In this article the classification will be made according to the nature of the infecting organism, viz., pyogenic (staphylococci and streptococci), tubercle, syphilis, etc.

A. **With Pyogenic Organisms.**

Acute infection may occur through wounds or injuries, or via the blood-stream. The resulting swelling is due to the formation of pus between the periosteum and the bone ; this may be a localized abscess, or the whole of the periosteum may be stripped off and the bone lie bare in a bag of pus. The disease usually occurs in young people, and the intimate attachment of the periosteum at the epiphyseal lines limits the spread of suppuration ; in long-standing cases the pus may burrow farther and even burst into the joint. Suppuration is rarely limited to the surface of the bone, but spreads into the marrow, causing osteomyelitis ; lymphatic absorption and septic embolism are liable to give rise to a general blood-infection and pyæmia.

The signs of inflammation are abundant : the swelling is acutely painful and tender, the skin over it red and œdematous, and the constitutional signs of fever are marked. If the blood is examined, a high leucocytosis will be found.

It is important not to mistake *erythema nodosum* for this affection ; in erythema nodosum the red swellings are generally multiple, bilateral, and confined to the shins ; it is rare for acute osteomyelitis to be bilateral and symmetrical, and confined to the parts between the knees and the ankles.

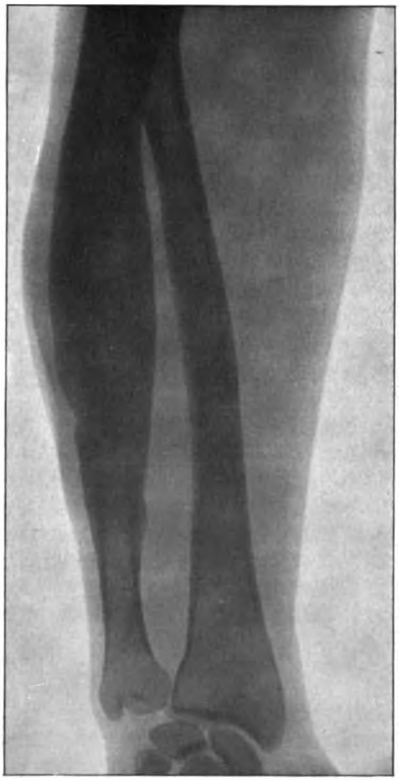


Fig. 278.—Skiagram from a case of chronic periostitis of the ulna, due to pyogenic infection. (*Skiagram by Dr. Hugh Walsham.*)

Chronic infection.—Such a condition as detailed above may often become chronic and cause a swelling which may last for months, years, or through life. If the pus formed under the periosteum escapes, either by bursting or through an incision, sinuses form, and the periosteum, in the process of repair, becomes thickened. If during the height of the inflammation, a portion of the bone has died—necrosis—this acts as a foreign body, keeps up inflammation and suppuration, and great thickening of all the constituent parts of the bone results (*Fig.* 278). Usually the diagnosis can be arrived at without difficulty. Occasionally, if the inflammatory changes have not been great, and the amount of necrosis is small and deeply-seated (central necrosis), a condition resembling a slow-growing sarcoma may result. If a skiagram is taken it will be observed that the chronic inflammatory periosteal thickening is added on to or 'applied' to the original compact layer of bone, whereas in the case of sarcoma, though there may be thickening and formation of bony or calcareous spicules in the growth, the compact layer is eaten away (*Figs.* 283, 286, 287, pp. 671, 673). However, this may be somewhat slender evidence on which to base the diagnosis between so important a condition as sarcoma and inflammation, and if doubt arises an incision should be made into the tumour, so that a portion may be removed for pathological investigation.

B. **Tuberculous Disease** usually starts in the cancellous tissue of the small bones of the carpus, tarsus, and phalanges, and at the ends of long bones. The inflammatory changes, which are slight, give rise to caries of the affected bone ; the external signs of inflammation are little marked, and it is comparatively rare for any swelling of the bone to result, though the soft parts around the bone may be swollen considerably.

Tuberculous dactylitis (*Fig.* 279) furnishes an instance in which the disease forms a periosteal swelling. It is found most often in quite young children, and the bones commonly affected are the metacarpal bones and phalanges of the hand. The affected digit exhibits a fusiform enlargement, slightly tender, which on rest tends to diminish. Tuberculous periostitis may develop in any long bone, on the ribs and the humerus most commonly, and it then has to be differentiated from syphilis.

Chronic tuberculous abscess of bone occurs most frequently in the young adult, and nearly always in the articular extremity of a long bone, by preference in the upper end of the tibia. Enlargement of the bone is found only when the abscess approaches the surface and involves the periosteum. The skin then becomes a little red and œdematous, and there is generally a small spot that is exquisitely tender on firm pressure. It is to be noted that when secondary infection with pyogenic organisms occurs—a not infrequent event—all the swellings described under " acute infection " may result. A skiagram will generally reveal the true condition ; if not, a diagnostic injection of Koch's old tuberculin may be made, or von Pirquet's skin reaction tested.

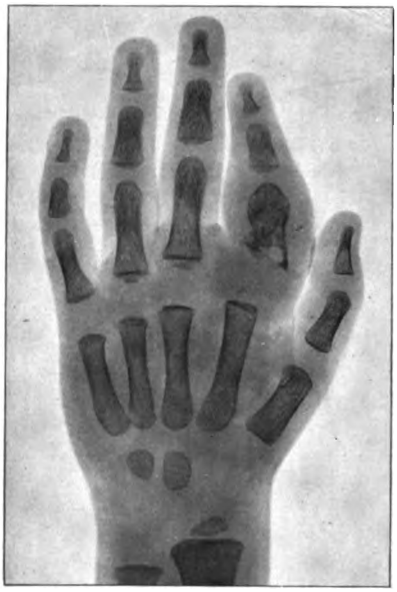


Fig. 279.—Skiagram from a case of tuberculous dactylitis of the first phalanx of the index finger of a child.

(*Skiagram by Dr. Hugh Walsham.*)

C. **Syphilis** in the acquired form may lead to periosteal thickenings in the secondary stage and to gummata in the tertiary. The former give rise to excessively tender swellings on the surface of the tibiæ, clavicles, sternum, ribs, or skull. They are generally multiple, two or three often being found on the same bone. The patient complains of pain, particularly in bed, when the extra warmth causes further dilatation of already inflamed vessels. Relief is given almost at once by potassium iodide. Sometimes one of these swellings is followed by the formation of compact periosteal bone, giving rise to a node which fades gradually into the surrounding parts, like a hill rising gently from a plain.

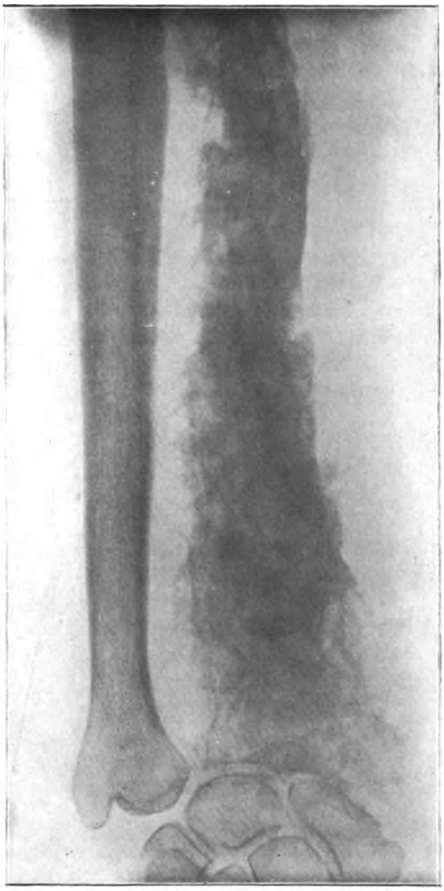


Fig. 280.—Skiagram showing typical syphilitic disease of the radius ; the appearances are diagnostic of this condition. The ulna is normal.

(*Skiagram by Dr. C. Thurstan Holland.*)

Gummata may form localized swellings, or may invade the whole substance of the bone, causing osteomyelitis and general thickening (*Fig.* 280). The condition has to be distinguished from tuberculosis, chronic pyogenic infection, and sarcoma ; such recognition is arrived at by means of the Wassermann test, and the fact that antisyphilitic remedies cause a marked and rapid improvement. Diagnosis by incision has rarely to be resorted to.

In *congenital syphilis* two forms of bony swelling are common : (*a*) Periosteal thickenings of the bones of the vault of the skull, called Parrot's nodes—the hot-cross bun or natiform skull ; (*b*) In new-born infants, epiphysitis and separation of the epiphyses. So painful is a limb thus affected that it is kept motionless, and may be thought to be paralyzed.

D. **Typhoid Fever.**—In the course of this disease a periosteal node or abscess may form. From the fluid a pure culture of typhoid bacilli may be obtained perhaps for a long time after the fever. The nodes by no means always break down into pus.

III. GENERAL DISEASES NOT LIMITED TO ONE BONE.

Rickets.—The ordinary form is well known, and can hardly be confused with any other disease.

Scurvy-rickets is quite distinct from rickets. It arises generally in infants under twelve months old, who have been fed too exclusively on artificial foods or preserved milk. The disease therefore is more common among the children of the rich than the poor. The child is often brought to the doctor on account of the sudden appearance of an exceedingly painful swelling of a long bone, such as the femur. The swelling may fluctuate, and yield on aspiration blood-stained fluid. Spontaneous fracture is liable to occur. The diagnosis is indicated by the fact that the child is anæmic, and has spongy gums and hæmorrhages from the mucous membranes. The condition is most likely to be confused with acute suppurative periostitis and traumatic fracture.

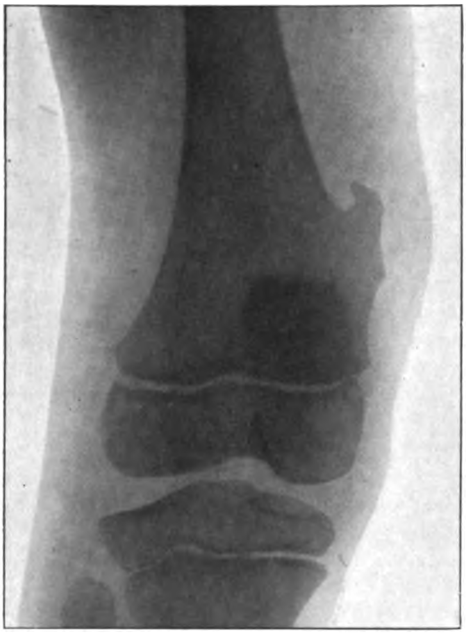


Fig. 281.—Skiagram of a common variety of cancellous exostosis of the femur.
(*Skiagram by Dr. Hugh Walsham.*)

Osteitis Deformans (see *Figs*. 63, 64, p. 155) is a senile disease, very chronic, and characterized by thickening, lengthening, and bending of the bones. The whole osseous system may be affected, but attention is first drawn to the disease by thickening of the tibiæ and forward bending of the knees, and by enlargement of the head. In the rare event of one bone only being affected, it may be confused with syphilitic osteitis, and only be recognized on the failure of antisyphilitic remedies and by the subsequent involvement of other bones. The patient suffers from neuralgic pains, and in the later stages from dyspnœa. In such cases death sometimes occurs from the development of multiple sarcomata of the bones.

Acromegaly (*Fig*. 116, p. 237) is described on p. 237.

Leontiasis Ossea.—In this disease there is general overgrowth of the cranial and facial bones, and one of the chief symptoms may be the fact that the patient has to get progressively increasing sizes of hats.

Swellings of bones associated with diseases of joints may be found in *gout*, *osteo-arthritis*, and *pulmonary hypertrophic osteo-arthropathy*. (See JOINTS, AFFECTIONS OF, p. 351.)

IV. TUMOURS.

These are innocent and malignant. Innocent tumours as a whole are characterized by their long history, slow growth, localized projection, and the absence of all signs of inflammation.

Varieties of Innocent Tumours.

Osteoma or *exostosis* is the commonest form (*Fig*. 281). The usual site is in the neighbourhood of the epiphyseal line of a long bone, particularly the lower end of the femur; the tumour is capped with cartilage, and often surmounted by an adventitious bursa containing fluid.

The ungual phalanx of the great toe is another common site for a similar tumour, which pushes up the nail and may be very painful if ordinary boots are worn. Multiple exostoses are not uncommon, and they may be hereditary. Diagnosis can be made at once by means of a skiagram, and with this aid it can be seen that the swelling is composed of cancellous tissue continuous with that of the bone. A spurious osteoma may arise by ossification of a tendon or by an extension of the ridge into which the tendon is inserted, the condition being generally consequent on some injury or repeated strain, as in the case of horse riders, who are apt to develop one on the inner aspect of the knee; another example is the spur that is apt to form on the under surface of the os calcis at the posterior end of the long plantar ligament, giving rise to much discomfort and even acute pain below the heel in walking. A skiagram may be required in establishing the diagnosis with certainty (*Fig.* 185, p. 440).

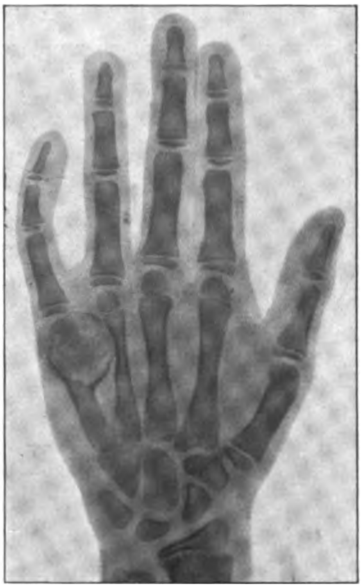


Fig. 282.—Skiagram of an enchondroma of the fifth metacarpal bone. Compare with *Figs.* 284, 286, 287. Enchondroma and sarcoma do not appear unlike in skiagrams.
(*Skiagram by Dr. Hugh Walsham.*)

Ivory exostoses may be found on the flat bones of the skull, or in the auditory meatus growing from the petrous bone, or causing unilateral exophthalmos if springing from the orbital plate of the frontal bone or the walls of the frontal sinus.

Chondromata may grow from any bone. They are most commonly multiple, affecting the phalanges and metacarpal bones of the hand (*Fig.* 282). The result is increasing deformity, with pain and ulceration of the skin.

Fibromata grow from the fibrous tissue of the periosteum, but are rare except in the form of an epulis of the jaw. (See SWELLING OF THE JAW, LOWER, p. 683.)

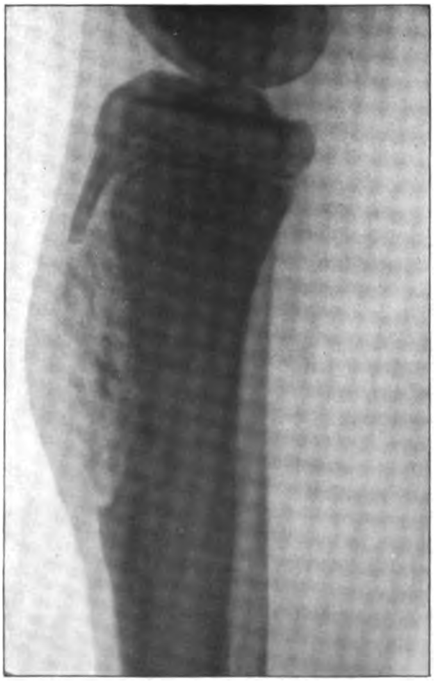


Fig. 283.—Skiagram of a periosteal sarcoma of the tibia.
(*Skiagram by Dr Hugh Walsham.*)

Lipomata are extremely rare. They grow from the outer layer of the periosteum.

Malignant Tumours may occur either primarily (sarcoma), or secondarily by metastasis or by invasion (sarcoma and carcinoma).

Periosteal sarcomata are of so many types, and of such varying degrees of malignancy, that it is a difficult task to lay down any rule as to their characteristics. The softer their consistency and the nearer they approach to the embryonic type of the tissue, the more malignant they are; the nearer they reach the fully-formed tissues and contain cartilage, bone, or fibrous tissue, the slower growing and less malignant they are. A typical case may be represented as a rapidly growing tumour, generally about the end of a long bone (*Fig.* 283). It is not usually painful, and the signs of local inflammation and general fever are little marked, or absent. The patient is commonly a young adult, who often gives a history of injury to the part, and may lose weight and strength before actual cachexia sets in. The veins over the swelling become prominent, the lymphatic glands enlarged, and metastases by the blood-stream occur early, especially in the lungs. It has to be distinguished from chronic and syphilitic periostitis. If a skiagram is insufficient, a piece of the tumour may have to be excised, decalcified, and a microscopic section from it prepared. This form of sarcoma is the worst possible, and seeing that amputation does not cure, and often does not prolong life, this extreme resource may be delayed where either gumma or chronic periostitis is still a possible diagnosis.

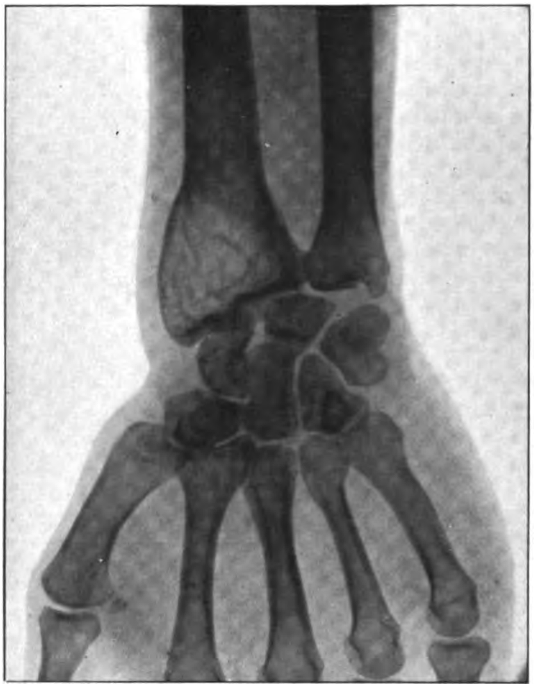


Fig. 284.—Skiagram of an early myeloid sarcoma of the lower end of the radius. This figure and *Figs.* 286, 287 should be compared with *Fig.* 282, which shows that enchondroma and sarcoma are not distinguishable by *x*-rays alone.
(*Skiagram by Dr. Hugh Walsham.*)

Endosteal or *myeloid sarcomata* are of much slower growth ; so slow are they that some pathologists are inclined to denote them as benign tumours. They are prone to affect the ends of the long bones, particularly the lower end of the femur, the upper end of the tibia, the upper end of the humerus, the lower end of the radius (***Figs.*** **284, 286, 287**), the sternal end of the clavicle, and the upper jaw (malignant epulis). Attention is first called to the part by pain ; then a more or less uniform swelling appears. This is at first bony hard, and only as the shell of bone yields does softening occur, or crackling on pressure. The lymphatic glands are not enlarged, and metastases do not occur. In the early stages, diagnosis has to be made from rheumatism and chronic abscess, and later from chronic osteomyelitis and periosteal sarcoma ; it is easily made by the aid of *x*-rays as a rule, but it is most important not to mistake the callus that is produced after fracture for a sarcoma ; this mistake is not always obviated even by the use of the *x*-rays, unless the latter reveal the line of fracture as well as the callus around it.

Sarcoma may also invade the bones from outside, having started in the subcutaneous or deeper soft tissues outside the bone (***Fig.*** **285**).

Carcinoma is always secondary (***Fig.*** **62, p. 152**). Squamous-celled carcinoma may spread to the tibia from an epitheliomatous ulcer of the leg, or to the jaw from the lip or floor of the mouth. It is mostly spheroidal-celled carcinoma which infects bone by metastatic growths, particularly from the breast or from the thyroid gland. A swelling of bone may be found, but this is rarely discovered until attention is called to it by a spontaneous fracture.

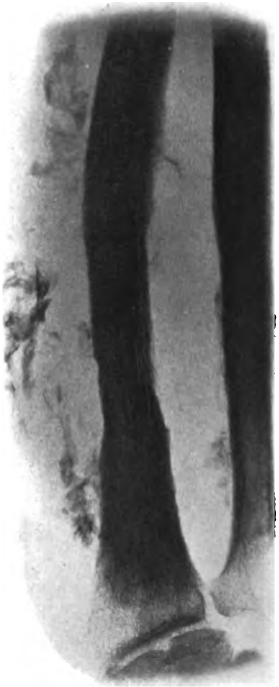


Fig. 285.—Skiagram of a sarcoma of radius and ulna : the growth surrounds the bones and is eating into them : it was not primarily a bone growth. Male, age 28.
(*Skiagram by Dr. C. Thurstan Holland.*)

V. CYSTS.

Blood cysts are found in degenerating sarcomata.

Hydatid cysts are uncommon in this country. They affect the diaphyses of the long bones, converting the shaft into a thin-walled tube, which undergoes spontaneous fracture. They would hardly be suspected unless there were known hydatid disease elsewhere, especially in the liver (p. 375).

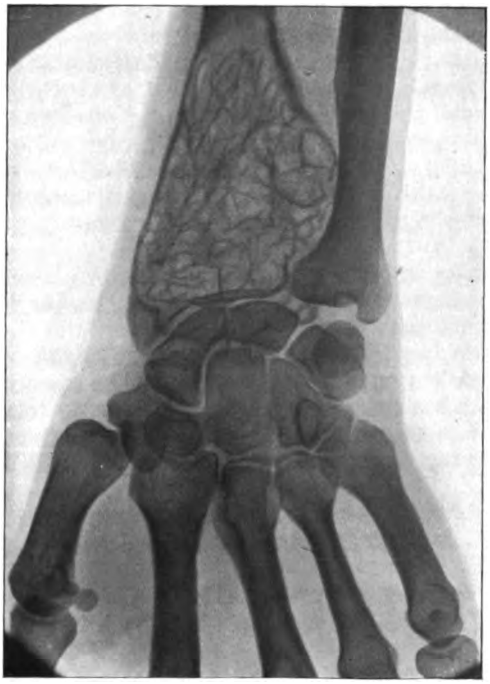


Fig. 286.—Skiagram giving the antero-posterior view of a myeloid sarcoma of the lower end of the radius. The growth is at a later stage than that depicted in *Fig.* 284.

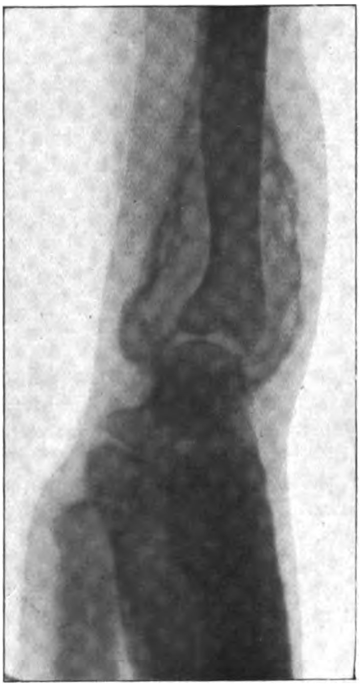


Fig. 287 shows the same growth as *Fig.* 286, but seen from the lateral aspect.
(*Skiagrams by Dr. Hugh Walsham.*)

Cysts of the jaw, or *dentigerous cysts*, are considered in the article on SWELLING OF THE LOWER JAW (p. 683).

George E. Gask.

SWELLING OF THE FACE.—In this article are included only swellings of the skin and subcutaneous tissues. Malignant and other diseases of the facial bones, etc., are considered under SWELLING OF THE JAW (p. 683), and SWELLING ON A BONE (p. 667). SWELLING OF THE SALIVARY GLANDS is discussed on p. 694. Contusions and injuries to the face are so obvious that they need no mention. The remaining swellings will be classified as (1) *Non-inflammatory*; and (2) *Inflammatory*.

Non-Inflammatory Swellings.

Renal and Cardiac Œdema.—If the whole face is puffy and the eyelids are œdematous, the urinary and cardiac systems are to be examined for disease. For swelling due to *obstruction of the superior vena cava* by mediastinal fibrosis, aneurysm, or new growth, see ŒDEMA (p. 411), and VEINS, VARICOSE THORACIC (p. 750).

Angioneurotic Œdema is a disease characterized by the occurrence, sometimes periodical, of local œdematous swellings, more or less limited in extent and of transient duration. It is not confined to the face, but the eyelid is a common situation (*Fig.* 178, p. 412), and also the lips and cheek. It may be simulated closely by *urticaria* following the taking of fish or pork; or a somewhat similar effect produced by some drugs, notably by aspirin in certain patients.

Tumours are not common. They may be fibroma, lipoma, epithelioma, or sebaceous cyst.

Inflammatory Swellings.—Often the cause is obvious: for instance, a *boil, carbuncle,* or *suppurating wound*; or the 'blubber-lips' that result from chronic *lymphangitis.*

Erysipelas is prone to occur on the face. It is marked by a vivid red œdematous swelling, associated with fever. The redness tends to spread, the edges being raised and well defined from the healthy skin. The œdema may be continuous, or it may disappear in one place and re-appear in another. In the very severe cases the fever is high, rigors occur (*Fig.* 245, p. 568), the cuticle may be raised in blebs, and sloughing may ensue.

Alveolar Abscess and *Dental Caries* are fertile sources of facial swelling, also abscess in the nasal sinuses. (See SWELLING OF THE JAW, p. 683.)

Anthrax chiefly affects operatives in wool and horse-hair factories and workers of raw hides. The disease is characterized by the formation of a vesicle, which bursts, forms a scab, and then becomes surrounded by a ring of vesicles, and around this is an area of œdema. The diagnosis is made by the microscope. A drop of fluid from one of the vesicles contains large, square-ended, Gram-staining bacilli, which have a characteristic growth on culture media.

Vaccinia.—An accidental infection about the face may be mistaken for an anthrax pustule. If inquiry into the attendant circumstances is not sufficient to exclude the graver disorder, a bacteriological examination should be made.

Primary Syphilitic Sore, if found on the face (*Fig.* 23, p. 73), is generally situated on the upper lip. It is not so indurated as when on the glans penis, but the surrounding œdema is more marked, and the neighbouring lymphatic glands are considerably enlarged. The condition is often missed because it is not expected. An absolute diagnosis can be made by finding the spirochætæ in the serum discharged from the ulcer (*Plate XXVIII, Fig. J,* p. 614), and by Wassermann's test.

Insect Bites—from mosquitoes, gnats, bees, etc.—often cause large, lumpy, irritating swellings. The only difficulty in diagnosis is when they become infected with pyogenic organisms.

The various skin diseases which may be associated with swelling of the face are considered under PUSTULES (p. 557); VESICLES (p. 753); WHEALS (p. 771); ETC.

George E. Gask.

SWELLING, FEMORAL.—By the femoral region is meant Scarpa's triangle. It is very easy to define on paper what is a femoral swelling, but in a fat patient it may be very difficult. The two great landmarks which, with care, can always be made out, however fat the patient, are the spine of the pubes and the anterior superior spine of the ilium; a line joining these two points and curving slightly downwards separates the inguinal from the femoral region, and indicates Poupart's ligament. Mistakes are often made, especially in fat people, because a horizontal crease in the thigh which lies below—sometimes as much as two inches below—is mistaken for the ligament. The first point in making the diagnosis is to decide definitely that the swelling is femoral, and then to decide its nature.

It may be obvious at once what the swelling is: for instance, a well-marked acute abscess, with redness and œdema of the skin and an undoubted source of infection, such as a sore toe; or, a rare occurrence, an aneurysm of the femoral artery, showing expansile pulsation. Supposing, however, the signs are not so clear, the various conditions may be classed broadly under two heads: (1) *Swellings that are reducible and give an impulse on coughing*; (2) *Swellings that are irreducible and do not give an impulse on coughing.*

Reducible Swellings with an Impulse are: (*a*) Femoral hernia—reducible; (*b*) Saphena varix; (*c*) Psoas abscess. All these give an impulse on coughing; are, or may be, reducible on pressure; may disappear on lying down and reappear on standing. How then is one to distinguish between them?

(*a*). *Femoral Hernia* (*reducible*).—The sex of the patient is no real guide, for though it is more common to find a femoral hernia in a woman than in a man, this is not sufficient to base the diagnosis on. Before puberty it is rare in either sex. A femoral hernia leaves the abdomen through the femoral canal and turns directly forward, forming a tumour in the upper and inner part of the femoral region; then, following the line of least resistance, it turns upwards, extending often above Poupart's ligament, thus simulating an inguinal hernia. More rarely, the hernia extends downwards along the femoral vessels. Its course must be remembered in attempting to discover whether the swelling is reducible. If it is large and contains intestine it will be resonant, and a gurgling may be heard or felt on reduction, distinguishing it at once from all other femoral swellings. If it is reduced and the finger held over the femoral aperture, the hernia will be felt projected forcibly against the finger when the patient is asked to cough. If a swelling is complained of, and none is found even on standing and straining, it is suggestive of femoral hernia with only occasional descent, and the patient should be examined at another time after exercise.

(*b*). *Saphena Varix* is a localized dilatation of the saphenous vein at the saphenous opening, immediately before it joins the femoral vein. It may easily be confounded with a femoral hernia, for it forms a swelling in the ordinary position of a femoral hernia, it disappears on lying down, reappears on standing, and gives an impulse on coughing. A little care, however, should suffice to distinguish the two. The impulse is quite different—in a saphena varix it is more in the nature of a thrill, such as may be felt in a varicocele or in big varicose veins in the leg. If, while the patient is standing, a finger is pressed on the swelling, it collapses gradually, and as the finger is withdrawn the swelling follows, regaining its shape like an air-ball, whereas a hernia comes out with a pop. A saphena varix is almost always associated with varicose veins in the leg, though, owing to the persistence of valves, none may show between the knee and Scarpa's triangle.

(*c*). *Psoas Abscess.*—The need to differentiate between this and the two conditions mentioned above exists only when the abscess has extended from the iliac region, has passed under Poupart's ligament and the femoral vessels, and is pointing in the inner part of Scarpa's triangle. There is an impulse on coughing and the swelling is reducible ; but another swelling is to be found above Poupart's ligament, and fluctuation is to be obtained between the two. Conclusive proof can be found by an examination of the back. This should be made with the patient standing and the whole length of the back and the hips exposed. An undoubted angular kyphotic curve may be seen at once, or, if that is not present, there may be rigidity and impaired movement denoting some disease on the anterior surfaces of the bodies of the vertebræ.

Irreducible Swellings without Impulse: (*a*) Femoral hernia—irreducible ; (*b*) Lymphatic glands — inflammatory or malignant ; (*c*) Primary tumours — lipoma, fibroma, sarcoma ; (*d*) Ectopic testis.

(*a*). *Femoral Hernia.*—The irreducibility may be accounted for in four ways: (i) Strangulation ; (ii) A piece of omentum adherent to and plugging the neck ; (iii) An empty sac, but a mass of extraperitoneal fat round it ; (iv) A hydrocele of the sac.

If strangulation has occurred there will be the signs of intestinal obstruction, viz., vomiting and constipation. It must be remembered that the swelling may be but a small one, and when the patient is very fat it may be missed.

It is usual to find around the sac of a femoral hernia a quantity of extraperitoneal fat, even in a thin person, and it is quite impossible to say without dissection whether the swelling is due to a plug of omentum inside the sac or to a collection of fat outside it.

A hydrocele may be formed as a result of a long-standing hernia into which there has been no descent of bowel or omentum, and in which the communication with the general peritoneal cavity has become constricted or closed. The sac may then become cystic and filled with fluid. The feeling of fluctuation may be obtained in the swelling, though it is often only on dissection that the exact nature of the condition is revealed. It is to be noted that in all cases of hernia the swelling is single, and that though it may be movable in some directions, it is always tied down by its neck to the aperture of the femoral canal.

(*b*). *Enlarged Glands* may be : (i) Inflammatory ; (ii) Malignant (see p. 381).

Chronically inflamed glands may be hard to differentiate from a small irreducible femoral hernia. The whole limb is to be examined to see whether there is any possible source of infection, and the whole patient to see whether there is a general enlargement of the glands, as in lymphadenoma. The chief distinguishing feature between the two conditions is that femoral hernia forms only one swelling, whilst it is very rare for only one gland in its group to be picked out by an infecting agent, and not the others. Therefore, if there is more than one swelling the chances are that these are glands. Perchance both conditions are present, a femoral hernia and enlarged glands : a very difficult combination unless the femoral hernia happens to be reducible or gives an impulse on coughing. In such a case an attempt should be made to feel the neck of the sac running up to the femoral canal.

(*c*). *Primary New Growths* are rare in this situation. They may be lipoma, fibroma, or sarcoma. The innocent tumours are noted for their free mobility in all directions. A primary sarcoma is diagnosed rather by exclusion and by its malignant characteristics.

(*d*). *Ectopic Testis.*—One of the places into which a testis may be drawn abnormally is Scarpa's triangle, which it reaches by passing over Poupart's ligament. The facts that the swelling has the shape of the testis, though generally smaller than normal, and that the corresponding half of the scrotum is empty, make the diagnosis easy.

Mention may be made here of those swellings which are neither truly femoral nor truly inguinal, but betwixt and between, and bulge Poupart's ligament forwards. They are generally deep, and on that account obscure. They may be due to :—

1. Distention of the hip-joint, as in tuberculous disease of the hip.
2. Distention of the bursa between the tendon of the ilio-psoas muscle and the capsule of the hip-joint. If large, the swelling may be quadrilateral in shape, and owing to its sensitiveness to pressure the leg is kept in the position of greatest ease, i.e., slightly flexed, abducted, and externally rotated. It is often difficult to distinguish from psoas abscess or from distention of the hip-joint, with which, indeed, it often communicates. Diagnosis may be aided by puncturing the swelling with an aspirating needle.
3. Osteophytic outgrowths from the acetabulum in osteo-arthritis of the hip-joint.
4. A parametric abscess.

George E. Gask.

SWELLING IN THE ILIAC FOSSA (LEFT).—For the general method of examination, compare Swelling in the Iliac Fossa (Right).

Swellings connected with Structures normally present in the Left Iliac Fossa.

The Sigmoid Flexure cannot be felt normally. It becomes palpable as a cylindrical swelling if distended with fæces ; or if it is thickened, as it may be in chronic ulcerative colitis or congenital dilatation of the colon.

Carcinoma of the Sigmoid.—Next to the rectum the sigmoid colon is the most common seat of cancer in the bowel. If of the scirrhous or ring type no lump may be felt, and the condition may not be discovered until intestinal obstruction has supervened. When infiltrating the bowel widely, and especially when the tumour is undergoing colloid degeneration, a swelling forms which is most evident on bimanual examination after the bowels have been well cleared by enemata. If a lump can be felt in the sigmoid flexure of a middle-aged patient, the strong probability is that it is a carcinoma, and whether there are other clinical signs or not, the diagnosis should be made sure by actual inspection of the swelling by means of the sigmoidoscope, or even through an abdominal incision. It may be simulated by subacute inflammatory changes around an acquired pouch or diverticulum of the colon—*diverticulitis* ; this sometimes subsides by itself, and the course of the case serves to exclude carcinoma ; more often the symptoms call for operation, and the diagnosis is made by laparotomy.

Enlarged Lymphatic Glands.—The glands forming a chain round the external iliac vessels may be swollen as the result of pyogenic infection, which has spread up through the femoral lymphatics or from secondary deposit of some malignant growth starting either in the leg, the external genitals, or the pelvis. The enlargement is seldom very great ; the source of infection is usually obvious.

Aneurysm of the External Iliac Artery is very rare. It is recognized at once by its expansile pulsations.

Swellings connected with Structures not normally present in the Left Iliac Fossa.—These may be : Swellings coming down from above, extending upwards from the pelvis, or pushing forward from behind.

Swellings coming down from above.—The *Spleen*, if much enlarged, may reach even as far as the left iliac fossa. It is recognized by its rounded margin, and the notch on the inner edge. (See Spleen, Enlargement of the, p. 628.) *A kidney*, if freely movable, may be displaced as far as the pelvis ; on rare occasions it becomes fixed there by inflammation.

For swellings extending up from the pelvis and those pushing up from behind, see the article on Swelling in the Iliac Fossa (Right).

George E. Gask.

SWELLING IN THE ILIAC FOSSA (RIGHT).—It is not always easy to say whether there is or is not a definite swelling in the right iliac fossa, for it may be only small and deep, or be masked by abdominal rigidity or fat. In all cases a careful inspection of the abdomen is first to be made, the patient lying on the back with the whole of the abdomen and the lower thorax exposed. Most mistakes result from want of a complete examination, which cannot be made through a tiny gap in the clothes. Before even touching the abdomen, much may be made out by the use of the eyes, and the points to observe are: (1) The presence or absence of an obvious tumour; (2) Whether the abdominal muscles move freely; (3) The conformation, etc., of the tumour, should one be present, and whether it moves on respiration.

Palpation is then to be employed, and this method will go a long way to elucidate the complaint, for it will be recognized at once whether there is a well-defined swelling, such as a carcinoma of the cæcum; or an indefinite swelling, such as is common in appendicitis. Distention with wind or an accumulation of fæces may cause a considerable swelling, but any doubt as to these may be cleared up by administering an enema. If the swelling persists, the questions that arise are: is it connected with one of the structures normally present in the right iliac fossa, especially the cæcum or the appendix; is it springing from the bone; is it arising from some organ invading this space, for instance the uterus or its appendages, the right ovary, or the bladder; or from some structure displaced downwards such as the gall-bladder, stomach, or right kidney?

A.—SWELLINGS CONNECTED WITH STRUCTURES NORMALLY PRESENT IN THE RIGHT ILIAC FOSSA.

The Appendix.—*Appendicitis* is so common that it is put first. Most well-marked attacks of appendicitis are associated at some period with a swelling, though in the acutest and gravest forms the latter may be absent. The appendix itself, even if swollen and thickened, can rarely be felt by palpating the abdomen, and the swelling is due to paralytic distention of the cæcum, local œdema, or the formation of an abscess. The chief indications of appendicitis are: pain, tenderness, local rigidity, and swelling in the right iliac fossa, associated with a furred tongue, vomiting, constipation, an increase of the pulse-rate, and a rise of temperature. Usually there is also diminished muscular movement in the lower part, or it may be over the whole, of the abdomen. Any movement is painful, and in order to relax the tension the patient lies with the right leg drawn up. A rectal examination should be made, for a bulging abscess may be felt by this route. Micturition is frequently abnormal, with a tendency to be either painful or unduly frequent. A leucocyte count is of great service, for in almost every case of acute appendicitis the number of white cells is increased.

Tuberculosis of the Cæcum or of the Lymphatic Glands in the neighbourhood of the Cæcum.—This is not nearly so common as appendicitis, but is not so rare as is often imagined, and when it does occur it is frequently mistaken for appendicitis; it may be only after the abdomen has been opened that the mistake is discovered. The glands become enlarged and painful, and there may be some local peritonitis over them which makes the diagnosis very difficult. Usually there is some other tuberculous focus about the patient, especially in the lungs, which should be examined with particular care, the *x*-rays and sputum analysis not being omitted. If doubt exists recourse may be had to a diagnostic injection of Koch's old tuberculin, and the opsonic index may be taken both before and after abdominal massage. Von Pirquet's test is not very trustworthy.

Actinomycosis starting in the cæcum and appendix is another inflammatory condition which may cause a swelling and give the signs of a chronic abscess. The diagnosis can only be made with certainty by an incision and the finding in the pus of the characteristic yellowish granules (occasionally black—the gunpowder variety), and the recognition under the microscope that these granules are formed of a Gram-staining streptothrix (*Plate XXVIII*, p. 614).

Carcinoma of the Cæcum gives rise to a swelling which occasions few symptoms, unless the passage of fæces is affected and intestinal obstruction results. It is important, however, from the point of view of treatment that an early diagnosis be made. The presence of a non-inflammatory swelling of long standing in the right iliac fossa, with a history of wasting, is very suggestive of a carcinoma, and early recourse should be had to the only sure diagnostic method, namely, laparotomy. Very rarely is there passage of blood or mucus by the bowel to help one.

Intussusception usually occurs in children, especially during the latter half of the first year of life, and its presence is indicated by the signs of intestinal obstruction, namely, vomiting and constipation, and by the passage of blood and mucus by the rectum. The intussuscepted portion may be palpable, and in some cases it lies in the right iliac fossa, though more frequently in the right hypochondrium. Chronic intussusception may also cause a swelling which generally baffles diagnosis, and is commonly mistaken for an enlarged kidney.

Aneurysm of the Iliac Arteries is very rare, but it is generally easy of diagnosis by reason of the expansile pulsation of the tumour.

B.—SWELLINGS CONNECTED WITH STRUCTURES NOT NORMALLY PRESENT IN THE RIGHT ILIAC FOSSA.

These may be: Swellings coming down from above, swellings extending upwards from the pelvis, or swellings pushing forward from behind.

Swellings coming down from above.

The liver, or an enlarged or abnormal lobe of the liver—Riedel's lobe (p. 366)—is sometimes very deceptive. The facts that there are few symptoms, that the mass moves on respiration and is continuous with the liver, and that there is no intervening area of resonance between it and the liver, should assist the diagnosis; but cases are not infrequently mistaken for enlargement of the gall-bladder. A *suppurating gall-bladder* has been opened in the right iliac fossa under the mistaken diagnosis of appendix abscess, for there is often no jaundice in these cases. Laparotomy may be the only means of certain diagnosis.

The kidney, if unduly movable, may be displaced and come to lie in the right iliac fossa. It is recognized by its shape and free mobility. A tumour of the kidney or a large hydro- or pyo-nephrosis may also invade the upper part of this fossa.

Carcinoma of the Stomach—with extreme distention of the stomach. It is a surprising fact that the stomach may be so distended as to enable the pylorus to lie in the right iliac fossa. The history of copious vomiting, the wasting, the distention of the stomach, and examination with the *x*-rays after the administration of bismuth make the diagnosis easy.

Swellings extending upwards from the Pelvis, and attached to the uterus and its appendages, can usually be felt dipping into the pelvis; vaginal and rectal examinations will assist the diagnosis; and there are symptoms, such as disturbances of menstruation, indicating their nature. Such swellings might be a large *fibroid of the uterus*, a laterally-placed *ovarian cyst*, *pregnancy*, an *abscess* extending from the broad ligament, or a *pouch of a bladder* distended from obstruction to the urethra.

It happens not infrequently that there may be difficulty in determining between an inflamed appendix and an enlarged and tender ovary, particularly when the attacks of pain are coincident with the menstrual periods. These generally turn out to be due to the appendix, though both may be implicated, the appendix having become adherent to the ovary or tube.

Pyosalpinx is easily confused with appendix abscess; the fact that it is associated with vaginal discharge, or is subsequent to parturition, puts one on the right track. Vaginal examination is essential in these cases.

Swellings pushing forward from behind.

These may be solid, such as *sarcoma* or *chondroma* of the pelvic bones. Here the tumour will be immovable apart from the pelvis, and a skiagram makes the condition clear. If the swelling is fluid it may depend on *suppurative osteomyelitis* of the ilium; or on a *tuberculous* affection of the ilium, either primary, or secondary to hip or sacro-iliac joint disease; or on *tuberculosis*, *necrosis*, or *suppuration of the lumbar vertebræ*.

If the swelling cannot be attributed to any of the causes mentioned above, it is to be remembered that a wandering organ, such as a spleen or kidney, may find its way into the right iliac fossa. Rarities such as hydatid cysts of the peritoneal cavity are met with so seldom that they merit no more than mention.

George E. Gask.

SWELLING, INGUINAL.—A variety of swellings may appear in the groin, and be very difficult to differentiate. The following are some of the most important:—(1) *Enlarged glands*: (*a*) inguinal; (*b*) femoral; (*c*) iliac. (2) *Abscess, acute or chronic.*

(3) *Hernia* : (*a*) inguinal ; (*b*) femoral ; (*c*) obturator. (4) *Retained testicle.* (5) *Hydrocele.* (6) *Tumours of the cord or round ligament.* (7) *Aneurysm and other vascular swellings.* (8) *New growths.* (9) *Distended psoas bursa and other cysts.*

1. **Enlarged Glands.**—There are two chief groups of glands in the groin. The most commonly affected are the *inguinal*, which lie in the subcutaneous tissues about Poupart's ligament, and drain the external genitals, the anus, the umbilicus, the lower parts of the abdomen and back, the buttock and the upper third of the thigh. The *femoral* glands rest below the saphenous opening and drain the lower limb below the upper third of the thigh. It must be remembered, however, that the lymphatic drainage is somewhat erratic, so that a sore toe may sometimes induce enlargement of an inguinal gland only. The *iliac* glands drain the inguinal and femoral set, and consequently often enlarge secondarily to these ; but they also communicate freely with the abdominal lymphatics and may become infected from them.

Enlarged glands in the groin are nearly always multiple, and usually subcutaneous, so that they are easy to recognize as glands ; but a solitary one adherent to the saphenous opening may be almost impossible to distinguish from an irreducible omental femoral hernia or a hydrocele of a hernia sac.

The iliac glands just above Poupart's ligament are more difficult to palpate, because they lie deep to the abdominal muscles, but their enlargement is generally secondary to disease of the superficial glands, and this often gives the key to the diagnosis of an obscure swelling in this region.

Some Causes of Enlargement of the Groin Glands.—(*a*) Mechanical or chemical irritation ; (*b*) Septic infection, for instance from genital sores or from sores on the toes or legs ; (*c*) Tubercle ; (*d*) Syphilis ; (*e*) Other specific diseases, such as rubella and bubonic plague ; (*f*) Lymphadenoma ; (*g*) Lymphatic leukæmia ; (*h*) Malignant diseases : secondary carcinoma ; secondary or primary sarcoma.

(*a*). The glands become slightly enlarged and tender as a result of the *mechanical irritation* of a truss, and more frequently the *bites of parasites* such as the *Pediculus pubis.* The glands generally remain movable, and they rarely suppurate.

(*b*). *Septic infection* may follow insect bites ; but more commonly a septic sore or recent scar can be discovered upon examination of the area drained by the glands. Septic glands either soon subside or cease to be tender after the removal of the source of infection, or they enlarge rapidly, become adherent, and suppurate within three or four weeks of their first enlargement.

(*c*). This, and the amount of inflammation of the skin over them, distinguishes septic from *tuberculous* glands which do not suppurate for some months, and then with but little inflammatory reaction. Epitheliomatous glands may suppurate towards the end.

(*d*). The true *syphilitic gland* is hard, movable, and only moderately enlarged, and the existence of the indurated chancre usually makes the diagnosis easy. The *Spirochæta pallida* may be detected, or Wassermann's serum test may be positive ; but a negative reaction is not conclusive. It must not be forgotten that, as an apparently soft sore (septic) may later become hard and definitely syphilitic ; therefore suppuration of a bubo does not disprove syphilitic infection. Instances of mixed infection by sepsis and syphilis are fairly common.

(*f*). In *lymphadenoma* the groin glands are rarely affected alone, and the smooth, soft enlargement of many glands without signs of inflammation, associated with increasing anæmia and intermittent pyrexia (*Fig.* 247, p. 570), makes the diagnosis fairly easy. The spleen may be affected at the same time.

(*g*). Blood examination will give pathognomonic results in cases of lymphatic leukæmia (p. 25).

(*h*). *Malignant disease* of the groin glands is nearly always epitheliomatous, and secondary to a primary epithelioma of the skin or mucous membrane in the area drained by the glands. The primary growth, especially at the anus, may be very small, and the patient may be unaware of its existence. The other main points in distinguishing epitheliomatous glands are their exceeding hardness ; their progressive but slow growth ; their early adhesion to the deep fascia and skin ; and the amount of pain to which they give rise without signs of inflammation. Late in the disease they may suppurate or slough, with severe hæmorrhage. Intra-abdominal carcinoma, especially of the ovary or colon, sometimes causes enlargement of the inguinal glands.

Sarcoma of the groin glands is rare ; it may be primary or secondary. Usually these are not the only glands affected. They grow with great rapidity and remain smooth and fairly soft until they attain a great size, when they may fungate through the skin. They are distinguished from lymphadenoma by their very rapid growth and the absence of pallor until late in the disease. Melanotic growths of the skin give rise to rapidly growing smooth glands, whose pigment may be visible through the skin. The primary growth or ulceration in connection with the skin, particularly of a toe, may not show pigmentation, and its serious import may thus be overlooked.

2. **Abscess.**— (*a*). *Acute.* The only common cause of acute abscess in the groin is suppuration of the glands, and a search must always be made for a primary source of infection, especially about the genitals. A hernia may occasionally suppurate, and an appendicular abscess may point just above Poupart's ligament ; but there is then a history of the characteristic symptoms of appendicitis, and the pus when released has the suggestive smell of the products of the *Bacillus coli communis.* Both tuberculous and epitheliomatous glands may become acutely inflamed and suppurate.

(*b*). *Chronic abscess* here may be due to caries of the sacro-iliac joint or to hip disease, or it may arise from tuberculosis of the superficial or deep glands. Psoas abscess, due to caries of the spine, is distinguished by fluctuation from the loin to the groin, and often bimanually, above and below Poupart's ligament, external to the femoral vessels. There are also some tenderness and rigidity, and often deformity of the lumbar or lumbo-dorsal spine. Iliac abscess does not extend up into the loin, and is placed farther out than psoas abscess. Moreover, there may be pain and tenderness over the sacro-iliac joint, and a limping gait. In hip disease, especially in children, the floor of the acetabulum may give way, and an abscess may thus enter the true pelvis, whence it often ascends and becomes palpable above Poupart's ligament. The diagnosis of the cause is easy from the well-marked signs of hip disease.

3. **Hernia.**—In examining swellings in the groin, hernia must always be considered. Three chief varieties occur here : inguinal, femoral, and very rarely obturator hernia. A hernia gives an impulse on coughing, but so do psoas abscess, psoas bursa, and a saphena varix. All these may also be reducible like a hernia. A psoas abscess presenting below Poupart's ligament has been mistaken for a femoral hernia ; but it is distinguished by its position—external instead of internal to the femoral vessels. Moreover, it is dull on percussion, whereas a hernia is resonant except when it contains omentum alone. Psoas bursa is also placed outside the vessels. A saphena varix has often been mistaken for femoral hernia ; but it can be distinguished from the latter easily because it returns after complete reduction, even though the finger is kept pressed against the femoral canal. It is not always easy to distinguish the three herniæ which occur in the groin, but close attention to the following points usually leads to a correct diagnosis. An inguinal hernia is both seen and felt to be *above* the fold of the groin and *above* Poupart's ligament ; whereas a femoral hernia is seen and felt to be *below* the fold of the groin and *below* Poupart's ligament. It is to be remembered that when a femoral hernia becomes very large and loculated, it generally extends upwards and inwards over Poupart's ligament. Still, the bulk of it remains below the fold of the groin in the upper and inner part of the thigh. An inguinal hernia often extends into the scrotum or labium ; a femoral hernia never does this. The neck of an inguino-scrotal hernia is above and internal to the spine of the pubis, whereas the neck of a femoral hernia is below and external to this bony prominence. Inguinal hernia is most easily reduced by pressure directed upwards, backwards, and outwards, whereas a large femoral hernia is most easily reduced by pressure directed at first backwards and downwards, and then directly upwards. In difficult cases it is a good plan to reduce the hernia, then to get the patient to stand up, while the surgeon makes firm pressure over the internal ring and asks the patient to cough. A femoral hernia may then come down, but not an inguinal. Similarly, pressure can be made on the femoral canal ; this prevents the descent of a femoral hernia, so that if the swelling now returns it is inguinal. In this connection it may be well to remember that femoral hernia is rare in males, and also in all females under maturity. The prevalent belief that femoral hernia is more common than inguinal in grown-up women is wrong, inguinal being more common at all ages and in both sexes. It is excessively difficult to differentiate between an irreducible femoral hernia containing omentum and an enlarged gland at the

saphenous opening or in the femoral canal. A hydrocele of a hernial sac gives rise to the same difficulty, and sometimes an exploration becomes necessary on account of the danger of overlooking femoral hernia, and the risk of strangulation.

The diagnosis between femoral and obturator hernia is not very difficult; it is far more common to overlook an obturator hernia altogether. When an external swelling is caused by an obturator hernia, it is placed farther inwards, and it is more vague than a femoral hernia. Moreover, there is pain shooting along the inner side of the thigh, and generally the signs and symptoms of strangulation. Further, a tender swelling can be felt at the obturator foramen upon vaginal or rectal examination.

The two chief varieties of inguinal hernia, the oblique and the direct, are usually distinguished quite easily. Direct hernia is rare, and is more globular in shape than the indirect or oblique hernia; the spermatic cord is antero-external to it, and postero-internal to the ordinary oblique hernia. Direct hernia is placed a little farther in and higher up than the oblique. It is generally much more easily reduced, but returns again with striking abruptness when the patient coughs. It rarely travels into the scrotum, and it is uncommon before the age of thirty. There is often a history of sudden onset after some violent straining effort.

4. **Retained Testicle.**—The most important points in the diagnosis of this condition are the absence of the organ from its proper place, and the presence of a swelling about the inguinal canal. Occasionally, the testicle may be maldescended, or after leaving the external ring may have found its way into the upper and inner part of the thigh, where it simulates a femoral hernia, or into the perineum. The swelling in the groin may give the characteristic testicular sensation, or the condition may be associated with attacks of pain which have been mistaken for appendicitis or intestinal colic. It is practically always accompanied by actual or potential hernia into the tunica vaginalis, which is in direct communication with the abdominal cavity.

5. **Hydrocele.**—The neck of the sac of either a femoral or an inguinal hernia may become obstructed, and a hydrocele of the sac may then develop. This may become inflamed and give rise to considerable difficulty in diagnosis. Strangulated or irreducible omental hernia may be simulated, and sometimes an exploration is the only way of settling the diagnosis. It is more easily distinguished from strangulated hernia containing bowel, because it is dull on percussion, and the bowels are not obstructed. An encysted hydrocele of the cord occupying the inguinal canal is sometimes difficult to distinguish from inguinal hernia; but it is not completely reducible, and it is dull on percussion. It is not granular like an omental hernia, and it can even be shown, with some difficulty, to be translucent. Like a hernia, it gives an impulse on coughing.

6. **Tumours of the Cord or Round Ligament.**—The only common tumours of these structures are (*a*) Lipoma and (*b*) Fibromyoma of the round ligament. The former is so soft and displaceable that it gives an impulse on coughing, and is often mistaken for an omental hernia, especially in stout patients. The latter is hard and smooth, somewhat simulating the ovary or a thick-walled hydrocele of the canal of Nuck, for either of which it may be mistaken, a certain diagnosis only being possible by exploration.

7. **Aneurysm and other Vascular Swellings.**—Aneurysm of the external iliac artery may be mistaken for a vascular sarcoma arising from the pelvis. It can generally be recognized by the classical signs of aneurysm, such as expansile pulsation, bruit, weakening and delay of the corresponding femoral pulse, and marked reduction of the size of the swelling as a result of pressure on the common iliac artery. Saphenous varix has been referred to above.

8. **New Growths.**—Sarcoma of the pelvic bones or of the soft parts in this neighbourhood is hardly altered in size by pressure upon the common iliac artery, nor does it give such a loud bruit or the *expansile* pulsation, which are characteristic of aneurysm. The *x*-rays may give evidence which is valuable in distinguishing aneurysm from sarcoma.

9. **Distended Psoas Bursa** may give rise to pulsation communicated from the external iliac artery. On careful examination it can be distinguished by the absence of the classical signs of aneurysm already mentioned, by its translucency and irreducibility. There may also be signs of osteo-arthritis of the hip-joint. *R. P. Rowlands.*

SWELLING, INGUINO-SCROTAL.—The most important swellings which occupy both the inguinal and scrotal regions are :—(1) *Hernia* ; (2) *Varicocele* ; (3) *New growth* ; (4) *Hydrocele* ; (5) *Lymphangioma.*

Hernia is by far the most common, and when it is reducible there is very little difficulty in the diagnosis. It gives the characteristic impulse on coughing, is resonant on percussion, and when it contains bowel it gurgles on reduction. When it contains omentum only, the diagnosis is more difficult. To distinguish it from a *varicocele* it is only necessary to reduce the swelling and then to place the finger firmly upon the inguinal canal ; a varicocele returns in a few seconds, but a hernia does not. Moreover, an omental hernia has a granular feel which distinguishes it from varicocele. An irreducible omental hernia is distinguished from varicocele by its irreducibility ; but it may be confused with a very rare condition, lymphangioma of the cord. An irreducible hernia may be confused with encysted hydrocele of the cord. When a hernia contains bowel its resonance distinguishes it ; but when it contains omentum there is more difficulty. *An encysted hydrocele* or *a hydrocele of a hernial sac* is more even and elastic than an omental hernia, which is usually nodular. Moreover, it may be possible to show that a hydrocele is translucent. This help is not available when the cyst is deep or contains blood, which it occasionally does as the result of injury or strangulation of the omentum at the neck of the hernial sac. A *strangulated hernia* is distinguished from an inflamed hydrocele by the greater severity of the vomiting and other constitutional symptoms, and the completeness of constipation. Moreover, as stated above, a strangulated hernia containing bowel is resonant on percussion. Strangulated omentum may be very difficult to distinguish from an inflamed hydrocele or a hydrocele of a hernial sac, especially as either of these may complicate it. In such cases an exploration is the final appeal. It should not be forgotten that two or more varieties of inguino-scrotal swellings may co-exist. For instance, it is common to overlook a hernia which may complicate a varicocele, and this is especially true when the hernia contains only omentum. Again, it is quite common for a hydrocele of the tunica vaginalis or of the lower part of the cord to complicate an ordinary omental hernia. In such a case, a part of the swelling may be reducible, and, unless the patient is examined in the upright position, the upper part of the hernia may fail to appear during the examination. Again, the bowel may be reducible, while the omentum, being adherent, is not reducible, and may be mistaken for an encysted hydrocele of the cord. It is very important in all these cases to examine for translucency.

Growths of the testicle invading the inguinal region are, as a rule, easily diagnosed, because of the history and the observed course of the disease, and the general condition of the patient at the later stages. *Growth of the retained testis* may give rise to more difficulty ; it may be confounded at first with hydrocele of the tunica vaginalis, hydrocele of the hernial sac, or omental hernia, unless care be taken to ascertain if both the testicles are present in the scrotum. *Torsion of a retained testicle* with strangulation of its vessels has sometimes given rise to inguinal or inguino-scrotal swelling which has closely simulated strangulated hernia ; but although there may be much abdominal pain and local tenderness, vomiting is rarely so severe as in strangulated hernia, and the bowels are not really obstructed. Retained testicle is dull on percussion, and thus is distinguished from strangulated hernia containing bowel, and can only be confused with strangulation of the omentum.

The oblique hernia is the only common one to reach the scrotum. It may be acquired or congenital. In about one-tenth of the congenital herniæ the bowel and the testicle are in the same peritoneal sac ; in the great majority of congenital herniæ the two sacs are distinct, the testicle lying below the hernia. The same is true of acquired inguinal hernia. It is important to remember that nearly all inguinal herniæ descend into congenital or pre-formed sacs, and this is especially true of herniæ appearing apparently for the first time in young adults. In such cases, on careful inquiry, it may be found that a hernia existed and was apparently cured by a truss, in infancy. Again, it may be learned that the hernia reached the scrotum on its first descent, whereas acquired inguinal hernia very gradually develops as the result of straining in men past middle age. The swelling at first appears only in the inguinal region, and there the swelling increases in size, and extends into the scrotum only after some months or years. Very rarely, a direct hernia may reach the scrotum ; it is distinguished from oblique hernia by the fact that the cord is antero-external to it, instead of postero-internal, as in an oblique hernia. It may be possible in some cases to identify the contents of a hernia. Attention has been drawn above to the method of distinguishing the bowel from the omentum. Sometimes the appendix can be felt distinctly, especially in right-sided hernia. Occasionally the bladder may be identified, as in Astley Cooper's classical case. When the patient has apparently emptied the bladder, the surgeon reduces the hernia, and the patient is immediately able to pass more water. *R. P. Rowlands.*

SWELLING OF THE JAW, LOWER.—Swelling of the lower jaw may sometimes be mistaken for, or masked by, swelling of the cellular tissues in front of it. The real site of the swelling is first to be ascertained by opening the mouth and running the finger along the outer and inner borders of the mandible and comparing the two sides.

There are many causes for enlargement, and they may be subdivided under the following headings :—

1. *Injury.*
2. *Inflammatory affections.*
3. *Tumours*
    - Innocent—Fibroma, osteoma, and odontoma.
    - Malignant—Sarcoma and epithelioma.
4. *Acromegaly.*
5. *Leontiasis ossea.*

Injury.—A *hæmatoma* or traumatic *periostitis* may follow on a blow. If the injury has been sufficient to cause a *fracture*, the abnormal mobility of the fragments, the irregularity of the line of the teeth and arch of the jaw, and the laceration of the gums, are sufficient to indicate the injury. The nearer the line of fracture is to the symphysis, the more marked is the mobility, and diagnosis is only difficult when the fracture is of the ascending ramus and underneath the masseter muscle. A skiagram may then be needed. Fracture of the mandible is commonly compound, and therefore is often complicated by septic infection. Later, *callus* will form a tumour which might be mistaken for one of some other kind until the course of the case has been watched.

Inflammatory Affections.

Alveolar Abscess.—This is a very common swelling, associated with toothache. An ordinary gum-boil forms at the edge of the gum, and is quite superficial. A more troublesome form of abscess is that which develops at the root of a tooth, which, generally carious, may yet appear healthy on the surface. Pus usually points between the gum and the cheek, but it may travel a long way between the bone and the mucous membrane, and point on the cheek, in the submaxillary region, or on the chin. As in the case of injury, periostitis extending up under the muscle may be difficult to diagnose, and it is sometimes mistaken for parotitis. In the early stages the only sign is toothache, but as suppuration becomes established there are also pain, swelling of the gums, furred tongue, trismus, enlargement of the lymphatic glands, raised temperature. The presence of a septic tooth indicates the diagnosis.

Necrosis of the Jaw, often preceded by an acute periosteal abscess, may follow injury, alveolar abscess, syphilis, or mercurial or phosphorus poisoning, and in rare cases acute exanthemata or typhoid fever. In many cases it may be impossible to say whether the bone is necrosed or not, for the signs are much the same as in suppuration in connection with alveolar abscess. It can only be diagnosed for certain if a piece of loose bone can be felt with a probe or seen by the aid of a skiagram. Its presence may be inferred by the long continuance and profuseness of the discharge.

Syphilitic disease of the lower jaw is rare, and if present will not usually be confined to the jaw. If there is doubt, a Wassermann's reaction will be of service.

Actinomycosis.—A long-standing and obstinate suppuration about the lower jaw, with cellulitis of the neck and formation of sinuses in the skin, should lead to the suspicion of the nature of the trouble. In the beginning it gives rise to inflammatory changes which simulate alveolar abscess, and the similarity is increased by the presence of carious teeth, through which the fungus is believed to gain access to the jaw. In the pus, the small yellow granules are to be sought for, and the Gram-staining mycelium on microscopical examination (*Plate XXVIII*, p. 614).

Tumours.—In many cases there will be no difficulty in deciding whether a swelling is inflammatory or a new growth. In the early stages, however—and it is never to be forgotten that an early diagnosis in the case of malignant disease is of extreme importance—there may be grave doubt. Therefore, all possibility of inflammatory mischief should be excluded by a careful, thorough examination of the mouth and teeth for any source of infection, and for this purpose it is frequently advisable to invite the co-operation of a dentist.

Innocent tumours are osteoma and fibroma (more commonly called a fibrous epulis). *Osteoma* is rare, very slow growing, well defined, bony hard, and it does not usually attain a very large size. A not uncommon place to find it is at the angle of the jaw, projecting into the mouth. It may be bilateral. *Fibrous epulis* is common, soft, composed of fibrous tissue, and covered by the mucous membrane of the gum. It arises in connection with the root of a decayed tooth, and if not treated may attain a sufficient size to cause displacement of the teeth or even distortion of the arch of the jaw. Sarcomata may start in this manner; therefore all such tumours should be submitted to microscopical examination before a definite diagnosis or prognosis is given.

Malignant tumours are *primary*, sarcomata, and *secondary*, epitheliomata, which start in the gum or on the floor of the mouth and invade the jaw by direct extension.

The diagnosis of *sarcomata* may be quite easy, or attended by the greatest difficulty. They occur at any age, even in young infants. They may be of rapid growth, associated with constitutional changes which simulate inflammatory conditions before a large size has been attained, or they may be of such slow development as to be confounded with innocent growths. The necessity of early diagnosis cannot be urged too strongly, for it is on this that successful treatment depends. Seeing that a growth may be mistaken for a swelling due to suppuration, examination should first be directed towards seeing if any of the ordinary signs of inflammation are present, and whether there is an obvious source of infection. The history of the duration of the illness may be of great service, and also the nature of the swelling itself. Is it hard or soft, is the bone expanded, are tissues round the bone infiltrated, are the glands enlarged? Exercising the greatest care, diagnosis may still be difficult, and much service is rendered by a skiagram, with the aid of which one may determine whether the swelling is really bony, or in the case of periosteal sarcoma if the bone has been eaten into. If the diagnosis can be settled by no other means, the growth should be cut into and a piece removed for microscopical examination, even if the tumour is bony and a chisel and mallet be required.

Epithelioma—better termed squamous-celled carcinoma—is a very insidious and dangerous form of growth, and in its early stages very apt to be overlooked. It may start as a small ulceration of the gum about a decayed tooth, and so be mistaken for a simple ulcer, and it may not be until a large tumour has formed that the condition is recognized, when most valuable time will have been lost from the point of view of treatment. The diagnosis will be made by careful examination, and noting that the ulcerated gum is hard and indurated and does not heal when the decayed tooth is removed. The name ‘ boring epithelioma ’ has been well applied to this condition. To make the diagnosis sure, a piece from the edge of the ulcer should be removed for histological examination at the earliest moment that suspicion is aroused as to its malignancy. An epithelioma may also spread from the tongue or floor of the mouth and cause a swelling involving the jaw. The diagnosis here is obvious.

Tumours of the Teeth, Odontomata, may arise from any portion of the dental tissue, either from the tooth germ or from the fully-formed tooth. Clinically they are innocent, and commoner in young people. The method of diagnosis is to examine the teeth and find out if any of them are missing or abnormally arranged. It is easy to distinguish them from a periosteal sarcoma, but confusion may arise between them and a very slow-growing endosteal or myeloid sarcoma. A skiagram will generally reveal the true state of affairs, for any abnormality or misplacement of the teeth is clearly shown. It is well to remember the existence of these tumours, for unnecessarily severe operations have often been performed in ignorance.

Two diseases in which the mandible becomes enlarged, but in which the swelling is not confined to the one bone, and is only one of the manifestations of the complaint, remain to be mentioned:—

Acromegaly.—The lower jaw is often enlarged conspicuously in this disease, becoming prominent and massive (*Fig.* 116, p. 237). There is hypertrophy of the whole bone rather than a swelling in it. The other bones of the face are enlarged, the superciliary ridges are exaggerated, and the general effect of the disease is to give the patient the appearance of a dull, coarse-featured person. In addition, the hands and feet become much enlarged; also, in the late stages of this very chronic illness, headache and muscular debility become prominent symptoms, and owing to swelling of the pituitary body, bilateral temporal hemianopia is to be expected (see *Fig.* 139, p. 300).

Leontiasis Ossea is the name given to a rare disease in which hyperostoses of the facial and cranial bones are the distinguishing features. It is not likely to be confounded with any of the above-mentioned swellings, except perhaps acromegaly, from which it is distinguished by the absence of changes in the hands and feet. *George E. Gask.*

SWELLING OF THE JAW, UPPER.—The remarks made in the article on SWELLING OF THE JAW, LOWER (p. 683), apply equally to swellings in the upper jaw. Tumours arising in the antrum of Highmore merit special mention, however, for many cause no pain or discomfort until the late stages. Though innocent tumours may start in the antrum, the commonest are sarcoma and endothelioma. Rapid growth, bulging into and invasion of surrounding fossæ, pain, discharge of blood and pus from one nostril, and invasion of the overlying skin, are momentous indications of malignant disease. In the case, though, of slow-growing tumours and in the early stages, differentiation between innocent growths or suppuration is extremely difficult. Transillumination (see PAIN IN THE JAW, UPPER, p. 462) is to be employed (*Fig.* 84, p. 180), also puncture of the antrum, and if necessary exploration and histological examination of the parts removed.

George E. Gask.

SWELLING OF THE LEGS.—(See ŒDEMA, p. 411.)

SWELLING, MAMMARY.—*Method of Examination.*—The clothes should be removed to the waist, so that a clear view of both breasts, the thorax, axillæ, and supraclavicular fossæ may be obtained. Both breasts should then be looked at to see whether there is any obvious enlargement or abnormality such as redness of the skin, dilatation of veins, tumour, or ulcer. Next, palpation is to be employed, using the flat of the hand and not the tips of the fingers; the surgeon should place himself in a convenient position behind the patient, using the right hand to examine the right breast and the left hand the left. The axillary fossæ should also be palpated carefully, it being remembered that the lymphatic glands affected in diseases of the breast lie on the surface of the thorax and not round the axillary vessels. (See SWELLING, AXILLARY, p. 666.) In cases of suspected cancer the examination must not be concluded without investigation of the supraclavicular fossæ for fullness or enlargement of glands, and of the thorax and liver for signs of secondary growths.

Swelling in Pregnancy and Lactation is normal and physiological. Both breasts are enlarged equally, and feel tense and nodular. The superficial veins are usually prominent, and on gentle squeezing a few drops of milk are discharged from the nipple.

True Hypertrophy of one breast is rare. It may be found in nursemaids who have put children to the breast. The enlargement in the majority of so-called cases of hypertrophy is really due to the presence of one or more fibro-adenomata.

Acute Mastitis occurs usually during lactation, occasionally during pregnancy, and is most often due to infection with pyogenic organisms which have gained entrance through cracks in the nipple. At the beginning of the illness there is shivering, followed by fever and a feeling of weight and pain in the breast; the pain soon becomes very acute. In the early stages the swelling is limited to one part of the breast, which feels more resistant than normal; the skin is not reddened at first, nor are the lymphatic glands enlarged. Pressure over the swelling may cause extrusion of a drop of pus from the nipple, and this is distinguished from milk by its viscidity and yellow colour. Later, fluctuation may become evident, and, as the inflammation approaches the skin, this becomes red and œdematous, and ultimately an abscess may point and burst through it; at the same time other foci of suppuration form, until the breast may be nothing but a bag of pus. The presence of fever and the intense tenderness of one portion of the breast are sufficient to distinguish acute mastitis from the physiological engorgement.

It is not uncommon to find a small *alveolar abscess*, the size of a hazel-nut, in virgins.

Soon after birth and at puberty, a diffuse enlargement may occur in both sexes, and a small quantity of milk may be secreted. If the breasts are handled or squeezed, this congestive condition may pass into true inflammation and suppuration.

Chronic Mastitis may attack numerous lobes of the breast, so that the whole organ has a granular feel (chronic lobular mastitis), or the inflammation may be confined to one segment and form an inflammatory swelling of considerable size. The attention of the patient is usually first called to the breast by the presence of vague pains and tenderness. If the lump is picked up with the fingers it is easily palpable, but if pressed back against the chest wall the induration is much less distinct than is carcinoma or fibro-adenoma. The swelling is elastic, and its outline quite diffuse, more so than in the case of carcinoma. The axillary glands may or may not be enlarged ; if they are, they are generally numerous, not so hard as in cancer, and are met with at an earlier period in the disease. The opposite breast is very liable to be diseased in a similar manner. The diagnosis is often very difficult, ordinary carcinoma of the breast being confounded with it. The two tumours resemble each other in that in both their outlines are badly defined and the axillary glands are enlarged. In cancer, however, the tumour is densely hard, and at an early period adhesions form so that the skin puckers on attempting to move it over the swelling. A further difficulty arises from the fact that a cyst may form in connection with chronic mastitis. If this is lax, fluctuation may be detected, but it is usually so tense that it feels hard and solid. This again may be mistaken for a carcinoma or a fibro-adenoma. Where there is the least doubt as to the nature of the swelling and any possibility of the presence of a carcinoma, the right course is to make an exploratory incision and cut microscopic sections from the suspected area.

Multiple Cystic Disease of the Breast.—This condition may follow on chronic lobular mastitis. One breast—sometimes both—becomes filled with cysts, some microscopic and others as large as walnuts, so that the organ has a bossy appearance. The whole organ is often very painful, the pain radiating from the breast and shooting down the arm. There are epithelial changes in the lining membrane of the cysts, and some authorities think that these are precursory stages in the formation of a cancer.

Cysts, unless in connection with chronic mastitis or fibro-adenomata, are rare. A simple serous cyst is described, due to lymphatic obstruction. *Galactocele*, a cyst containing milk, is formed by dilatation of one of the larger lacteals owing to obstruction ; galactoceles occur only during lactation ; they form movable, fluctuating swellings, and on pressure milk can be squeezed out of the nipple.

Tuberculosis of the Breast is not so uncommon as was formerly supposed, and a certain number of cases of chronic mastitis and chronic abscess are really tuberculous. The disease is insidious, starting as a painless irregular swelling, the periphery of which is hard and the centre soft. Later, the skin becomes reddened, and an abscess forms which may burst and leave a sinus. It differs from an acute abscess in that the duration is much longer, there is little or no pain or fever, and the pus, if examined, reveals no organisms. The facts that its history is a long one, that the swelling or the edges of it are hard, and that the axillary glands are enlarged, render this condition liable to be confounded with carcinoma, of the ordinary form, or one in which suppuration has occurred. The various clinical pathological tests for tuberculous disease may be applied, but the best method is to cut into the swelling and remove a portion of the wall for histological examination.

Chronic Submammary Abscess causes a projection forward of the whole breast ; it is due to tuberculosis of the underlying ribs, or in rare instances to post-typhoidal periostitis which may have remained latent. The diagnosis is made by opening the abscess and examining the pus from it bacteriologically.

Innocent Tumours.—*Pure fibromata*, *lipomata*, and *enchondromata* are of rare occurrence, and merely call for mention here. *Fibro-adenoma* is the only common innocent tumour, and though there are many pathological varieties, and some contain cysts and some intracystic growths, for the purposes of this article all may be classed under one heading. A *fibro-adenoma* is an encapsulated tumour, generally single, sometimes multiple varying from the size of a nut to that of an orange. Because it is encapsulated, the surrounding tissues are not infiltrated ; therefore, if superficial, the outline is clearly defined and the mass is freely movable both under the skin, over the pectoral muscle, and, most important of all, in the breast substance. The axillary glands are not enlarged. The tumours cause no pain, and are usually discovered accidentally. Generally they occur in women between the ages of twenty and thirty. After attaining a certain size they remain more or less stationary, unless they are cystic, when they may go on growing as the result of dilatation of the cyst by fluid. The diagnosis is generally quite easy, but if the breast is fat and the tumour deep-seated, it may not always be quite easy to distinguish a fibro-adenoma from an early carcinoma, without operation and microscopical examination. A fibro-adenoma is elastic in consistency rather than hard like a carcinoma. From chronic mastitis it is distinguished by being less intimately associated with the breast than is the case with the inflammatory nodules, and by its sharper definition. On account of the well-known possibility of error, however, no definite diagnosis or prognosis should be given until the tumour has been removed and a pathological report on its character received.

Malignant Tumours of the breast are nearly always primary; sarcoma is rare, carcinoma common; and the latter is the most important tumour that affects the breast. It is essentially a disease of the female, only about one per cent of the cases occurring in males; most patients have been married, and are between the ages of thirty-five and sixty. In advanced cases the disease is obvious; the tumour is large and hard, fixed to and often fungating through the skin; the axillary glands are enlarged and hard, and the patient is often cachectic. What is wanted is a diagnosis in the early stages, while the patient still looks and feels in perfect health, before secondary deposits are found in the axillary glands and while successful treatment is still possible. Too much insistence cannot be laid on this. Usually the patient feels no pain, but discovers a lump in the breast accidentally during ablutions; therefore its duration must generally be a matter of doubt. Clinically, it is felt as a small tumour which, unless the patient is very fat, can be palpated easily with the flat of the hand. Its chief characteristic is that its outline is not sharply defined, and that it is hard—stony hard. In the very early stage the tumour is freely movable over the pectoral muscles and under the skin, but it is not so movable in the breast substance as is a fibro-adenoma. Very soon bands of fibrous tissue that connect the breast with the skin become involved, and by their contraction prevent free movement of the skin over the swelling, and cause dimpling and puckering. If the tumour is situated anywhere near the centre of the breast milk-ducts become involved in the growth, and as they contract cause retraction of the nipple. If a nipple, previously well formed, becomes retracted, this is a very important sign, though it is to be remembered that nipples are often permanently retracted. Many cancerous tumours, even when extensive infiltration has occurred, cause shrinkage, so that the affected breast may appear smaller than the healthy one, and in the atrophic form the gland may almost disappear. In the ordinary form (scirrhus) it will be rare to find any discharge from the nipple: a blood-stained discharge often indicates a duct-carcinoma. (See DISCHARGE FROM THE NIPPLE, p. 181.) After the disease has lasted six months the axillary glands are usually enlarged and hard, the first affected being those running along the lower border of the pectoralis minor. Too much attention must not be given to the absence of palpable glands, because, first, it is hoped that the diagnosis may be made before they are enlarged; and secondly, if the patient is at all fat, it is exceedingly easy to overlook them. Attention is to be centred on the lump itself. Its stony hardness may alone be sufficient ground on which to base a diagnosis. The two main conditions which have to be distinguished from an early carcinoma are fibro-adenoma and chronic mastitis. In the former, the swelling is well defined, elastic, and freely movable; in the latter, a tumour cannot be felt distinctly with the flat of the hand; it is soft, and the whole breast is often nodular.

The difficulties in diagnosis are great and the sources of error numerous; none of the swellings may be typical: they may be obscured by the obesity of the patient, and a fluid swelling may be so tense as to simulate a solid one. This being so, the course to adopt, whenever the slightest doubt arises, is to incise the swelling and submit a portion to microscopical examination. Seeing the vital importance of avoiding mistakes in this connection there is a growing feeling among surgeons that all tumours of the breast, whatever the belief as to their character, should be removed, or at least cut into, so that their true histological constitution may be ascertained early and with accuracy.

Sarcoma of the breast is rare. It generally occurs in women under the age of thirty. In the early stage it is not easily distinguishable from a fibro-adenoma, particularly one which is enlarging rapidly on account of a cyst or intracystic growth. It is soft, grows rapidly, infiltrates the tissues, and forms a large fungating tumour. It disseminates rapidly, both via the lymphatics and by the blood-stream. *George E. Gask.*

SWELLING, PELVIC.—There are so many swellings which may rise up out of the pelvis into the abdomen, and also which may appear to be pelvic when they are really primarily abdominal, that a list in tabulated form may be of value :—

Bladder.—Simple distention. New growth.
Vagina.—Hæmatocolpos.
Uterus.—Pregnancy : normal or abnormal, or associated with tumours of the uterus or ovary.
New growths : Fibromyoma. Sarcoma. Carcinoma. Chorion-epithelioma.
Hæmatometra.
Ovary.—Cysts. Solid new growths.
Fallopian Tubes.—Hydrosalpinx
Pyosalpinx
Salpingo-oöphoritis
New growths
Carcinoma
Tubal gestation
Progressive extra-uterine gestation.
Pelvic Peritoneum.—Encysted peritoneal fluid
Hæmatocele due to extra-uterine gestation
Hæmatocele due to hæmorrhage from a corpus luteum
Pelvic abscess
Ascites
Hydatid cysts
Retroperitoneal lipoma.
Pelvic Cellular Tissue.—Cellulitis. Pelvic hæmatoma.
Appendix Vermiformis.—Abscess around
Appendicitis with pregnancy.
Pelvic Bones.—New growths of.
Omentum.—New growths of. Cysts of.
Phantom Tumours.
Pancreatic Cysts.
Kidney.—Tumours of. Hydronephrosis. Pyonephrosis.
Gall-bladder.—Distention of.
Spleen.—Enlargement of.
Urachus.—Cyst of.

It is obvious that many of these lesions are not pelvic at all ; but they are not omitted from the list because they are liable to be mistaken for pelvic tumours. Thus *pancreatic, renal, splenic,* and *gall-bladder tumours* may reach the pelvic brim, but the history ought to show that they have grown down from above, not up from below. Further, *renal tumours* may be associated with urinary changes, or absence of urinary secretion on the affected side, as detected by the cystoscope. *Splenic enlargements* may be associated with blood-changes, and *gall-bladder distention* with icterus. *Pancreatic cysts* are the least likely to be mistaken for pelvic swellings, but they have been difficult to distinguish from ovarian tumours with long pedicles.

Naturally, the commonest difficulty which arises in the diagnosis of pelvic swellings is to differentiate between the *distended bladder, pregnant uterus, ovarian cyst,* and *uterine fibromyoma,* and the commonest mistakes are made between these swellings. The *distended bladder* is clearly the easiest to dispose of, because the passage of a catheter will settle the question ; and yet the neglect of this simple procedure has led to more than one abdomen being opened.

The history is of value in differentiating the other swellings, for amenorrhœa is the rule in pregnancy, menorrhagia in fibromyoma, and no change in menstruation in ovarian tumours. These assumptions are correct in almost 99 out of every 100 cases, but exceptions do exist. The cardinal point in diagnosis is not to think of the possible fallacies until the common rule has been considered thoroughly. Normal menstruation during pregnancy is almost unknown, but it is believed that menstruation is possible up to the third month. This is physiologically unsound, for menstruation represents the failure of the uterus to receive a fertilized ovum, and should not be even possible if conception does occur. That hæmorrhages occur during the early months of pregnancy is true; but in most cases these hæmorrhages represent threatened abortion, and not menstruation. Further, fibroids are associated with hæmorrhages. This is true in the case of interstitial or submucous growths; but there may be no disturbance of menstruation in subperitoneal fibroids. Ovarian tumours only disturb menstruation when they are double, and destroy all ovarian tissue. As long as a small piece of ovarian tissue remains there is no reason why menstruation should not occur normally.

Palpation of these tumours may be fallacious, although there is no difficulty in distinguishing fœtal parts when the fœtus is big enough. In the early months the pregnant uterus may fluctuate like a cyst; a softened fibroid may do the same, whilst on the other hand a tense ovarian cyst may feel so hard as to be mistaken for a fibroid. Whilst the presence of the fœtal heart is characteristic of pregnancy, its absence cannot be taken as evidence of a fibroid or of an ovarian tumour. It is not always possible to hear the fœtal heart even in advanced pregnancy. If the pedicle of a tumour can be felt definitely attached to one uterine cornu it is strong presumptive evidence of an ovarian tumour. It is useful to pull down the uterus with a tenaculum, at the same time pushing up the tumour so as to make tense the pedicle, which might then be palpated by the vaginal touch. When small tumours are in question the first point which arises is, Can the tumour be separated from the uterus bimanually? If so, it can be neither a fibromyoma of the uterus nor a normal uterine pregnancy. This point can only be made out by careful bimanual examination, and undoubtedly may require considerable skill in some cases.

Early pregnancy in a retroverted uterus should not give rise to diagnostic difficulties if it be remembered that the soft, boggy fundus is felt through the posterior fornix, that the cervix looks down the vagina or forwards to the symphysis, and that the posterior mass is continuous with the cervix. If the retroverted uterus is associated with vesical distention the picture is usually clear enough. The history of constant dribbling of urine (distention with overflow), amenorrhœa, other signs of pregnancy, the presence of two tumours—one in front, tense and elastic, the other behind, soft and boggy—and finally, the passage of a catheter, will settle the question. The diagnosis of solid ovarian tumours is not always possible, for the pedicle is often short, and the tumour is then so close to the uterus that the two cannot be separated. They are therefore likely to be mistaken for fibroids of the uterus. They do not often cause menorrhagia, however, and this may be remembered as a cardinal point.

Large tumours arising in the pelvis are not often difficult to differentiate from one another, bearing in mind that ovarian tumours, uterine fibroids, pregnancy, and ascites are the common conditions which are met with. In this connection, it cannot be repeated too often that amenorrhœa stands for pregnancy, and occasionally for ovarian tumours when double. Menorrhagia goes with uterine fibroids except in the case of subperitoneal tumours. Exceptions to these general statements are uncommon, and mistakes in diagnosis will occur but seldom if they are borne in mind. Ascites has to be differentiated from ovarian cysts, and occasionally from hydramnios. In general, ascites gives dullness in the flanks on percussion, with resonance over an area somewhere about the umbilicus, whilst ovarian cysts give dullness over the front of the abdomen, with resonant areas in the flanks and epigastric angle. When ascites exists along with ovarian tumours the free fluid may be so large in amount that the tumour cannot be felt; as a rule, however, it can be touched on dipping through the fluid. Ascites with an ovarian tumour does not necessarily mean malignancy, but it may do so. Fibroma of the ovary, and simple ovarian cyst, with a twisted pedicle, will always be accompanied by some fluid.

When *pregnancy is associated with tumours*, the diagnosis may be of great difficulty. This does not lie in the recognition of the pregnancy; amenorrhœa, breast changes, fœtal movements, and the fœtal heart will usually make that clear enough; it lies in deciding the nature, or even the presence, of a tumour along with the pregnant uterus. In the early months, when the presence of two tumours can be demonstrated, the diagnosis is easier, but in the later months the great size of the abdomen, and the way in which the swellings merge into one another, may obscure the picture. The relation to the uterus, whether a part of it, or attached to it by a pedicle; the feel of the tumour, whether solid or cystic, soft or hard; and the previous history; will always be of assistance in making out the nature of the growth. Fibroids are extremely likely to soften and degenerate during pregnancy, so that they are liable to be mistaken for ovarian cysts.

In the case of *ovarian tumours*, it is often impossible to be sure of the exact nature of the growth, and this has to be decided microscopically after removal. It is, however, important to distinguish malignancy in growths of the ovary, and certain points will stand out in favour of this. Thus, fixation of the growth in the pelvis, obvious ascites, emaciation of the patient, and rapid growth in size of the abdomen, are points in favour of malignancy.

In the case of definitely *uterine tumours*, the diagnosis of malignant growths is not often difficult, but may have to be settled by microscopic examination of curetted fragments. Fibroids are only likely to be mistaken for malignant growths when they produce constant bleeding as a result of extrusion, infection, and sloughing. Rapid growth of a fibroid is more likely to be the result of degenerative changes, such as formation of cysts or necrobiosis, than to the development of a sarcoma or other malignant growth along with it.

With small tumours confined to the pelvis, or rising only a little above the brim, diagnosis is often a matter of extreme difficulty. In practice, however, *extra-uterine gestation* and its resulting blood-tumours stand out pre-eminently as swellings which must be recognized at once, if successful treatment is to be adopted. Before rupture or abortion has occurred a tubal gestation is essentially a small tumour in one postero-lateral corner of the pelvis, attached to the uterus, indefinite in consistence, and perhaps—though not always—associated with amenorrhœa of short duration, and attacks of pain in the pelvis of an acute nature. Definite signs of pregnancy may be entirely wanting. It may be mistaken for a chronic salpingo-oöphoritis, a small cystic ovary, a small pedunculated fibroid, or a small ovarian dermoid. The differential diagnosis may be absolutely impossible ; but attacks of pain unassociated with menstruation are not likely to occur in any of the latter conditions. The attacks of pain are usually the result of over-distention and stretching of the tube from hæmorrhage into its wall or lumen around the fertilized ovum. When tubal abortion has occurred, or tubal rupture, the signs of internal bleeding, accompanied by sudden pain and collapse, with hæmorrhage from the uterus, usually make an unmistakable picture. Hæmorrhage is more commonly severe and copious in tubal rupture than in tubal abortion. If the patient recovers from the initial bleeding the clinical picture may be that of a retro-uterine *hæmatocele*, or of a peritubal hæmatocele. In this form the uterus is pushed forwards and upwards against the symphysis pubis, and the mass of blood-clot can be felt posteriorly bulging the posterior fornix, and also the anterior wall of the rectum. The tumour is usually partly resonant in front, because intestine adheres to it. Tubal abortion is most likely to be mistaken for an ordinary uterine abortion ; but the presence of a mass on one side of the uterus, with a closed cervix, and the absence of uterine contractions or extrusion of any products of conception, should make the case clear.

Progressive extra-uterine gestation is a rare occurrence, and is the result of continued growth of an embryo after a partial separation from the tube as a result of rupture, or extrusion from the fimbriated end (abortion). The continued enlargement of a mass beside the uterus, with amenorrhœa and progressive signs of pregnancy, are the most characteristic points. The diagnosis, however, is difficult, because there is always some effused blood which is likely to obscure the outlines of the uterus, and make it appear to be a part of the pelvic mass.

The swellings due to *salpingo-oöphoritis* are usually quite easy to distinguish. They form fixed masses in the pelvis, seldom of any definite shape, but occasionally presenting the characteristic retort shape, with its narrow end near the uterus, which the tube assumes when distended with fluid. The history is usually that of an acute illness at some period, with pain in the pelvis, rise of temperature, and peritoneal irritation. It is preceded, as a rule, by uterine discharges and menorrhagia. This inflammatory disturbance in married women is associated with long periods of sterility, owing to the sealing up of the tubes. The diagnosis of suppuration with salpingo-oöphoritis is often impossible, but is always important, because the treatment may depend on it. Constant rises of temperature of the hectic type, wasting, and daily sweating, are the usual accompaniments of suppuration here as elsewhere.

A large *pelvic abscess* may accompany salpingo-oöphoritis, or may occur alone without infection of the tubes, as we see occasionally in puerperal septic infections. When it does occur, it is of course peritoneal; it fixes the uterus in a central position, bulges into the posterior fornix and rectum, tends to rupture into the rectum, is acute in onset, and accompanied by signs of local peritonitis. It is likely to be confounded with *pelvic cellulitis*, in which the uterus is fixed in a laterally displaced position. It bulges one lateral fornix, tends to burrow along the round ligament to the groin, is slow in onset, chronic, and not accompanied by signs of local peritonitis. It always follows labour, whereas pelvic abscess of peritoneal origin may occur with salpingo-oöphoritis quite apart from pregnancy. Pelvic cellulitis never bears any relation to salpingo-oöphoritis.

Encysted peritoneal fluid, *hydatid cysts*, and *retroperitoneal lipoma* are generally diagnosed as ovarian cysts, and their true nature is only discovered at operation. There are no definite signs by which these conditions may be diagnosed, and as they all require operative treatment, post-operative diagnosis meets their requirements.

Distention of the vagina by menstrual fluid is not likely to be mistaken for anything else, if only on account of the absolute closure of the hymen which gives rise to it. Hæmatocolpos is practically the only central tumour met with between the rectum and the bladder reaching from the hymen to the pelvic brim. The uterus can usually be felt like a cork movable upon its upper extremity.

Urachal cysts occur in *front* of the uterus and in close relation to the bladder; but in spite of this they are usually mistaken for ovarian cysts. It is to be remembered, however, that ovarian cysts only get in front of and above the uterus when they are large. Urachal cysts rarely attain a large size.

Appendicitis with pregnancy occurs occasionally, and may be mistaken for such a condition as torsion of an ovarian pedicle. The swelling due to appendix inflammations is, however, in close relation to the anterior superior spine of the ilium, and apparently adherent to the iliac fossa. The lump is ill defined, and rarely fluctuates unless there is a large abscess. The acute onset may be similar to that of torsion of an ovarian pedicle. There is usually a definite fluctuating tumour when an ovarian cyst is present, and some interval between it and the iliac crest can usually be felt.

Phantom tumours are due to diaphragmatic contraction, causing the abdominal wall to bulge. They are usually mistaken by patients for pregnancy, but are not accompanied by any of the signs of pregnancy. Amenorrhœa must be excepted from this, however, because these cases usually occur about the menopause. Their true nature can usually be discovered by making the patient breathe normally, relaxing the diaphragm; but if any doubt exists, the protrusion will disappear under an anæsthetic.

Growths of the pelvic bones are very rare tumours, usually cartilaginous or sarcomatous. They are only likely to be mistaken for adherent inflammatory masses, due to salpingo-oöphoritis. They will be found to be continuous with the bones forming the pelvis, and when growing from the sacrum may have the rectum in front of them; all other tumours have the rectum behind them. They may, however, bear no relation to the rectum at all if they occur on the right side of the pelvis. In most cases of this nature the uterus and adnexa can be palpated bimanually, and shown to be free from disease and unconnected with the mass. When complicated by the presence of a pregnant uterus their true nature may be very difficult to determine. Bearing in mind that they are absolutely fixed and continuous with the bones of the pelvis, the diagnosis ought not to be uncertain.

Thos. G. Stevens

SWELLING, PERINEPHRIC.—(See KIDNEY, ENLARGEMENT OF, p. 352.)

SWELLING, POPLITEAL.—Popliteal swellings may be divided into:—

1. **Fluid Swellings:**
   - Bursa
   - Baker's cyst
   - Varicose veins
   - Abscess
   - Aneurysm.
2. **Solid Swellings not connected with Bone:**
   - Enlarged glands
   - Malignant tumours
   - Innocent tumours.
3. **Solid Swellings connected with Bone:**
   - Exostosis
   - Sarcoma
   - Periostitis
   - Separation of the epiphysis.

FLUID SWELLINGS.

Bursa.—The bursa underneath the insertion of the semimembranosus muscle into the posterior aspect of the inner tuberosity of the tibia is often enlarged. When the leg is extended it stands out as a tense fluctuating swelling on the inner side of the popliteal space ; on flexion it disappears completely. It may be found enlarged in young athletes and cause no symptoms whatever. On account of its frequent communication with the knee-joint it is often distended when that joint is the seat of osteo-arthritis, and the changes found in the synovial membrane of the knee are found also in the synovial membrane lining the bursa, for the two are continuous. When much fluid is present, fluctuation can be detected between the joint and the bursa.

The bursa under either of the two heads of the gastrocnemius muscle may be enlarged similarly, but this is rare.

Baker's Cyst occurs in connection with chronic tuberculosis of the knee-joint, and is formed by the extension of a chronic abscess which spreads along a plane of fascia. Such an abscess may present itself in the popliteal space. The condition of the knee-joint will indicate the disease.

Varicose Veins are often present; the diagnosis presents no difficulties, as the veins in the lower part of the leg will be varicose also.

Acute Abscess is recognized by the signs of acute inflammation ; the skin is red and œdematous, the pulse and temperature are raised, and the swelling is very painful. The knee is kept flexed in order to minimize the tension of the part. The abscess may be caused by suppurating lymphatic glands or by suppurative periostitis or necrosis of the lower end of the femur. In the former case the abscess will be superficial, and in the latter deep to the popliteal vessels.

Aneurysm of the Popliteal Artery (*Plate XXX*) gives rise to an expansile pulsating tumour, the pulsation being synchronous with the heart's beat. Pressure on the femoral artery above will cause a diminution in size of the swelling and cessation of pulsation. The pulse at the ankle on the affected side may be smaller than that on the opposite, and delayed. If a stethoscope be placed over the swelling a distinct bruit can be heard. The complaint of the patient will probably be of pain, which may be referred down the leg if either popliteal nerve is pressed on, or in the site of the swelling if the bone is eroded. Varicose veins are almost always present also, on account of pressure on the popliteal vein. Owing to its pulsatile character, an aneurysm is not often mistaken for anything else, but it must be remembered that every swelling that pulsates is not an aneurysm. A soft vascular sarcoma growing from the end of the femur may be pulsatile, and over it a bruit may be heard, but the tumour is not as compressible as an aneurysm, and the effects on the distal pulse are not so marked. A skiagram will usually settle the question at once. Distinction must also be drawn between a tumour that pulsates and a tumour to which pulsation is communicated. For instance, an abscess or a solid swelling lying over the popliteal artery may appear to pulsate, but the movement is heaving in character and not expansile. In the rare event of an aneurysm having become filled with clot it might be taken for a solid tumour growing either from the soft parts or from the bone. Under this delusion a leg has been amputated for sarcoma.

SOLID SWELLINGS NOT CONNECTED WITH BONE.

Enlarged Glands.—It is not common to find the popliteal glands enlarged from any cause. It is possible that they may become infected with pyogenic organisms from a sore on the back of the leg.

Tumours are rare. They may be innocent, e.g., *lipoma* ; or *sarcomatous*, starting in the connective tissue of the popliteal space, or attached to one of the muscles. The innocent tumours are of long history and well defined ; the malignant, rapidly growing and infiltrating.

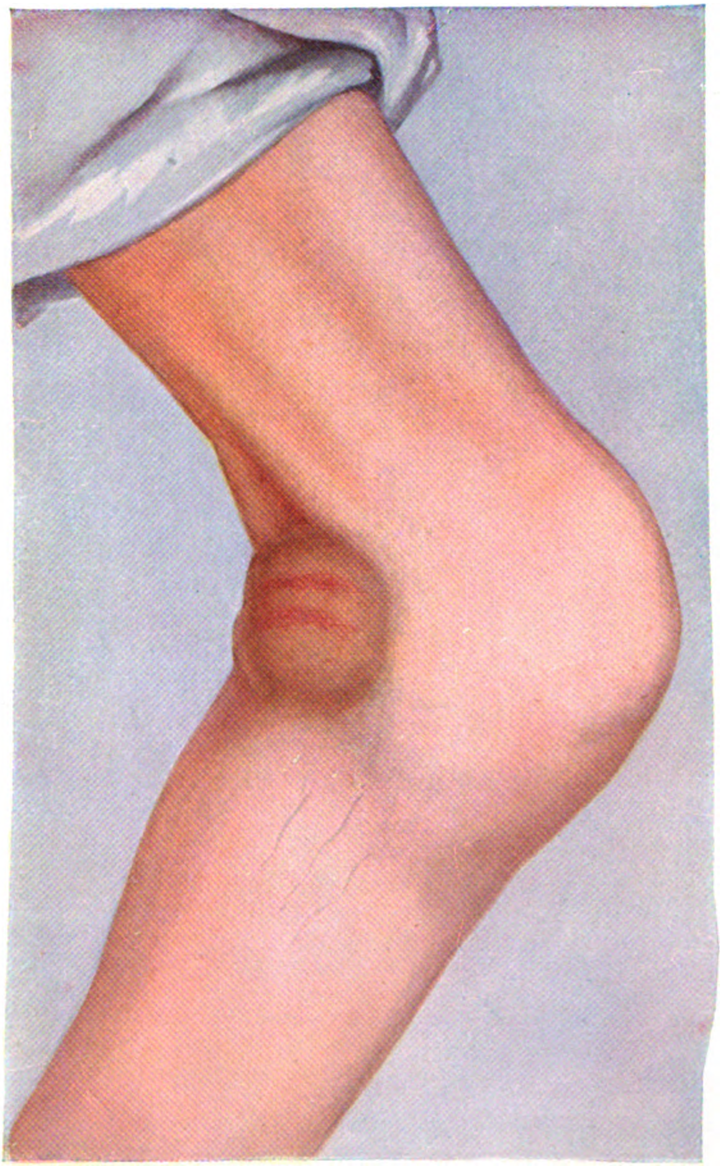
SOLID SWELLINGS CONNECTED WITH BONE.

In all cases of bony tumour a skiagram is of immense service, and should always be obtained if possible.

Innocent Tumours.—*Cancellous exostoses* may be found, generally in children and young adults, growing from the region of the epiphyseal cartilage of the femur (*Fig.* 281,

PLATE XXX

POPLITEAL ANEURYSM



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p. 670). There may be others in other parts of the skeleton, and sometimes several members of the family are affected similarly. The swelling is of slow growth, well defined, and rarely gives any trouble. It is most often found at the inner side of the popliteal space. There is one thing that may be confounded with it, namely, *ossification of the insertion of a tendon* or muscle. The adductor longus muscle is the one most commonly affected.

Malignant Tumours are endosteal and periosteal sarcoma. *Central sarcoma* in its early stages resembles chronic osteitis and periostitis so closely that it may be impossible to come to a correct conclusion without the aid of a skiagram. With this help the difficulty vanishes, for a myeloid tumour is seen clearly as a well-defined tumour causing enlargement of the bone (compare *Figs.* 284, 286, 287, pp. 672, 673).

Periosteal sarcoma causes a general enlargement of the whole of the lower end of the femur or upper end of the tibia (*Fig.* 283, p. 671), not swelling in the popliteal space only. It is mentioned here because of its occasional confusion with periostitis and popliteal necrosis.

Periostitis.—Popliteal necrosis with abscess formation may give rise to a big swelling. The signs of inflammation will usually be well marked and accompanied by constitutional symptoms and leucocytosis. Chronic periostitis, or chronic abscess of the bone, or central necrosis, may be extremely difficult to distinguish from a periosteal sarcoma. A skiagram should be taken, and if necessary an incision made down to the tumour for a piece to be removed for histological examination. (See SWELLING ON A BONE, p. 667.)

Separation of the Epiphysis.—In the somewhat rare accident of separation of the lower epiphysis of the femur, the lower fragment becomes displaced backwards, forms a prominence in the popliteal space, and presses on the vessels, sometimes to a dangerous extent.

George E. Gask.

SWELLING, PULSATILE.—When a tumour can be felt pulsating, the first point to decide, if possible, is whether the pulsation is expansile or whether it is merely transmitted by a non-expansile tumour which is in direct contact with large pulsating vessels. The distinction is sometimes obvious, especially when the tumour has developed in a place where there are no particularly large blood-vessels to transmit pulsation, for instance in the foot, or in direct connection with a long bone at some spot not immediately adjacent to the main artery of the limb. The chief difficulty arises when the mass is either in the root of the neck or in the abdomen and, to a less extent, when it is in the axilla, the inner aspect of the upper arm, in front of the elbow, in the groin, or in the popliteal space. Careful palpation is probably the best means of determining whether there is actual expansile pulsation or not; in the case of the abdomen it is important to examine the patient not only when he lies on his back, but also in the knee-elbow posture, for sometimes a tumour which is in contact with the aorta in the former position falls away from it and ceases to transmit pulsation in the latter.

If it can be decided definitely that the tumour is itself pulsating, most probably it is either an *aneurysm* of an artery or else a very vascular growth, especially *osteosarcoma.* The existence of egg-shell crackling with pulsation in a tumour would be highly suggestive of osteosarcoma, though it is conceivable that it might also be felt over an aneurysm that had extensively eroded the adjacent bones. Aneurysm will be the probable diagnosis when the markedly pulsatile swelling occurs directly along the course of a known artery. Absence of pulsation does not, however, exclude aneurysm, for the latter may either be situated too deeply for the pulsation to be felt, or else the sac may be filled partly or wholly by organized or organizing clot.

Sometimes there may be doubt as to whether there is really pulsation or not, when digital examination alone is relied upon; in such cases, direct application of the ear to the part in such a way that the pinna is in uniform contact with the patient's skin will sometimes bring pulsation to the notice very clearly when its amount, appreciable to the membrana tympani, is too slight for the hand to detect; this applies particularly to deep-seated intrathoracic aneurysms.

It must be remembered, on the other hand, that marked pulsation may suggest aneurysm without any being present, particularly at the root of the neck and in the abdomen (*Fig.* 288); a normal subclavian artery may sometimes seem to be abnormal, particularly if it is pushed forward or displaced by a mass below or behind it, for instance an accessory cervical rib. Undue pulsation of the abdominal aorta, especially in women, is also to be remembered as a possible source of erroneous diagnosis (see PULSATION, UNDUE ABDOMINAL AORTIC, p. 543).

It should also be remembered that normal arteries cause very violent pulsation in cases of marked aortic regurgitation, and in severe cases of exophthalmic goitre, in which the whole neck, including the enlarged thyroid gland, may be seen to be pulsating vigorously.

We need not here discuss in detail the differential diagnosis between one kind of aneurysm and another, though one might mention in particular the so-called cirsoid aneurysm of the scalp (*Plate XXXI*), which is rather a conglomeration of many abnormally dilated arteries in the form of an arterial nævus than a true aneurysm. Its position on the scalp will at once suggest the diagnosis.

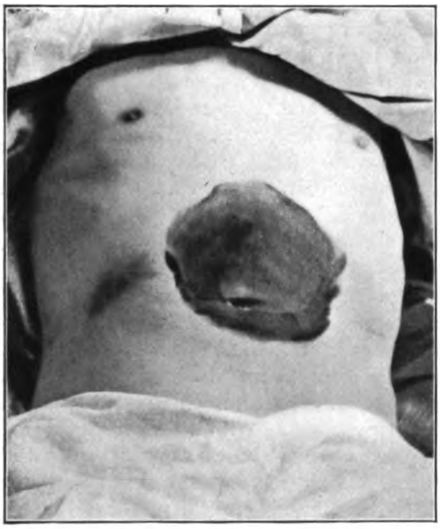


Fig. 288.—Photograph illustrating the difficulty which may arise in diagnosing between abdominal aneurysm and carcinoma of the stomach. This patient had a large rounded epigastric tumour (demarcated by painting with ink before the photograph was taken) which pulsated forcibly, and the pulsation seemed to be expansile. Aneurysm of the cœliac axis was diagnosed, but post-mortem examination revealed a large carcinoma of the stomach, and no aneurysm.

A pulsatile orbital tumour will generally be due either to an osteosarcoma, or to an arteriovenous aneurysmal communication between the internal carotid artery or its ophthalmic branch, and the cavernous sinus. The presence of a loud bruit would be in favour of the latter.

It is important not to mistake for the ordinary pulsatile tumours those which may move synchronously with respiration, for instance hernia pulmonalis, hernia cerebri, and certain congenital abnormalities of the brain and spinal cord, such as meningomyelocele (*Fig.* 102, p. 230).

It is unlikely that a pulsatile liver will be mistaken for any other kind of pulsatile tumour. The cases in which it occurs are those of chronic failure of cardiac compensation, generally mitral stenosis and tricuspid stenosis with œdema of the legs, lividity, orthopnœa, and perhaps ascites, which have generally been present for some time before the nutmeg liver becomes obviously pulsating.

Rarely, the cardiac pulsations may be transmitted direct to fluid contained in a pleural cavity, so that the bulging intercostal spaces may pulsate synchronously with the radial artery and simulate some more serious pulsatile tumour. The history and the physical signs, including displacement of the heart towards the opposite side, will generally indicate the correct diagnosis, though there may be some trepidation on the part of the operator who decides to insert the exploring needle into the pulsating swelling.

Herbert French.

SWELLING OF THE SALIVARY GLANDS.

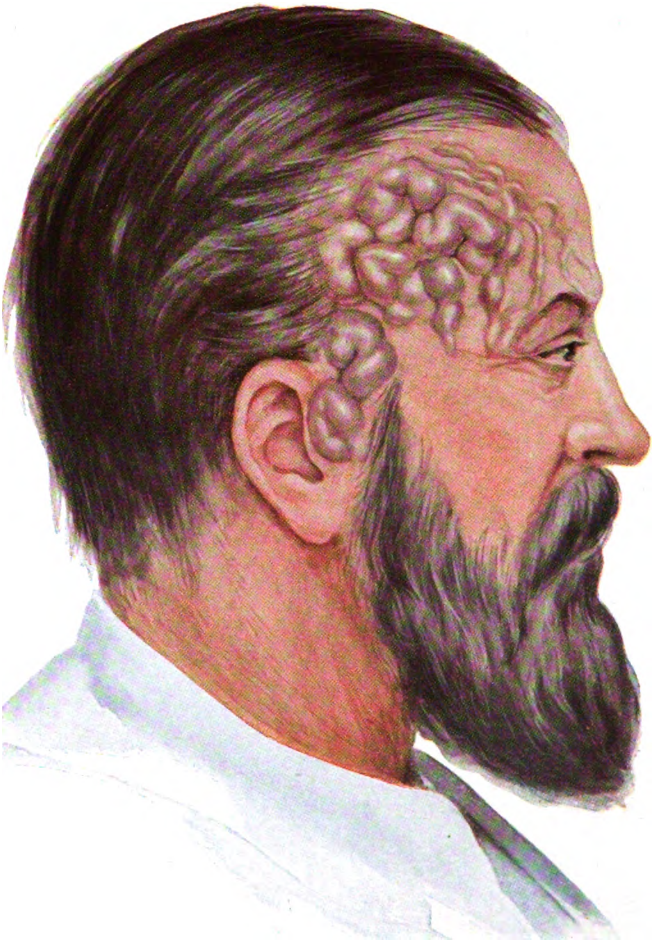
Epidemic Parotitis—Mumps—is the commonest cause of swelling of the salivary glands; it is discussed on p. 617.

Infective Parotitis is characterized by a sudden, acute and generally unilateral enlargement, and is accompanied often by a rise of temperature or a rigor. It is not an uncommon complication in certain specific fevers, e.g., typhoid fever and pneumonia; and in surgical practice it may complicate any septic case, but especially abdominal, pelvic, and genito-urinary operations, puerperal infection, and pyæmia. Resolution usually occurs, but suppuration may ensue.

Salivary Calculus.—A calculus may be found in Stenson's duct, but is more common in the duct of the submaxillary gland. In the early stages such a calculus gives rise to sudden intermittent swelling of the affected gland at times when the stimulus of food gives

PLATE XXXI

CIRSOID ANEURYSM



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rise to active secretion of saliva. As the result of long-standing distention and chronic inflammation, the gland may become permanently swollen. The diagnosis of stone may be made with the finger in the mouth, by pricking the calculus with a needle through the buccal mucous membrane, by passing a fine probe up the duct and feeling the grating on the stone, and by the use of the x-rays.

Parotid Tumours.—Both innocent and malignant tumours arise in the parotid gland. The innocent tumours (fibromyxoma, endothelioma, teratoma) are encapsuled, grow slowly, press aside the rest of the parotid gland, shell out freely, and if removed do not recur. If left alone they may attain a large size in the course of years. The malignant tumours (sarcoma and carcinoma) grow rapidly, soon affecting the whole gland, extending deeply among the important structures behind the ramus of the jaw and soon involving the facial nerve, causing facial palsy.

The diagnosis has to be made from simple enlargements of the gland and from enlargement of the pre-auricular lymphatic glands due to infection with pyogenic organisms, tubercle, or syphilis.

Tumours in the Submaxillary Salivary Gland are similar to but rarer than those which occur in the parotid gland.

Bilateral Salivary Swelling—Miculicz's Syndrome.—This condition is characterized by a chronic bilateral swelling of the parotid, submaxillary, and sublingual salivary, together in many cases with simultaneous swelling of the lachrymal glands (*Fig.* 3, p. 25), and it is sometimes associated with enlargement of the spleen and lymphatic glands, and with changes in the blood. The general appearances of the face are those of persistent mumps, so to speak. The syndrome is not a disease in itself, but results most often from *lymphadenoma* or *lymphatic leukæmia*; less often from some infection, including *tuberculosis* and secondary *syphilis*. A few cases have been attributed to *gout*. Each case should be investigated for any source of pyogenic infection from the mouth or gums, for syphilis by the Wassermann test, for leukæmia by blood examination (p. 24), and for any evidence elsewhere of tuberculosis.

George E. Gask.

SWELLING, SCROTAL.—It is first essential to prove that the swelling is really limited to the scrotal region, and this is best done by grasping the root of the scrotum between the fingers and thumb, and thus ascertaining if the swelling does or does not extend into the inguinal region along the cord. Failure to take this obvious precaution has led to the tapping of a hernia with disastrous results. True scrotal swellings may arise in any of the following tissues: (1) Skin; (2) The various connective-tissue coverings of the testicle; (3) Tunica vaginalis; (4) Testicle; (5) Epididymis; (6) The lower end of the spermatic cord; (7) The urethra; (8) The bones of the pubic arch.

1. The nature of **swellings affecting the Skin** is usually obvious. The only common ones are: Boils, soft sores and chancre, sebaceous cysts, warts, and epithelioma. The latter soon ulcerates, commonly occurs in sweeps, and the groin glands soon become enlarged.

2. **Swellings of the various Connective-tissue Coverings** are very rare, but occasionally a fibrosarcoma may occur. These swellings are movable upon the testicle. The symmetrical enlargement called *elephantiasis scroti*, due to the *Filaria sanguinis hominis*, is limited to the tropics.

3. The **Tunica Vaginalis** may become distended with fluid, thus forming the ordinary *vaginal hydrocele*. Except in late cases this is translucent, and is thus distinguished from a hæmatocele of the same cavity. It should not be forgotten that a hydrocele with thick walls may fail to give translucency. When proved to be translucent, it has to be distinguished from encysted hydrocele of the epididymis and encysted hydrocele of the cord. Vaginal hydrocele occupies the lower part of the scrotum and *envelops the testicle*, which cannot be felt as a separate object. *Encysted hydrocele of the epididymis* is placed *behind and above the testicle*, from which it is distinct, although attached at the upper and posterior part. Moreover, this variety of hydrocele never attains a large size, rarely getting larger than a Tangerine orange. It is not tightly distended, but is usually flabby, and it contains a characteristic milky fluid in which cholesterin crystals (*Fig.* 124, p. 254) are present. *Encysted hydrocele of the cord* is placed *above the testicle*, which can be felt as a separate object. It rarely attains a large size, and is often elliptical in shape, extending upwards

along the cord. All the hydroceles fluctuate. To test for this it is necessary to fix the swelling against some hard object. Bleeding may occur into any of them as a result of injury or constitutional disease. It is almost impossible to distinguish between an opaque hydrocele and a hæmatocele without tapping the swelling. In syphilitic disease, with irregular adhesion between the parietal and the visceral walls, a loculated hydrocele may occur.

4. **Swellings of the Body of the Testicle** may be inflammatory or neoplastic. Acute inflammatory swellings rarely attain a large size, and they are usually associated with enlargement of the epididymis, and occur as a part of acute epididymo-orchitis due to urethritis of some kind, or to *mumps* or as a *post-typhoidal* phenomenon. Chronic inflammatory swellings give rise to more difficulty. They are usually either *tuberculous* or *syphilitic*, or else due to *chronic torsion*. In the former disease, swelling of the epididymis is practically always primary and more advanced; but in infants the body of the testis becomes involved at a very early stage. The enlarged epididymis can be felt enveloping the posterior border, and the upper and lower poles of the testicle. There is often a little hydrocele which may obscure the shape of the testicle. If there is adhesion, with perhaps an abscess or a sinus at the posterior and lower part of the scrotum, it is characteristic of suppurative disease of the epididymis, usually of a tuberculous nature. Moreover, in tuberculous disease the vas is thickened, usually in a nodular manner. It is important to examine all the palpable part of the vas, for sometimes the nodules are limited to the inguinal region. Von Pirquet's tuberculin reaction is a valuable aid if its limitations are remembered. In striking contrast with this, syphilitic enlargement of the testicle leaves the epididymis unaffected, and is limited to the testicle, which enlarges unevenly, often affecting the tunica albuginea and the tunica vaginalis in a nodular manner. The syphilitic testicle rarely attains three times the natural size. It is curiously devoid of pain. The testicular sensation is often lost, and there is little or no thickening of the cord. Its anterior surface is uneven and may become adherent to the coverings, which may later ulcerate, and ultimately give rise to a *hernia testis* on the front of the swelling. This contrasts with the postero-infero-lateral position of tuberculous sinus or hernia testis. *Chronic torsion of the testis* is generally the result of a blow, or of an injury in the saddle; the symptoms may be obscure until the testicle begins to swell. Operation is generally resorted to with the idea that the condition is tuberculous or malignant, and even then the diagnosis may be in doubt until microscopical examination of the organ has been made.

It is often very difficult to distinguish syphilitic enlargement of the testicle from that due to *growth*; but a course of large doses of antisyphilitic remedies and the Wassermann reaction may settle the matter. Malignant new growth nearly always grows steadily, and being entirely within the tunica albuginea it maintains the shape and smooth surface of the testicle until it reaches a size much larger than that of a syphilitic testicle. Moreover, it causes much more pain, and usually some thickening of the cord, with later enlargement of the glands in the pelvis. In some cases the diagnosis between syphilitic testicle, growth, and hæmatocele may be so difficult and so urgently necessary as to demand an exploration.

Malignant growths of the testicle can be divided into four varieties: (*a*) Carcinoma; (*b*) Sarcoma; (*c*) Embryoma; (*d*) Endothelioma.

Carcinoma is far more common than sarcoma, although the contrary has been believed for many years, owing to the fact that many carcinomatous growths with small alveoli have been wrongly labelled sarcoma. The average age of patients with carcinoma testis is 43, and of those with sarcoma testis, 34. The average duration of carcinoma before operation is 1½ years; of sarcoma, 11 months. Sarcoma advances much more rapidly and kills earlier than carcinoma. The former disseminates through the veins, whereas the latter travels along the lymphatics and infects the lumbar glands. Embryoma is, according to Nicholson, "the commonest new growth of the testicle, but it is often overlooked." It can be shown to contain structures derived from all the three blastodermic layers of the embryo. The average age at the time of operation is 29, the average known duration before operation is 5½ years. "Although not necessarily malignant, it may produce metastases composed of all the tissues of the primary growth, or one tissue may become actively malignant, in which case the deposits will be formed of that tissue alone." It may spread along the lymphatics or disseminate through the veins.

5. **The Epididymis** may become enlarged as the result of (*a*) Inflammation ; (*b*) New growth ; (*c*) Cystic degeneration.

a. Inflammatory swellings are characterized by being elongated in a vertical direction ; by their relation to the testicle, which they overlap at its posterior border, and its upper and lower poles ; and lastly, by being flattened from side to side, so that the antero-posterior diameter is greatly increased. Inflammatory swellings may be :—(i) Gonorrhœal ; (ii) Septic, secondary to some other form of urethritis ; (iii) Tuberculous.

i. The *gonorrhœal* variety is distinguished by its acuteness, great tenderness, the surrounding œdema, and the bacteriological examination of the urethral discharge. Its onset is usually between the second and tenth week. Occasionally a subacute form develops later, at any time during the course of gleet. This is very difficult to distinguish from the tuberculous variety. Most cases of tuberculous epididymitis end in suppuration, but the gonorrhœal variety very rarely breaks down.

ii. The inflammation of the epididymis following other varieties of *urethritis* (such as ulceration near a stricture or due to impacted calculus, instrumentation, or prostatectomy), is often sufficiently indicated by the history if care be taken to go into this thoroughly. The swelling following prostatectomy is apt to suppurate. Some of these can be mistaken very easily for tuberculous disease.

iii. *Tuberculous epididymitis*, as a rule, is far more insidious and painless in its onset than other forms of epididymitis ; but it should not be forgotten that early subacute or even acute attacks of inflammation may accompany this disease, and that these are often the means of drawing the patient's attention for the first time to a disease which has been going on insidiously for some months. It has frequently been said that tuberculous nodules are limited to the globus major, and that those left after gonorrhœal urethritis are confined to the globus minor. It is more true to say that the latter are limited to the globus minor, whereas tuberculous disease may attack any part of the epididymis. Wherever the tuberculous disease starts, the inflammatory products soon spread through the thin fibrous capsule of the epididymis, and then gravitate towards the postero-infero-lateral corner of the scrotum, where adhesion occurs, followed later by an abscess and a sinus. In the diagnosis of tuberculous from other forms of epididymitis, the general state of health, and especially the presence or absence of other tuberculous lesions, are of great importance. Nodular thickening of the vas deferens and of the vesiculæ seminales and prostate are also valuable signs when the disease is well advanced. It should be remembered that the disease travels upwards along the vas, so that in its early and hopeful stages the upper part of the vas and vesiculæ seminales are not enlarged.

b. Primary new growth of the epididymis is excessively rare, so that it need not give rise to much concern in diagnosis ; it will generally be regarded as tubercle until after operation and microscopical examination of part of the tissue excised.

c. Cystic disease of the epididymis may occur in the form of : (i) Solitary cysts (*vide supra*) ; (ii) Multiple cysts. The latter condition rarely occurs except in men past middle age, and is analogous to cystic degeneration of the breast. The condition is almost painless and harmless. These swellings are translucent.

6. **Swellings of the Lower End of the Cord.**—The most important swelling of the lower part of the spermatic cord is *varicocele*. It is apt to be mistaken for omental hernia, but the mistake should never be made, because of the characteristic feel of the varicocele, and the reappearance of the swelling after it has been completely reduced and the finger is firmly pressed on the external abdominal ring.

7. **Urethral Conditions.**—Occasionally a *peri-urethral abscess* may form a swelling in the scrotum. Tenderness, œdema, and fluctuation, together with the history and evidence of urethral disease, serve to make the diagnosis clear. *Primary epithelioma of the urethra* is distinguished by the great pain and urethral obstruction that it engenders.

8. **Diseases of the Pubic Bones.**—Inflammatory products may travel into the scrotum from disease of the bones of the pubic arch, especially from the neighbourhood of the symphysis pubis. *Acute necrosis* of these bones is sufficiently indicated by the grave constitutional symptoms which always accompany it. *Caries* gives rise to more difficulty. The writer has known a case of tuberculous caries of the lower part of the symphysis pubis

in which the inflammatory products gravitated backwards and to the left, so as to form a large firm swelling in the left half of the scrotum, where it gave rise to much difficulty in diagnosis, and was thought to be either a sarcoma arising from the fibrous covering of the crus penis, or possibly a gummatous mass in the same situation. Sufficient attention was not paid to the fact that the man had chronic phthisis. *George E. Gask.*

SWELLING OF THE TONGUE is a condition the nature of which is generally obvious on inspection and palpation, if the history is taken into account at the same time, and many of the causes given in the following list need little detailed discussion :—

1. Causes of Acute Swelling of the Tongue:

- A bite or sting—wasp-sting for example
- Injury, for instance by a fish-bone, or by biting during an epileptic fit
- Angina Ludovici
- Corrosives or acute irritant applications
- Acute œdema, secondary to :—
    - (1). Inflammatory conditions within the mouth—Stomatitis (p. 542)
    - (2). The effects of certain drugs, especially mercury
    - (3). Erythema bullosum or pemphigus (p. 98)
    - (4). Variola
    - (5). Serum injections and other conditions liable to cause giant urticaria
    - (6). Angioneurotic œdema
    - (7). Raynaud's disease
- Hæmorrhage with swelling of tongue, as in scurvy, leukæmia and other causes of purpura (p. 552).

2. Causes of Chronic or Persistent Swelling of the Tongue :

(1). *Where the swelling is general :—*

- Macroglossia
- Cretinism
- Myxœdema
- Mongolian idiocy
- Acromegaly
- Chronic dyspepsia.

(2). *Where the swelling is local or asymmetrical :—*

- Irritation by a tooth-plate or decayed tooth
- Epithelioma
- Leukoplakia (chronic superficial glossitis)
- Gumma
- Tuberculous infiltration
- Actinomycosis
- Calculus in a sublingual salivary gland
- Ranula
- Suprahyoid cyst
- Angioma
- Sarcoma.

If the nature of the tongue enlargement is not obvious from the history and simple inspection and palpation, as will probably be the case when it is due to a *bite*, *sting*, *injury*, *corrosive* or *irritant* application, after the use of *mercury*, *serum*, or other drugs, *variola* or *pemphigus*—it may be so from the concomitant symptoms, as in the case of *cretinism* (p. 234), *acromegaly* (p. 237), *mongolian idiocy* (p. 190), or *myxœdema* (p. 38). The swollen tongue of *dyspepsia* is seldom very large, though it may cause the patient discomfort at times from the sense of its being too big for the mouth; it is seldom difficult to recognize from its pale flabby look and its marginal indentation by the teeth.

Simple *macroglossia* is rare ; when it does occur, the history is that it dates from youth or childhood, and the patient may otherwise be perfectly normal, unless he also has some other congenital peculiarity, such as macrocheilia (blubber-lips).

The chronic local lesions associated with swelling are in many cases associated with superficial ulceration, and the difficulties that may arise in distinguishing *simple*, *syphilitic*, and *epitheliomatous* trouble are discussed under ULCERATION OF THE TONGUE (p. 738). *Tuberculous* and *actinomycotic* lingual mischief are both very rare, and on that very account may be mistaken for malignant or syphilitic disease unless bacteriological or histological methods of diagnosis are resorted to. *Ranula* and *sublingual salivary gland calculus or cyst* both cause swellings that are beneath the front part of the tongue rather than in its substance ; generally bulging up one side of the floor of the mouth near the frænum linguæ. A ranula is a distended mucous gland, and after enlarging slowly to the size of a chestnut perhaps, it often ceases to grow further ; but it does not fluctuate in its dimensions in relationship to meals, like a salivary gland swelling often does.

A *suprahyoid cyst* is situated in the root of the tongue posteriorly, where it arises from remains of the obsolete thyroglossal duct. It is seldom large ; its nature may be suggested by its situation.

An *angioma* of the tongue is rare; sometimes, however, after remaining latent for years, it grows with rapidity and necessitates an operation. The diagnosis may be suggested by the colour of the tumour, but histological examination subsequent to removal may be required before one can be sure whether the tumour is a simple angioma, or whether it has taken on the malignant characters of an angiosarcoma or is a pure *sarcoma*.

Hæmorrhage in the substance of the tongue, with consequent great swelling of the organ and inability to use it for speaking or eating, may result from many of the different blood conditions that produce purpura. Only in very exceptional cases would such spontaneous bleeding be confined to the tongue, though conceivably this might be the first symptom in a case of acute lymphatic leukæmia, for example, or of purpura hæmorrhagica. Other hæmorrhages would follow, however, and indicate the need for blood-counts and other measures that are discussed under the heading of PURPURA (p. 552).

Raynaud's disease (p. 256) affects the fingers and toes more commonly than any other parts; it may, however, involve other distal tissues in a similar way, including the penis, the ears, the nose, and the tongue. In the latter it sometimes produces acute attacks of purple or almost black cyanosis, followed occasionally by local necrosis and subsequent scarring; acute swelling of the tongue may ensue when the paroxysm of vasoconstriction is passing off. I have never seen a case, however, in which the tongue alone was affected, and when the fingers and toes are attacked at the same time as the tongue the diagnosis is easy.

There remain for discussion acute œdema of the tongue due to *severe stomatitis*, *angioneurotic œdema of the tongue*, and *angina Ludovici*. The latter is an acute, virulent and generally fatal condition, in which streptococci or other organisms attack the floor of the mouth and root of the tongue and—without producing much pus, or even none at all—spread almost like wild-fire through the deeper structures of the mouth, throat, and neck, and cause extreme swelling of all the tissues in the neighbourhood; there is high fever, often a severe rigor, the patient is soon in a state of being hardly able to breathe, and extreme œdema of the glottis is apt to cause death from asphyxiation even when multiple incisions have been made into the brawny swollen parts. The condition is almost unmistakable; fortunately it is rare. It may be simulated by similar widespread œdema that results from infection of the deeper parts secondarily to one or other of the types of stomatitis discussed on p. 542; indeed, such stomatitis, when it has spread to the deeper tissues in this way, has virtually led to a secondary angina Ludovici; the latter name, however, is applied as a rule only to cases in which the acute overwhelming infection described above arises without any obvious preceding inflammation of the tongue or mouth.

Angioneurotic œdema of the tongue is rare, but it is of great importance because it is one of the purely functional conditions which may kill the patient. As a rule there is a history of similar attacks in other parts of the body previously (*Fig.* 178, p. 412), and other members of the family will be familiar with acute causeless swellings from personal experience, for it is a familial affection. Should it involve the tongue during a first attack, however, it would be mistaken for angina Ludovici very easily, especially as the patient may have pyrexia or a rigor notwithstanding the functional nature of the malady. Tracheotomy has been resorted to as the only means of saving the patient's life, and the diagnosis has only become clear when the œdema of the tongue and adjacent parts has subsided almost as rapidly as it came on, and the patient has had similar neurotic œdema, probably in other parts, on subsequent occasions.

Herbert French.

SWELLING, VULVAL.—The differential diagnosis of vulval tumours must necessarily include not only true swellings of the vulva, but also swellings which appear at the vulva as a result of the displacement of other structures, such as occur in prolapse and cystocele, and in addition lesions like kraurosis vulvæ, which are not strictly swellings at all. The lesions of the vulva may be tabulated under various headings, as set forth in the following scheme:—

Inflammatory Lesions.—

Simple vulvitis
Gonorrhœal vulvitis
Soft chancre
Papillomata

Syphilis:
  Hunterian chancre
  Condyloma
  Tertiary lesions
Tuberculosis

Furunculosis
Leukoplakic vulvitis
Kraurosis vulvæ
Pseudo-elephantiasis
Esthiomène.

Cystic Swellings.—

Hydrocele of the canal of Nuck
Sebaceous cysts
Mucous cysts
Implantation cysts
Dermoid cysts.

Blood Cysts.—

Varicocele
Rupture of a varicose vein
Traumatic hæmatoma.

New Growths.—

Caruncle
Fibroma
Lipoma
Angioma
Neuroma
Fibromyoma of round ligament
Endothelioma
Squamous-celled carcinoma (epithelioma)
Columnar-celled carcinoma
Sarcomata of various kinds.

Herniæ.—

Inguinal
Posterior labial
Perineal.

Displacement.—

Prolapse of urethral mucous membrane
Prolapse of uterus
Cystocele
Inversion of the uterus
Fibromyoma of the vaginal wall.

Unclassified.—Simple anasarca.

Certain of these lesions stand out pre-eminently as presenting difficulties in diagnosis. The general principles by which solid tumours are distinguished from cystic, inflammatory swellings from new growths, or new growths from herniæ, need not be insisted upon here. Perhaps the commonest difficulty which arises in practice is the diagnosis of gonorrhœal vulvitis from simple vulvitis, and also between the gonorrhœal soft chancre and the syphilitic condyloma, the latter differentiation being of much more practical importance than the former as far as the patient is concerned. In the acute stage of a *gonorrhœal vulvitis* there is a chance of recognizing the gonococcus in the discharge, if films made from it are suitably stained. Practically, *all* acute forms of vulvitis appear alike clinically, so that the recognition of the gonococcus becomes a matter of importance (see p. 185). In chronic gonorrhœal infections with vulval swelling, as a rule the organism cannot be found in the general vulval discharge, but might be found in the urethra or in the cervix. A gonorrhœal infection may be suspected if the patient gives a history of an acute onset, accompanied by scalding on micturition, and when there are redness of the orifices of Bartholin's glands, and much redness and swelling of the carunculæ myrtiformes. Papillomata or warts of the vulva may occur also in chronic gonorrhœal infections, and there is no evidence of a reliable nature to show that they occur in any other kind of infection.

The *soft chancre of gonorrhœa* may be mistaken for the *condyloma of secondary syphilis*, but as a rule this difficulty should not occur. The soft chancre is a typical punched-out ulcer with a somewhat red base and clean edges, discharging pus. The condyloma, on the other hand, is a raised, flat-topped excrescence, with sodden, epithelium-covered surface. Soft chancres are not very numerous, as a rule, and are generally limited to the vulva. Condylomata are numerous, and may occur all over the labia, around the anus, and even on the skin of the thighs and gluteal region. Condylomata are from the start, or very soon after, accompanied by a sore throat and a typical papular skin rash, for they are secondary syphilitic lesions. Soft chancres clear up with antiseptics; condylomata persist for long periods, but clear up in two or three weeks as a rule under mercurial treatment or salvarsan. It must not be forgotten that soft sores and condylomata may occur together in the same patient, in which case the diagnosis may be still more difficult.

Another practical differentiation which gives rise to anxiety is that between the *Hunterian chancre* or primary syphilitic sore, and *squamous epithelioma* of the vulva. This is a question which is of vital importance to the patient if valuable time is not to be lost in the treatment of a malignant epithelioma. The two lesions look much alike at first; they form raised hard indurated masses in the skin, which may ulcerate quickly as a result of necrosis of the superficial portions. Both give rise to a thin watery discharge, and to enlarged glands in the inguinal region which do not suppurate at first, but may do so later in the case of an epithelioma. It must not be forgotten that a primary chancre is very seldom seen in women, whilst squamous epithelioma is relatively common. Of course the chancre will be followed in due course by secondary lesions, but it is not safe to wait for these to appear in a doubtful case. The only reasonable way to deal with such a case is to excise the doubtful swelling at once and submit it to microscope examination by an expert. A squamous epithelioma is easily detected in this manner in quite early stages, and does not in the least resemble a syphilitic lesion microscopically. The *Spirochœta pallida* may be recognized in scrapings of a hard chancre by the Indian ink method, or when fixed and stained by Giemsa's or Levaditi's methods (*Plate XXVIII, Fig. J*, p. 614). In sections, too, the spirochæte may be demonstrated, but it must be remembered that for this purpose the excised growth *must* be fixed in 5 per cent formalin solution. Wassermann's serum test may assist the diagnosis.

Tertiary syphilitic lesions are by no means common on the vulva. When they do occur they give rise to spreading ulceration with great destruction of tissue, and scarring in the older healed portions. Here, the only likely lesions to be mistaken are some forms of epithelioma, and tubercle. Obviously, in such conditions the only reliable method of diagnosis is to be found in excision of parts of the lesion and microscopic examination of sections made from them. The disease known as *esthiomène* is probably a tertiary syphilitic affection.

Pseudo-elephantiasis of the vulva is usually a syphilitic affection of the labia minora, giving rise to great enlargement, with a rough and thickened appearance of the skin. It could only be mistaken for real elephantiasis due to lymphatic obstruction by the *Filaria sanguinis hominis* (*Plate XXVIII, Fig. F*, p. 614), a disease which is practically never seen in this country.

Unilateral œdema of a labium minus is a fairly common condition, and is usually associated with an infected wound or with a primary syphilitic chancre. *Bilateral œdema* is almost always associated with general anasarca, the result of renal disease, cardiac disease, or pressure upon pelvic veins. It is not likely to be mistaken for any other disease.

Leukoplakic vulvitis and *kraurosis vulvæ* have certainly been confounded with one another clinically, and also in the published descriptions of the lesions. In the former the labia majora and minora and the prepuce of the clitoris are affected, whilst the vestibule always escapes. In the latter the lesion affects the vestibule, the orifice of the vagina, and the labia minora. There is much greater contraction of the vaginal orifice in kraurosis. Leukoplakia often precedes a squamous epithelioma; kraurosis is said not to do so. Leukoplakia occurs at all ages, whilst kraurosis is a disease of post-menstrual life. Leukoplakic vulvitis appears as a white sodden hardening of the skin, with flattening and shrinkage of the labia. Kraurosis at first looks red and swollen, but later takes a yellowish tinge. Leukoplakia causes intense itching; kraurosis gives rise to great pain and tenderness, with a very severe form of dyspareunia.

Apart from a cyst developing in Bartholin's gland or duct, cystic swellings of the vulva are not common. A *Bartholinian cyst* is recognized by its position on one side of the vaginal entrance, distending the posterior part of the conjoined labia, and also within the hymeneal ring. As a rule the orifice of the gland can be seen on the inner side of the cyst. The contents of this form of cyst may be glairy mucoid fluid, or pus. In practice, a Bartholin cyst is not likely to be mistaken for anything else; but it is wise to remember that the *posterior labial hernia* occurs in the same situation, and that new growths of the vulva may occur there as elsewhere. Bartholin cysts are always the result of infection, and as a rule a history of vulval inflammation can be obtained.

Varicocele of the vulva occurs practically only in connection with pregnancy, and is unmistakable. It has the same 'bag of worms' feel as a varicocele in a man, and as the veins are close to the skin a bluish colour is always to be noted. It is attended by much aching pain, especially on standing. The veins are degenerate, and liable to rupture as a result of labour or traumatism.

Hæmatoma of the vulva is recognized as a blue or violet-coloured swelling covered by tense shiny skin, and often spreading up into the pelvis by the side of the vagina. The history alone will often decide the nature of the swelling, but the appearance is quite typical as a rule. Hæmatoma of the vulva may occur apart from pregnancy, and then is always traumatic.

Urethral caruncle and *prolapse of the urethral mucous membrane* may be mistaken for one another. The former, however, is always a pedunculated or sessile new formation, invariably springing from the posterior wall of the urethral orifice. It bleeds readily, is often, but not always, exquisitely painful, and is usually the result of infection. Prolapse, on the other hand, appears as a raised projection with rounded margins, and with the urethral canal in the centre as a dimple. The prolapsed portion may not necessarily include the whole ring of the mucous membrane. It may give rise to pain, and being always more or less strangulated, it is prone to bleed, much in the same way as a caruncle. It occurs as a result of some straining effort, or may accompany pelvic floor prolapse ; it is not the result of infection.

The differential diagnosis of the new growths of the vulva presents no points of difference from their diagnosis in other parts of the body. The only common benign tumour is the *pedunculated fibroma*, or *molluscum fibrosum*, whilst *squamous carcinoma* (*epithelioma*) is the only malignant growth which occurs at all frequently.

If the general characters of a *hernia* are borne in mind, there should be no risk of overlooking or mistaking any of the varieties which occur in the vulva. The resonance on percussion if the hernia contains bowel, the reducibility of the contents, and the protrusion through a pre-existing opening, will usually suffice to distinguish herniæ from other swellings. An obstructed or strangulated hernia is not so easy to recognize, but the accompanying acute symptoms and the previous history usually suffice to make the case clear.

Hydrocele of the canal of Nuck, an uncommon condition, may be mistaken for an inguinal hernia ; but as a rule it is irreducible, definitely fluctuating and circumscribed, and has no obvious neck running into the inguinal canal. When the canal of Nuck has a patent peritoneal communication the swelling disappears as the patient lies down, but it is not reducible in the characteristic manner of a hernia. Such a condition is very rare.

The *displacements* included in the list above are all dealt with under the heading of PROLAPSE OF THE UTERUS (p. 538). *Thos. G. Stevens.*

SYNCOPE.—(See COMA, p. 117.)

TACHE CÉRÉBRALE is the term used to denote that condition in which, after the finger has been drawn with moderate firmness across the patient's skin, the line along which it has passed becomes of a bright red colour from dilatation of the superficial arterioles and capillaries ; the phenomenon develops within thirty seconds or a minute of the finger stroke, and the red mark remains evident for two or three minutes, or more. If letters or figures are marked out on the skin in this way, they appear as though they had been written in red, so that the condition has also been termed dermatographia. It was at one time thought to be a characteristic symptom of tuberculous meningitis, but not only is it sometimes absent in cases of the latter, but it is also present in a very large number of other different conditions, and sometimes in perfectly healthy people. All forms of meningitis may give rise to it, so that it is not even a means of distinguishing one type from another. It is seen in an extreme degree in cases of urticaria, particularly the factitious variety in which numerous wheals may develop as the result of hardly more than ordinary touching of the skin. A similar condition has sometimes been observed in the later stages of severe febrile illnesses in general. *Herbert French.*

TACHYCARDIA, or abnormal rapidity of the heart's action, might, strictly speaking, be held to include every condition under which the pulse-rate is faster than the normal ; but by common consent it is restricted for clinical purposes to cases in which there is no pyrexia. Nearly all fevers produce undue rapidity of the heart's action, though some, such as typhoid fever, tuberculous meningitis, cerebral abscess, yellow fever, and influenza, do so to a much less extent than others. The rapid heart-action of fevers, however, does not generally come into one's mind when one uses the term tachycardia ; indeed the latter is chiefly employed for conditions in which it is rapid without there being anything which at first sight would seem to be a sufficient cause. Probably the best example of it is to be found in cases of pronounced *Graves's disease.*

The following is a list including this and some other causes of tachycardia :—

Graves's disease or exophthalmic goitre
Paroxysmal tachycardia
Nervousness and excitement

Exertion, especially when the patient is out of training or anæmic
Tobacco heart
Mitral stenosis
Pneumogastric irritation by :—
  Caseous glands | Mediastinal fibrosis | Thoracic aneurysm | Thoracic new growth.

Pneumogastric 'neuritis' after :—
  Diphtheria, influenza, and other microbial affections.

Drugs :
  Digitalis | Alcohol | Belladonna | Thyroid extract.

The four classical symptoms of *Graves's disease* are : A staring appearance of the eyes, generally spoken of as exophthalmos, though there need be no actual protrusion of the eye-balls (***Fig.*** 222, p. 527) ; moderate and almost symmetrical enlargement of the thyroid gland ; a pulse-rate between 120 and 180 per minute—usually about 140 when the attack is moderately severe ; and extreme nervousness, with fine tremor of the outstretched fingers. When all these symptoms are present at the same time, there can be no doubt as to the diagnosis, but very often some of them are absent, and it is possible for tachycardia to be the only symptom of the disease ; indeed, in a patient, particularly a woman between twenty and forty years of age, a persistent pulse-rate of over 120 would arouse serious suspicion that the case was really one of Graves's disease, even if the other three classical signs were absent.

Paroxysmal tachycardia should be distinguished at once from Graves's disease in which tachycardia alone has developed, by the fact that the tachycardia is not persistent, but recurs periodically with intervals of normal pulse-rate ; the patient is more often a woman than a man, and may have long periods of perfect health ; almost suddenly, the result sometimes of a fright or shock, sometimes without apparent cause, there is a sense of something being the matter in the precordial region, amounting as a rule to little more than a fluttering or palpitation, together with a feeling of faintness and lack of strength, and perhaps of numbness or of pins-and-needles in the extremities ; when examined the patient may present no other abnormality than a pulse-rate of perhaps 160 or even 200 to the minute (***Fig.*** 289). The attack may last a few minutes, or an hour or two, or for days, or

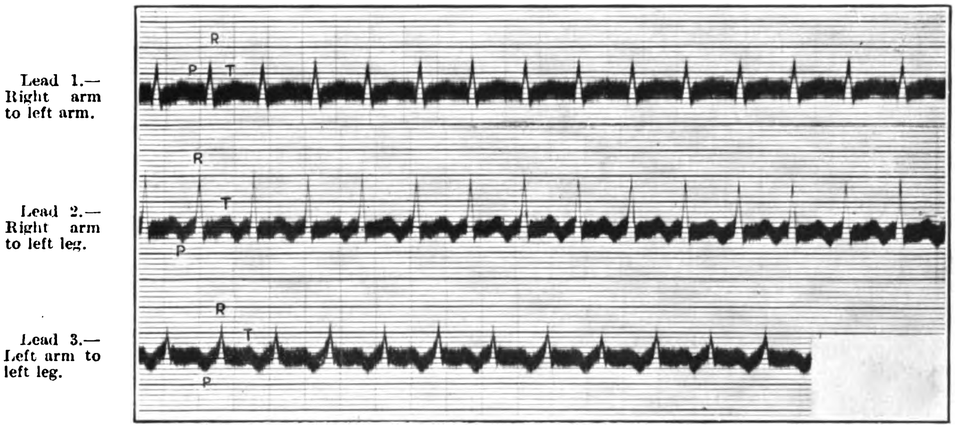
Lead 1.—Right arm to left arm.
R
P
T
Lead 2.—Right arm to left leg.
R
T
P
Lead 3.—Left arm to left leg.
R
T
P

Fig. 289.—Electrocardiogram in a case of paroxysmal tachycardia in a man. Lead 1 is from right arm to left arm : Lead 2 from right arm to left leg : Lead 3 from left arm to left leg. P = Auricle wave : R. T = Ventricular wave. Time marking in $\frac{1}{5}$ seconds. At first sight the tracing looks normal, but when time relationships are considered, it will be seen that there are 16 heart-beats in each 23 one-fifths of a second, so that the heart is making 208 beats per minute. The tracing is from a young man seemingly in perfect health, who had presented himself for examination as a recruit. It was found that when he had been lying perfectly still for a little time his pulse-rate was 72 per minute, but upon any exertion, such as holding his breath as long as he could, stooping to touch his toes, or on walking about, he felt a curious sensation in his chest lasting for a few minutes, and simultaneously the pulse-rate rose to over 200 per minute, auricles and ventricles both contracting at this rate. In spite of this he was able to do ordinary work ; there was little breathlessness : and there was no trace of any Graves's disease. The condition appeared to be one of simple paroxysmal tachycardia.

more rarely for weeks; it is apt to cease as suddenly as it began, and a similar attack is almost certain to recur after a longer or shorter interval—the main symptom of the complaint being summarized by the title 'paroxysmal tachycardia.'

The very rapid heart action that may be produced by *nervousness*, or *excitement*, or by some ordinary exertion such as coming rather rapidly upstairs when one is *out of training*, or when the patient is suffering from *anæmia*, or during *convalescence* after an illness, or after the over-use of *tobacco*, is a familiar phenomenon; the tachycardia disappears rapidly when the patient rests, and the diagnosis is not as a rule difficult. If ordinary resting for a while does not cause the rate of the heart-beat to return nearly or quite to normal, there may be doubt as to the diagnosis, unless the patient can be re-examined on another occasion; if there is persistent tachycardia a suspicion of Graves's disease will be aroused, or there may have been acute overstrain of a weakened *fatty*, *fibroid*, *rheumatic*, *alcoholic*, or *syphilitic* myocardium, followed by long-continued tachycardia without bruit, but perhaps with *auricular fibrillation*; electrocardiograms may be required before an exact diagnosis can be arrived at in many of these cases.

Mitral stenosis is of all the valvular lesions of the heart the most liable to lead to rapidity of the heart's action; but it seldom happens that the pulse-beat is fast until there has been other evidence of failure of the cardiac compensation. The diagnosis will generally be obvious from the history of acute rheumatism or chorea, the typical facies and malar flush, and the cardiac bruits.

It is exceedingly difficult to be certain of a diagnosis of *irritation of a pneumogastric nerve* within the thorax unless the existence of mediastinal *new growth*, *aneurysm*, or *fibrosis* is already known on account of the abnormal physical signs, the *x*-ray appearances, the visible tumour, or the varicose distention of the superficial thoracic veins; if an intrathoracic abnormality is known to exist, and tachycardia becomes a prominent feature of the case, it will probably be due either to mechanical interference with the heart's action or to similar interference with one or other vagus nerve. *Caseous glands* irritating the pneumogastric nerve are still more difficult to be sure of; but occasionally one ventures upon this diagnosis when a child who has been fed on untested or unsterilized cow's milk, develops obscure ill-health associated with persistent tachycardia. Such diagnosis would be still further suggested if there were at the same time enlarged glands in both sides of the neck, if there were pyrexia without any obvious explanation of it, or if there were any evidence of obstruction to the right bronchus, for the right bronchial gland is enlarged far more often than is the left. An *x*-ray examination may serve to confirm the suspicion (*Fig.* 61, p. 149).

Diphtheria, *influenza*, and possibly other microbial infections, are occasionally followed by marked and persistent tachycardia during convalescence, or even for weeks, months, or years afterwards. After diphtheria the condition is generally fatal. Influenza is always a dangerous diagnosis because it is so difficult to establish, but in certain cases in which the original diagnosis has been influenza, tachycardia to the extent of 200 heart-beats per minute may be present for months without the patient suffering from any severe cardiac symptoms, and the condition ultimately terminates in recovery with a return of the heart-beat to the normal rate. Precisely what is the nature of these cases it is impossible to say, but it has been thought by some that the symptom is due to inflammatory changes in the pneumogastric nerve, produced by whatever one means by the toxins of the disease. Whether this be so or not, the fact that persistent tachycardia may arise out of febrile illnesses should be borne in mind.

There are certain drugs which cause the heart's beat to be very rapid, the best known perhaps being *digitalis*, *belladonna*, *thyroid extract*, and *alcohol*. Certain patients suffering from cardiac symptoms seem unable to bear digitalis, the heart being driven into the condition spoken of as delirium cordis, though the reverse effect—slowing of the heart, bradycardia—is to be expected in persons who take digitalis well over a long period. When alcohol is the cause of the tachycardia, the fact may be obvious, the only difficulty arising in patients, mainly women, who may be regarded by all as entirely above reproach, but who nevertheless may be addicted to secret drinking. Belladonna in small doses slows the heart, but there are great variations in the degree to which different patients tolerate this remedy, even pharmacopœial doses sometimes producing toxic symptoms of which tachycardia is one. Widely dilated pupils and dryness of the tongue will help to point to the diagnosis in cases in which the belladonna is taken otherwise than medicinally. Tachycardia is the chief symptom by which one recognizes that a patient for whom thyroid extract has been prescribed is receiving too large a dose.

Herbert French.

TALIPES.—(See CLUB-FOOT, p. 111.)

TASTE, ABNORMALITIES OF.—Abnormalities of taste may be grouped under three main headings, namely: (1) *Impairment or loss of ordinary taste sensations*; (2) *Perverted taste sensations*; (3) *Sensations of a foul taste in the mouth.* The following conditions may produce these:—

1. Impairment or Loss of Taste (*Ageustia*):

(*a*). *Due to nerve lesions*:

Paresis or paralysis of the lingual branch of the fifth nerve
Paralysis of the facial nerve, including the chorda tympani
Glosso-pharyngeal nerve paralysis
Bulbar paralysis
Cerebral tumour, especially of the uncinate gyrus
Hysteria.

(*b*). *Due to affections either of the mouth or nose*:

A common cold
Hay fever (coryza e feno)
Atrophic rhinitis
Hypertrophic rhinitis
Nasal polypus
Adenoids
Other varieties of nasal obstruction
Bromism
Iodism
Mercurial and other varieties of stomatitis (p. 542).

(*c*). *Febrile conditions*, especially when associated with coating of the tongue.

(*d*). *After destruction of the nerve endings* in the tongue by corrosives taken accidentally or with suicidal intent.

2. Perverted Taste Sensations (*Parageustia*):

Pregnancy
Hysteria
Epileptic aura
Insanity.

3. Foul Taste in the Mouth (*Cacogeustia*):

(*a*). *Local conditions of the mouth or nose*:

Caries of the teeth
Retention of food particles between healthy teeth
Furred tongue from any cause
Excessive smoking
Mouth breathing at night
Gumboil
Septic stumps under tooth-plate
Gumma of the tongue or palate
Epithelioma of the tongue or mouth
Stomatitis from any cause (p. 542)
Septic infection of the antrum of Highmore, or an ethmoid, sphenoid, or frontal sinus.

(*b*). *Severe fevers* associated with dryness of the mouth and coating of the tongue, especially in:—

Pneumonia
Typhoid fever
Peritonitis
Septicæmia, etc.

(*c*). *Septic lung conditions*, especially—

Phthisis, with secondary infection of cavities
Bronchiectasis
Bronchiolectasis
Fœtid bronchitis
Gangrene of the lung
Empyema ruptured into the lung
Liver abscess ruptured into the lung
Subdiaphragmatic abscess ruptured into the lung.

(*d*). *Certain drugs or poisons*, especially—

Mercury
Copper
Arsenic
Lead
Iodides
Bromides
Sulphides
Paraldehyde
Asafœtida
Creosote
Guaiacol
Valerian
Cod-liver oil
Castor oil.

(*e*). *Certain foods*, such as:—

Garlic
Onions
Leeks
Shallots.

(*f*). *Hysteria and functional conditions.*

From a diagnostic point of view, impairment of taste sensations is of importance only in rare cases. When the impairment is unilateral, it is almost certainly due to a lesion involving either some portion of the third branch of the *fifth nerve*, the *chorda tympani*, or the *glosso-pharyngeal nerve*. It is not often difficult to distinguish between these three. If the chorda tympani is involved, it is almost certain that the facial nerve will also be affected upon the same side, and this will be evidenced by paresis or paralysis of the face of the infranuclear type (p. 493); there may be excessive watery secretion from the submaxillary gland upon the same side; the commonest condition to cause these symptoms is disease of the middle ear extending to the Fallopian canal. If it is found that taste is impaired only in the posterior third of the tongue upon one side, the lesion probably affects the glosso-pharyngeal nerve, and it is very possible that there may be paresis of the same side of the palate or partial paralysis of the pharynx at the same time. When the lingual branch of the fifth is involved, the impairment of sensation is in the anterior two-thirds of the tongue on the same side. The lesion may be a tumour or an injury affecting the lingual nerve in the mouth; or it may be part of a more general affection of the fifth nerve of that side, with corresponding interference with cutaneous sensibility of more or less of the skin of the face, according to the extent to which the different branches of the fifth nerve are involved; if the motor root is affected, the fact can be ascertained by feeling the masseter and temporal muscles, which, when the patient clenches his teeth, do not harden so much on the affected as on the sound side.

When sensation on both sides of the tongue is affected, it is possible that the lesions described above may be bilateral; but it is much more likely that the defect is then not primarily nervous, unless it is due to *bulbar paralysis*, the progressive labio-glosso-pharyngo-laryngeal weakness of which is pathognomonic.

When the cause of impaired sensation is in the nose, as in the case of *coryza*, *rhinitis*, *polypi*, or *adenoids*, it will be found that some substances can be tasted easily and others not at all; this depends upon the fact that taste consists of two parts, flavour and savour; savour depends upon sensation transmitted by the olfactory nerves—the so-called taste of roast beef for instance; savours will be defective when the nose is the cause of abnormal taste-sensations; while flavours such as the taste of sugar, gentian, or salt, which are transmitted by the gustatory nerves of the tongue, will still be fully preserved.

The differential diagnosis of the other conditions enumerated in the above list need not be detailed, for the conclusion come to will depend upon the result of careful inquiry into the history, investigation of the abnormal physical signs, and the other symptoms in the case. One would only emphasize the possibility of caries, or decomposing food between teeth that superficially look sound, or septic infection of the accessory sinuses of the nose being long overlooked, though in each case abnormal taste sensations may be prominent. *Herbert French.*

TEETH, GRINDING OF.—(See GRINDING OF THE TEETH, p. 265.)

TENDERNESS IN THE CHEST implies that pain is felt when some part of the chest wall is touched or pressed; it is a common symptom; in some instances the pain felt is a direct pain, due to stimulation of sensory nerves actually in the diseased area; in others—perhaps the majority—the pain is a referred pain ('somatic pain'), felt in the skin and subcutaneous tissues that are tender, but due to a visceral lesion remote from the tender area.

CAUSES OF TENDERNESS IN THE CHEST.

These may be classified according to the situation of the lesion to which it is due.

1. **Lesions of the Chest-Wall:** the pain is for the most part direct:—

- Inflammations of the skin and underlying tissue
- Intercostal myositis
- Myalgia
- Pleurodynia
- Affections of the ribs and sternum
- Blood diseases
- Intercostal neuritis
- Injury of the intercostal nerves
- Intercostal neuralgia
- Hysteria
- Herpes zoster
- Pleurisy
- Mediastinal disease
- Pericarditis.

2. **Lesions of Thoracic and Abdominal Viscera**: the pain is usually a referred pain; felt in lesions of the—

Lungs	Diaphragm	Liver.
Heart and aorta	Stomach and œsophagus	

Lesions of the Chest-Wall.—Tenderness in the chest is probably the chief complaint in *superficial inflammatory lesions* of the chest wall, such as bruises, burns, cuts, mastitis, and superficial infections of all sorts, the diagnosis of which will probably leap to the eye, and need not be discussed further.

Pain will be the chief complaint in *intercostal myositis*, often vaguely called rheumatic, that occurs after chill or strain of the intercostal muscles; but the affected muscles will also be tender on pressure, the tenderness being in the deeper structures, not in the superficial tissues. The condition is also known as *intercostal myalgia* or *pleurodynia*; it has to be distinguished from pleurisy by the absence of friction-sounds on auscultation; and from disease of, or pressure on, the intercostal nerves. No doubt the tenderness is due to irritation of the sensory fibres in the intercostal muscles. Similar, but more transient, pain and tenderness may be met with in the *stitch* to which the untrained athlete is prone.

Tenderness in the chest may result from *disease* or *injury of the ribs or sternum*, when it will be localized to the injured spot; fracture, inflammation, or new growth may be the immediate cause. If *fracture* is present, a history of injury should be obtainable; the *x*-rays may show the fracture; or crepitus between the fragments on movement, or deformity may be made out. *Sternal* or *costal ostitis*, or *periostitis*, may follow injury; or occur in the course of such diseases as enteric fever, tuberculosis, pyæmia or septicopyæmia; the local signs of inflammation (pain, redness, heat, swelling) and the general condition of the patient should make the diagnosis fairly simple. Tenderness in the chest due to *new growth* in the ribs or sternum—such as hydatid, sarcoma, secondary deposits from carcinoma—is a rarity that need only be mentioned. Tenderness of the ribs and sternum, as well as of the long bones of the limbs, is not uncommon in certain *blood diseases*, in which hyperplasia of the red marrow, or excessive accumulation of white cells in it, may occur: such as pernicious anæmia or leukæmia. The diagnosis here must be made on the results of examination of the blood (p. 24). In all these instances the tenderness is deep, and due to irritation of the sensory nerves of the periosteum or bone; the pain felt on pressure is a direct pain.

Tenderness at certain points of, or all along, the course of an *intercostal nerve* is common in various affections of these structures. The particularly tender spots are three in number, and correspond to the points at which the posterior primary, the lateral cutaneous, and the anterior cutaneous branches are given off, near the spinal column, the mid-axillary line, and the sternal margin, respectively. Such tenderness may be marked in *intercostal neuritis*, which is rare; in *intercostal neuralgia*, which is often diagnosed when some more serious intrathoracic disorder is really present, such as pneumonia or pleurisy; and in cases of pressure on an intercostal nerve, such as may be set up by *abscess* about the spinal column, *aneurysm* of the descending aorta, or *new growth* invading the spinal canal. Whenever a patient complains of severe or obstinate pain and tenderness in the side, careful and repeated physical and *x*-ray examinations should be made, the possibility that some such deep-seated disease may be present being kept in view before the diagnosis of intercostal neuralgia, or of functional nervous disease (hysteria), is made. In exceptional cases of *hysteria*, zones of tenderness in the chest, possibly, too, Charcot's spasmogenic zones, may be found.

Pain and tenderness along an intercostal nerve are common in *herpes zoster*, and may be present before, during, and after the appearance of the characteristic rash. The tenderness often has the three spots of maximum development mentioned above; it is particularly when it occurs in the second half of life that herpes may be followed by a long period of pain and tenderness along the course of the affected nerve. Until the rash has appeared, or in the comparatively infrequent cases when the rash leaves no scarring behind it, the diagnosis of herpes may be difficult; the rash, once seen, can hardly be mistaken.

Lesions of the Underlying Viscera.—Tenderness in the chest is very frequently a symptom of disease in the underlying viscera, thoracic or abdominal, when the pains to which it gives rise are in most cases referred pains. The tenderness is therefore as a rule superficial, confined to the skin and subjacent areolar and fatty tissues; if these can be drawn aside, pressure can be made on the deeper tissues that normally underlie the tender area without provoking pain. Properly speaking, 'tenderness in the chest' can only refer to tactile hyperæsthesia, or the eliciting of pain on pressure, whether light or heavy. Such tactile hyperæsthesia, or the production of unpleasant sensations or pain by the very lightest touch, is common in neuralgia and in neuroses, or in cases of referred pain. But a similar hyperæsthesia for cold, or less often for heat, sometimes occurs in the chest—in tabetic patients, for example; this may perhaps be regarded as a special form of 'tenderness.' In the same way hyperæsthesia for pain, or hyperalgesia, in which a normally painless stimulus or impression becomes transformed into an acutely painful sensation, is to be regarded as a form of 'tenderness' in the chest. Further, perversions of sensation sometimes occur in organic nervous diseases, such as syringomyelia or tabes. Thus, tenderness may be elicited by the continuous application of a pressure that is painless if applied only for a short time (summation of painful stimuli); or the pain may be first felt some little time after the application of the stimulus to the tender area (retarded sensation).

Tenderness of the chest is a common complaint in *pleurisy*. The physical signs should suffice to make the diagnosis simple if a careful physical examination be made. The tenderness is deep as a rule, and not in the skin and loose subcutaneous tissues.

The sternum may be tender in the rare cases of *mediastinal inflammation* or *tumour* that are met with from time to time; tenderness and direct pain may similarly be caused by the pressure of *aneurysms* on the internal surface of the chest-wall. The diagnosis in these cases must be made on the results of the physical and *x*-ray examination of the patients.

Tenderness with pain over the precordia is fairly common in *pericarditis*, diagnosed by the canter rhythm and the pericarditic rub. It may be so extreme as to preclude percussion or a satisfactory physical examination. Similar pain and tenderness have also been found at the epigastrium and the upper costal angles in these cases; due, perhaps, to involvement of the diaphragm in the inflammatory process.

Chest tenderness is not rare in cases of *acute* or *chronic disease of the lungs*, particularly *tuberculosis*; in these, it is hard to be sure that one is not dealing with referred pains due to old or recent pleurisy or pleural adhesions. The tenderness may be either superficial or deep; sometimes it is so marked as to be elicited even by the pressure of the clothes. It is generally felt most about the region of the apices of the lungs, the curve of the shoulder, or the scapula. It is often a very chronic trouble, vanishing during periods of general improvement, returning again when the patient's health is low or the pulmonary lesion is progressing. Similar tenderness is often met with in *acute bronchitis*, or with *chronic bronchitis and emphysema*; the diagnosis must be made on general lines. It must be remembered that identical areas of referred chest-tenderness may be observed in disorders of such various organs as the heart, lungs, liver, and stomach; and that a patient may be long treated for 'rheumatism' of the shoulder, for example, when he is really suffering from such widely different disorders as tuberculosis, gall-stones, gastritis carcinoma mammæ, or coronary artery sclerosis.

Direct tenderness about the precordia is sometimes prominent in *heart disease*: as a rule, however, the tenderness is due to hyperæsthesia of referred origin. It is most marked in *angina pectoris*, and often persists after the anginal pains have passed off. Both the pain and the tenderness are felt within the area of distribution of the first to the eighth dorsal nerve-roots; the roots usually receiving the first and most intense impressions are the second dorsal. The left ventricle, the commonest primary seat of pain, is in relation with the second to the fifth dorsal nerve-roots; the auricle with the fifth to the eighth; the ascending aorta with the third and fourth cervical and the first to the third dorsal. These nerve-connections explain the extensive radiation and wide distribution of the tenderness and pain in the superficial tissues that may form such prominent symptoms of heart disease; for the chest, neck, and arm may all be affected. The tenderness of angina pectoris commonly occupies the same areas as the pain, takes the form of a soreness, smarting, or of hyperalgesia to touch, and may last for days after the pain is over. In some cases, touching or stimulating the hyperalgesic area on the chest, arm, or neck, may reflexly induce an anginal attack—even the pressure of a stethoscope applied for auscultation may suffice—which is a strong argument for regarding the tenderness as a viscero-sensory reflex or a referred tenderness. Such anginal attacks and tenderness are commonest in coronary sclerosis, aortic aneurysm, aortic reflux, and acute aortitis; they may also be seen in any form of heart-disease in which hypertrophy and dilatation have taken place, and the heart has to do more work than it can manage, for example with raised blood-pressure and arteriosclerosis, or with adherent pericardium. In well-marked cases, the cardiac origin of areas of tenderness in the chest should not be difficult to diagnose, owing to their association with severe anginal pains on the one hand, and with the fact that the pain is brought on by exertions or emotions that increase the work of the heart. Identical areas of tenderness may be found in pleurisy or chronic pulmonary tuberculosis; but here the pain will be connected with respiration or coughing in an unmistakable manner, and there will be the history and signs of pulmonary rather than of cardiac disease. Identical areas of chest tenderness may be found in diseases of the stomach, in the areas of distribution of at any rate the fourth and fifth dorsal nerves; the diagnosis here will turn on the history of gastro-intestinal disorder, and on the radiation of the pain and the discovery of tenderness in the epigastrium.

Tenderness in the chest may result from *injuries* or *inflammations of the diaphragm*, the lower costo-chondral margin being affected. The diaphragm is innervated by the phrenic nerves mainly, and so is connected with the third, fourth, and fifth cervical nerve-roots; accordingly, referred diaphragmatic pain and tenderness may also be felt in the top of the shoulder, an area innervated by the fourth cervical nerve. In most cases, the tenderness of these areas will be due to *diaphragmatic pleurisy*.

Diseases of the stomach, particularly *gastric ulcer* and *flatulent dyspepsia*, may give rise to pain and tenderness in the chest that may be very hard to distinguish from those due to cardiac disease. As a rule, the history of gastric disturbances should be of great assistance in coming to a correct diagnosis; although it must be remembered that flatulence and temporary gastric upsets are seen not infrequently in true angina pectoris. Further, the pain and tenderness due to diseases of the stomach are mainly abdominal, are in the epigastric and left hypochondriac regions, and in the lower half of the back of the chest; whereas in cardiac disorders they are characteristically situated higher up in the chest and back. Electrocardiograms may be required to show whether the heart action is normal or not.

It is possible that disease or painful stimulation (as by hot drinks) of the *œsophagus* may produce an area of referred tenderness in the chest, over the lower third of the sternum and in the middle line, in correspondence with the pain that is felt here in these conditions.

Tenderness in the right side of the chest near the costal margin is not rare in *diseases of the liver* and *gall-bladder*, corresponding to the cutaneous distribution of the seventh, eighth, and ninth dorsal nerves; for the most part, however, the pain and tenderness are in the epigastrium and the right hypochondrium. The right phrenic nerve (third to fifth cervical) sends twigs to the liver and gall-bladder, so that tenderness and pain may also be felt in the right shoulder, just as they may be in disorders of the diaphragm. It is particularly in cases of gall-stone or biliary colic that these areas of tenderness are likely to be found. In patients with hepatic abscess, the spread of inflammation to the chest-wall may give rise to direct pain and tenderness in the chest, with the development of characteristic local and general symptoms and signs; the diagnosis here will have to be made from such things as axillary abscess, empyema making its way through the chest-wall, or abscess arising in the chest-wall. *A. J. Jex-Blake.*

TENDERNESS IN THE EPIGASTRIUM.—(See PAIN IN THE EPIGASTRIUM, p. 436.)

TENDERNESS OF THE HYPOCHONDRIUM.—(See PAIN IN THE HYPOCHONDRIUM, p. 450.)

TENDERNESS IN THE ILIAC FOSSA.—(See PAIN IN THE ILIAC FOSSA, p. 452.)

TENDERNESS IN THE JOINTS.—(See JOINTS, AFFECTIONS OF THE, p. 337.)

TENDERNESS IN THE LIMBS.—(See PAIN IN THE LIMBS, GENERAL, p. 463; and SENSATION, SOME ABNORMALITIES OF, p. 604.)

TENDERNESS IN THE SCALP occurs in two main varieties :—

1. **Direct Tenderness,** due to injury or disease, such as—

Bruising or infected wounds
Inflammation or suppuration complicating pediculosis, ringworm, favus, eczema, pruritus, acne, etc.
Herpes and dermatitis herpetiformis, erysipelas
Lupus erythematosus, von Recklinghausen's disease
Sclerodermia, Brocq's 'pseudopelade'
Diseases of the skull—rickets, syphilis, tumour.

2. **Referred Tenderness,** either due to disease elsewhere, or functional :—

Meningitis, increased intracranial pressure, intracranial tumour or abscess, concussion of the brain, otitis media
Neuralgia, major and minor, whether primary or due to disease of the eyes, ears, teeth, or viscera
Neurasthenia and hysteria.

Fig. 290 exhibits the cutaneous nerve-supply of the scalp and face, indicating the areas in which tenderness and pain are to be expected when disease or disorder of the various nerves is present.

If tenderness in the scalp is due to *bruising* or *wounds*, it should not be difficult of diagnosis when the history has been obtained. A similar tenderness is naturally to be expected when inflammation or suppuration occurs as a complication or later stage of any of the numerous skin diseases to which the scalp is liable, such as *pediculosis*, *ringworm*—a suppurating ringworm is known as *kerion*—*seborrhœic dermatitis*, *favus* ; the itching of *eczema* or *pruritus* may be so severe as to lead to scratching which breaks the skin, with the result that impetigo ensues. In young men and women *acne* may spread back to the scalp from the forehead, face, or neck ; *acne decalvans* is a mild staphylococcal infection of the hair-follicles that creeps slowly across the scalp, and leaves it bald by destroying the hair-follicles. *Furunculosis* of the scalp, and inflammation of a *sebaceous cyst*, need only be mentioned in this connection.

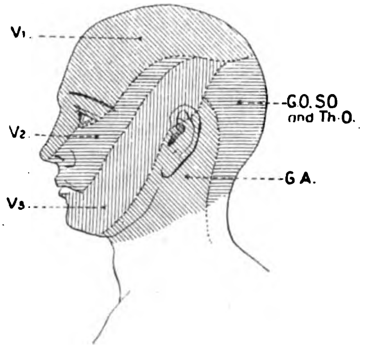

Fig. 290.—The cutaneous nerve-supply of the scalp. G A, Great auricular nerve ; G C, S O, and Th. O, Great, small, and third occipital nerves ; V^1, V^2, V^3, Ophthalmic, superior maxillary, and inferior maxillary divisions of the trigeminal (or fifth cranial) nerve.

In *herpes ophthalmicus*, or herpes zoster of the area supplied by the ophthalmic or first branch of the trigeminal or fifth cranial nerve, extreme tenderness over the affected area may be noted while the eruption lasts ; and after it has disappeared, tenderness and itching may be left behind for many months or years, sometimes with abnormal pigmentation. *Dermatitis herpetiformis* (p. 755) is a somewhat similar grouped vesicular or bullous eruption, with ringed and other erythematous lesions, but characterized by a much more extensive distribution than herpes zoster ; when it involves the scalp much tenderness may ensue, although the chief complaint will be of itching, and the course of the disorder is long and uncertain. *Erysipelas* is common in the scalp, and should be diagnosed readily. *Lupus erythematosus* of the scalp may cause tenderness while progressing actively, when it may resemble even a severe persistent erysipelas ; as a rule it is a very chronic, slowly progressive disorder, commoner in females than in males, starting between the ages of twenty-five and forty-five. It produces smooth and depressed areas of complete and permanent baldness, reddened by abundant injected venules. In *von Recklinghausen's disease*, subcutaneous neurofibromas are found all over the body, in association with freckling and pigmentation ; occurring on the scalp, these tumours will make it tender, whereas the tumours of fibroma molluscum (*Fig.* 291), a disorder at first sight resembling von Recklinghausen's disease, are not sensitive to pressure. *Sclerodermia* of the scalp may occasion much tenderness, particularly in its early stages ; it is a chronic diffuse infiltration of the skin that ends in atrophy, and by many is supposed to include the '*pseudopelade*' of Brocq, an atrophic indurative affection of the scalp giving rise to depressed areas of absolute and permanent baldness that adhere to the underlying skull, and connected by Brocq with alopecia areata.

Tenderness of the scalp is common in *rickets*, and is the main cause of the head-rolling and restlessness of the recumbent rickety child. It is due to hyperæmia and disordered growth of the cranial bones; and can often be diagnosed at sight by the thinness of the hair or positive baldness of the occipital region to which the head-rolling leads. A similar tenderness of the cranial bones is seen in *congenital syphilis*, and is caused by the rarefying (craniotabes) or hyperplastic (hot-cross-bun skull) osteitis present. In adults with neglected syphilis the skull may be tender from *secondary syphilitic periostitis* or *tertiary gumma*; besides the tenderness, pain is present, and is characteristically worse at night. *Tumour of the cranial bones* may give rise to tenderness of the overlying scalp or periosteum; in adults such tumours are usually secondary to malignant disease of the breast, thyroid gland, testis, or prostate. In children they are often secondary to sarcoma of the suprarenal gland, and may be the first clinical evidence that anything is amiss.

Tenderness in the scalp may be due to organic disease that is not in direct connection with it. In *meningitis*, whether syphilitic, tuberculous, or due to pus-producing microbes, local or general tenderness of the scalp may be a marked feature; and the same is true in cases with *increased intracranial pressure* due to any cause whatever. The chief complaint, however, will be of HEADACHE (p. 293). In *intracranial tumour* the scalp and periosteum are sometimes tender to pressure in the neighbourhood of the growth; the associated signs, such as vomiting on change of position, slow pulse, optic neuritis, and local paresis or paralysis, should aid the diagnosis. Tenderness of the scalp in the occipital region and below it has often been noted after *concussion of the brain*, whether mild in degree or severe, and apart from neurasthenia; the pain and tenderness may each be both superficial and deep.

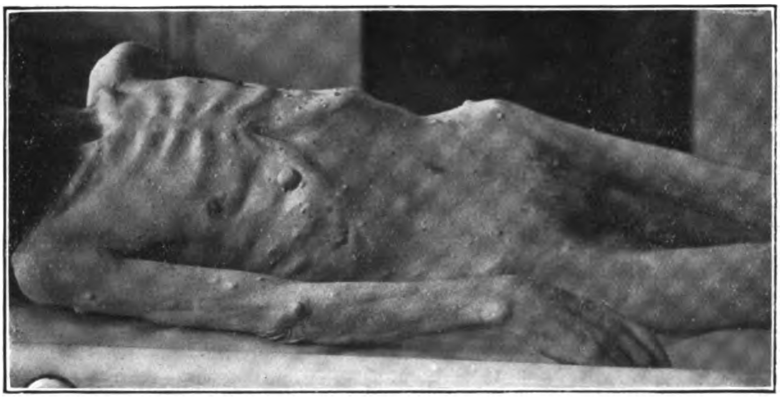

Fig. 291.—Molluscum fibrosum in a comparatively early stage.
(*From photograph lent by Dr. A. Rendle Short.*)

Tenderness of the scalp is often marked in *neuralgia*, a vague term applied to any severe pain that follows, or seems to follow, the distribution of a nerve. In *trigeminal neuralgia, neuralgia major*, or *tic douloureux*, the pain and tenderness often spread back to the vertex and parietal eminence, in correspondence with the cutaneous distribution of the first or ophthalmic branch of the fifth nerve. Pressure over the tender area will often bring on a paroxysm of pain; yet while the pain is raging, the patient often gains some relief by firm pressure over the painful part. When the paroxysm is recently past, pressure does not have any obvious effect in some cases. Identical neuralgic pain and tenderness may be met with in the rare cases where a tumour presses on the trigeminal nerve or its roots, as may happen in patients with meningeal new growths. Definite loss of sensation occurs if the nerve is involved in a tumour, whereas in tic douloureux there is no anæsthesia; in addition, the other signs of intracranial tumour should be looked for.

In another group come the cases of *neuralgia minor*, in which pain and tenderness in the scalp form a visceral reflex, and are due to disease in the eyes, teeth, ear, and thoracic or abdominal viscera. A referred visceral pain usually brings with it superficial tenderness, and both the pain and the tenderness are found over 'segmental' areas, or areas that do not correspond with the distribution of the peripheral nerves, but follow a central distribution (*Figs.* 292, 293). In other patients, however, the same lesions produce areas of pain, and less often of tenderness also, that do follow distributions corresponding with those of the peripheral nerves; and these are described as cases of *neuralgia minor proper*. To give examples of reflex neuralgia, disease of the *upper bicuspids* may cause pain and tenderness in the temporal region; disorders of the *eye*, particularly astigmatism and hypermetropia, iritis, and glaucoma, may cause headache and tenderness spreading from the forehead to the vertex and to the temporal area; suppuration in the *middle ear* may make the whole side of the head tender. Certain areas on the head are segmentally united with other areas on the body; the temporal area of the scalp is connected thus with the seventh dorsal segment, and so diseases of the *heart*, *lungs*, or *stomach* may all bring about temporal pain and tenderness, associated with the segmental area of cutaneous tenderness about the level of the epigastrium that directly represents the seventh pair of dorsal nerves. It is probable that a number of patients with undetected disease of the teeth, eyes, ears, or viscera, are treated for 'neuralgia' for long periods, when a more careful examination of their history and investigation of their physical condition would lead at once to the proper diagnosis. In a certain number of cases pain and tenderness in the scalp are due to *general diseases* such as diabetes mellitus, malaria, and rheumatism; a fact that leaves room for much latitude in diagnosis.

THE SEGMENTAL AREAS OF THE SCALP (*after Head.*)

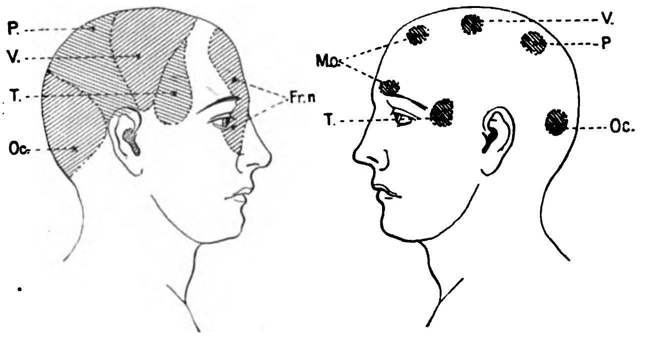

Fig. 292.—The segmental areas.

Fig. 293.—The maxima of the segmental areas shown in *Fig.* 292.

Fr.n, Fronto-nasal; **Mo**, Mid-orbital; **Oc**, Occipital; **P**, Parietal; **T**, Temporal; **V**, Vertical.

In both *neurasthenia* and *hysteria* complaints of pains and tenderness are common, and the scalp may be affected just as any other part of the body may. The neurasthenic often has occipital tenderness, with pain referred to the hair; brief mental effort may bring on pain and tenderness in the sinciput or vertex. The hysterical patient may be prostrated by headache, with extreme tenderness of the scalp. It is not necessary to say that the greatest care to exclude organic disease of every sort should be taken before the diagnosis of neurasthenia or hysteria is made in a patient complaining of tenderness in the scalp. It is noticeable that any conditions tending to build up the strength and improve the nutrition of neuralgic, neurasthenic, or hysterical patients, are likely to lessen the pains and areas of tenderness of which they so often complain. Conversely, these persons are always much worse when their health is low, and particularly when they are anæmic.

A. J. Jex-Blake.

TENDERNESS IN THE SPINE occurs in two different sets of conditions. In the first it is due to local disease of the skin or subcutaneous tissues, fasciæ, muscles, bones, or nerve-tissue in the immediate neighbourhood of the spine; and the pain felt when the tender spot is touched is a direct pain. In the second there is no local disease, and the pain felt on stimulation of the tender area is a referred pain, due in most cases to organic disease of one or other of the viscera, in a few to some obscure nervous disorder. The tenderness varies widely in degree. In the severest cases, whether direct or referred, the pain may be such that the patient cannot endure even the light pressure of the clothes ordinarily worn, and is in agony the moment a finger is laid upon the tender place.

When due to Local Disease, the tenderness is usually associated with rigidity of the spine in the tender section, a protective reflex designed to give rest to the diseased part. This is particularly well marked when it is bone—the vertebral column—that is diseased. A similar but less complete and more extensive rigidity will be noted when the local disorder is in the muscles or fasciæ of the back. Should the local disease or injury be so extensive as to involve or compress the spinal cord, special symptoms (girdle-pain, paresis, anæsthesia, etc.) will be added. The chief morbid states in which such tenderness of the spine occurs are summarized in the following table :—

Diseases of the skin and subcutaneous tissue	Injury, infected wounds, abscess-formation, etc.
Diseases of the muscles, fasciæ, or nerves	Gout, rheumatism, injury, herpes, etc.
Diseases affecting the vertebræ	Tuberculosis and other infections Caries sicca, spondylitis deformans, 'typhoid spine' Erosion by aortic aneurysm Invasion by malignant disease Injury
Traumatic neurasthenia, with local lesions that are not demonstrable	'Railway spine.'

To consider these lesions in detail : Obviously the skin and subcutaneous tissues may be tender over the spine after falls or blows on the back, infected wounds, in acne and furunculosis, in abscess-formation, whether the infection is derived from without, or from within as in pyæmia ; a *psoas abscess* may point and discharge on the back over the vertebral column. Tenderness in the spine due to *affections of the fasciæ* and *muscles* may be experienced by any ill-trained person who over-uses or strains his spinal muscles ; it is also common in *gouty* patients ; and frequently it is associated in the rheumatic with attacks of *lumbago*. Deep-seated inflammations in this region are not rare, and are seen usually in connection with *spinal caries* ; less often the inflammation may be due to *pyæmia*, *empyema* perforating spontaneously, *trichiniasis* and other very rare forms of *myositis*, when they chance to attack the spinal region. In a few instances, no doubt, *disease of the spinal nerves*, particularly when their posterior primary divisions are affected, gives rise to tenderness in the spine as well as along the course of the nerves themselves ; this may occur when pressure on the nerves or their roots exists, and in cases of *herpes zoster* or *neuritis*. Most of the causes of spinal tenderness enumerated above should not be difficult of diagnosis if a careful examination of the patient be made, and his other signs and symptoms of disease be noted.

The cases in which the tenderness is due to disease of the vertebræ are far more important than the above, and probably commoner also, as well as far more serious from the point of view both of prognosis and treatment. Excluding spinal trauma, which usually declares itself obviously and is considered below, the three disorders to which the vertebræ are liable in this connection are *tuberculosis*, invasion by *malignant disease*, and erosion by an *aneurysm*. In other rarer instances they may be affected with similar symptoms and results by *actinomycosis*, *pyæmic abscess*, the spread of infection from adjoining parts (retropharyngeal, mediastinal, subdiaphragmatic, perinephric, or pelvic abscesses), *hydatid disease*, *spondylitis deformans*, and *vertebral arthritis* due to the gonococcus and other microbes. When caused by *vertebral tuberculosis*, the spinal tenderness is local, and is generally accompanied by more or less angular deformity of the spinal column, collapse of the diseased and softened anterior part of the vertebral body, causing abnormal projection of its dorsal spine at the same time. If it is the posterior part of the affected vertebra that collapses, the spinous process will sink inwards ; it must be remembered, however, that congenital defect or deficiency of a spinous process is not very rare, and may be mistaken for the result of injury or disease. Whether deformity accompanies spinal caries or no, rigidity of the diseased part of the spinal column is sure to be present. It is maintained by involuntary contraction of the appropriate muscles, and becomes conspicuous when the patient is encouraged to bend his back in any direction, or to rotate the body on the pelvis. In addition, pain will be felt in the back when the patient's vertex, shoulders, sacrum, or legs are jarred ; his gait, too, and method of holding himself and turning, designed to relieve the diseased part of the spinal column from shock or strain, will be characteristic. In children who are not well looked after, this spinal tenderness and deformity may be unnoticed and the diagnosis of spinal caries not established until a psoas abscess has formed and has declared itself by pain in the leg, or lameness. The importance here of early diagnosis cannot be overstated ; spinal tuberculosis is commonest in children, but may occur at any age. It often happens that rickety children are suspected of 'spinal disease' by their parents; they present marked spinal curvature, due to flabbiness of the muscles, and, like all their bones, their spines may be tender on pressure. But there is no localized spinal tenderness in rickets, there is no angular deformity, the spinal curvature vanishes when the child is suspended by the head or arms, no pain is caused by jarring or rotating the spinal column, and there is no rigidity of the back; the other ordinary evidences of rickets will be present, so that the diagnosis should not be difficult. In adults, however, and particularly during the second half of life, it may often be difficult to determine whether a persistent tenderness over some part of the spine, associated with pain and rigidity, is due to *tuberculosis*, *aneurysm*, or *malignant disease* affecting the vertebral column. The occurrence of angular curvature, due to softening and collapse of the vertebral body, would argue in favour of tuberculosis, being comparatively rare in aneurysm or malignant disease; evidence of tuberculous mischief in the patient's joints, lungs, or larynx, a history of cough or blood-spitting, or a marked family history of tuberculosis, would all point in the same direction. Aortic aneurysm, eroding the vertebral column and causing pain and tenderness by pressing on the nerves in its vicinity, would be suggested if the patient were a middle-aged man giving a history of syphilis. Examination under the x-rays and testing for Wassermann's reaction might be of great assistance here; deep abdominal palpation, under an anæsthetic if necessary, might reveal the expansile pulsation of an aortic aneurysm. Secondary deposits of malignant disease, invading or encompassing a vertebra, may occasion marked spinal tenderness and pain in the back of the severest description; in rare cases, the malignant growth may be primary. The vertebræ are the bones most often invaded by secondary malignant growths; the primary growths most frequently responsible for secondary deposits in the bones are carcinoma of the thyroid, testis, prostate, and mamma, primary sarcoma of bone, and melanotic sarcoma. Here again the diagnosis may be very difficult, in the earlier stages of the disorder particularly, because the primary growth may be small and deep-seated, and may have given rise to no signs or symptoms leading to its discovery, so that the presence of secondary deposits is not suspected. In the later stages, the growth often burgeons into the spinal canal, and causes symptoms of paraplegia by compressing the spinal cord. When this occurs the diagnosis is easier, for the site of the compression may be indicated by a girdle-pain and a zone of hyperæsthesia; while anæsthesia, with paresis or paraplegia, is found below it, the sphincters are affected, the knee-jerks are increased, and ankle-clonus and Babinski's extensor plantar reflex can be elicited. But, as has been pointed out already, it may be impossible to find any definite physical signs in a patient complaining of very severe and intractable pain and tenderness in some part of his spinal column; and most physicians of experience must have met with sad cases where such patients have been treated as malingerers, the honesty of their complaints failing to win recognition until a pathological basis for them has been established at an autopsy.

Little more need be said about most of the other local diseases that may make the affected region of the spine both tender and painful. *Caries sicca* is the name given to an obscure rarefying osteitis of chronic course, non-suppurative, that may attack the vertebræ. The signs and symptoms of *vertebral actinomycosis* resemble those of tuberculosis. In *chronic pyæmia* a vertebral abscess may arise, and in patients with abscesses in the spinal region—such as *pelvic*, *perinephric*, *subdiaphragmatic*, *mediastinal*, or *retropharyngeal*—a spread of infection to the vertebræ may conceivably occur, giving rise to tenderness in the affected part of the spine; *hydatid disease* of the spinal canal or vertebral column may do the same in persons exposed to echinococcus infection. But in all these instances the tenderness in the spine will be but a minor symptom of a serious and more or less acute disorder, with other features that are more characteristic. Tenderness in the spine is often marked in *spondylitis deformans* (*Fig.* 274, p. 648), the name given to practically any chronic non-suppurative form of vertebral arthritis. It is no doubt an infectious process, and occurs after gonorrhœa, influenza, enteric fever (the 'typhoid spine'), tonsillitis, and other bacterial disorders. It is characterized by stiffness in some portion of the vertebral column, with irregular deposits of new bone in the adjoining ligaments, particularly the anterior common ligament, well seen by the use of x-rays. The chief sign is stiffness in the back, and in a few of the cases osteo-arthritis of some joints of the limbs occurs as well; in instances where the hip or shoulder are thus involved the disease has been named '*spondylose rhizo-*

mélique' by Marie. Men are affected four or five times as often as women, and the disease usually begins between the ages of twenty and fifty. Its diagnosis may be difficult, because the chief complaint may be of pain in the hips, legs, abdomen, or thorax, or of 'sciatica' or 'lumbago,' so that disease of the vertebral column may be neither suspected nor looked for. In most patients, the affected region of the spine is tender ; much spasm of the dorsal muscles is found in the more acute cases, while in those of long-standing, atrophy from disuse will be found. The *typhoid spine* is a rare sequela of enteric fever, usually occurring early in convalescence. The patient complains of tenderness and the most acute pain in the lower part of the vertebral column, after an initial stage of backache. Fever is present at first in half the cases, and no doubt the condition is commonly due to vertebral periostitis set up by the *Bacillus typhosus*. The symptoms last for many months as a rule, and deformity of the spine is left in half the patients ; but suppuration of the affected vertebræ seems to be unknown. Men are affected more often than women. In milder cases no physical signs of vertebral disease appear, and so the affection has been described as hysterical, the spine as an irritable spine ; in yet other instances, the spinal cord appears to be involved, as if the periostitis affected the spinal canal, loss of control over the sphincters being observed, with paresis of the legs, and changes—usually increase—in the reflexes.

Tenderness in the spine due to *injury* may be the expression of either organic or functional disease resulting therefrom, and the precise diagnosis may be extremely difficult. The trauma is usually a railway or other accident of locomotion ('railway spine'), a fall, a sudden shock or concussion ; in another group of cases it is either a single sudden muscular over-strain, due to over-exertion or the effort to avoid an accident, or the more chronic overstrain to which rowing men, football players, and the like are exposed. A gross injury may produce fracture of a vertebra, with or without displacement of the fragments such as can be demonstrated by the use of *x*-rays ; subperiosteal or subdural hæmorrhage, hæmorrhage into the spinal canal, hæmorrhage into or bruising of the cord, all of which will give rise to localizing cord-symptoms (girdle-pain at the level of the lesion, varying degrees of paresis and anæsthesia below it) when the lesion is marked. At the other end of the scale are found the sufferers from *traumatic neurasthenia*, who have been exposed to identical injury or over-strain, but present no definite signs of disease in the spine or cord, although quite incapacitated for months or years, by weakness and severe pains in the injured region. These patients often have increased knee-jerks and even ankle-clonus ; but definite evidences of organic disease are wanting, the sphincters are unaffected, Babinski's extensor plantar reflex is not obtained, muscular wasting is not found, unless from disuse, and the various pains and tendernesses of which complaint is made have a neurasthenic or even a hysterical distribution and character. Traumatic neurasthenia may follow surgical operations or comparatively slight injuries to the head, back, or testicle, in addition to the severer traumas and strains already mentioned ; and it must be noted that a delay of one or more weeks, an incubation-period, may intervene between the receipt of the injury and the development of the neurasthenic pains. It would be unfair to take such a delay as evidence of a hysterical factor in the case, or of malingering.

It is clear from the foregoing paragraph that traumatic neurasthenia includes cases in which it is not possible to say for certain whether a local organic lesion of the spine exists or not. Such instances form a natural transition to those in which there is :—

Tenderness in the Spine due to Functional Disorders, or to Disease in Other Parts of the Body.—In very few of these is there any deformity of the spinal column ; it is flexile and not rigid ; and pain is rarely produced when it is carefully bent, twisted, or jarred, so long as direct stimulation of the tender part is avoided. As a rule, the tenderness is superficial rather than deep, and it is often associated with other areas of tenderness in the side or front of the body. In *hysteria*, complaint of pain and tenderness in the spine and back is not rare—the 'hysterical spine.' The tenderness over the vertebræ is often accompanied by tenderness on either side of them ; in extent it may change from time to time, involving a single vertebra or even most of the vertebral column. In *neurasthenia* the spine may be tender from top to bottom, and more or less rigidity is often found also. When the tenderness is localized to a small part of the back, it may easily be taken as evidence of local organic disease ; but the presence of other neurasthenic symptoms—headache, irritability, fatigability after brief exertion—and the absence of signs of definite local disease or involvement of the cord, should help in the diagnosis. To distinguish clearly between neurasthenia and hysteria is often difficult, and particularly so in the milder cases of traumatic neurasthenia, because they may develop hysterical features such as areas of anæsthesia, a craving for sympathy, a tendency to exaggerate the symptoms, and so forth. The harmful effects of mental worry on neurasthenia, of the uncertainty attaching to an impending law-suit in which, perhaps, damages for injury are being claimed, are well known.

Tenderness in the spine is very commonly a reflex from disease in one or other of the thoracic, abdominal, or pelvic viscera. The tenderness is characteristically superficial in these cases, and acute pain may result from light pressure on the area involved ; and if the tender tissues can be pulled aside sufficiently, it will be found that pressure on the spine itself causes no pain whatever. The different viscera produce this tenderness with some regularity in different and definite spinal areas, a scheme of which is given in *Fig.* 294.

The organs and diseases most often giving rise to this referred tenderness and pain in the spine are as follows : The *aorta*, in aortitis, arteriosclerosis, and aneurysm ; the *heart*, in coronary sclerosis particularly, myocarditis, myocardial fibrosis, acute dilatation and failing compensation ; the *stomach*, in gastric ulcer, malignant disease, gastritis ; the *liver*, in cholelithiasis, cholangitis, new growth, and the venous congestion of tricuspid reflux ; the *intestine* and *rectum*, in acute inflammatory disorders, constipation, and carcinoma ; the *uterus*, in labour, menstruation, inflammatory affections, and new growth. It would appear that the lungs, whether inflamed or wounded, do not give rise to a referred tenderness ; on the other hand, the whole or any part of the thoracic spine may become tender in disorders of the *pleura*, such as pleurisy, pleural adhesions, or new growth. To illustrate the frequency with which pain and tenderness of the spine occur, the axiom of many hospital out-patient departments may be quoted, that there is no woman in London who has not got a pain at the bottom of her back—a libel on the sex, one may hope.

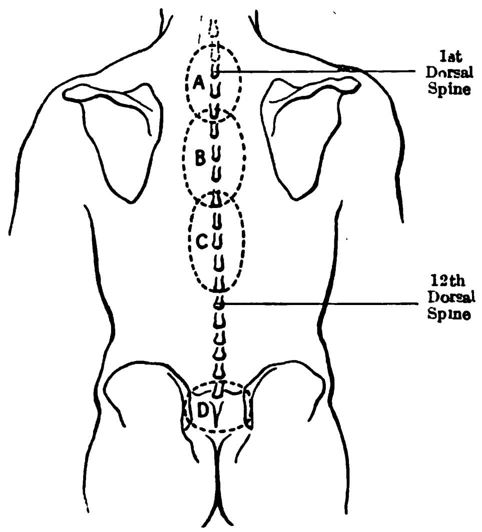

Fig. 294.—Areas of referred spinal pain and tenderness (*after Mackenzie*). A, In diseases of the heart ; B, In diseases of the stomach ; C, In diseases of the liver ; D, In diseases of the rectum and uterus.

The importance of distinguishing between the cases in which the physical signs of organic disease in the vertebral column or cord are absent, and those detailed in Class 1 above, need not be emphasized further. The referred pains and tendernesses disappear or are relieved with the cure or relief of the cardiac, gastric, or other disorder to which they are due. The diagnosis of the cause of tenderness over the fourth dorsal vertebra, for example, which may be due to disease of the heart, pleura, or stomach, must be made on general lines, and by consideration of the other signs and symptoms exhibited by the patient. *A. J. Jex-Blake.*

TENESMUS signifies frequent and painful inclination to go to stool, associated generally with straining and griping but with very little evacuant result. A precisely similar condition affecting the bladder is spoken of sometimes as *vesical tenesmus*, but a better known term for this is STRANGURY (p. 649).

The severest examples of rectal tenesmus are afforded by *acute dysentery*, in which, after the acute onset of the disease, copious loose fæcal motions are passed to start with, then smaller and smaller quantities at a time, and after this, when there is practically nothing left to come away from the bowel, the desire to defæcate urgently and repeatedly may still recur perhaps every ten minutes with painful straining, causing the patient to groan or cry out, but with practically no evacuant result beyond a little fluid with mucus and blood. The diagnosis in these cases is generally based upon the fact that the patient is or has been resident in some part of the tropics where dysentery is endemic ; the nature of the dysentery itself, whether due to the amœba of dysentery, or to Shiga's dysentery bacillus, or to other less well known bacilli, is made upon the results of bacteriological investigations of the stools.

Similar tenesmus may also occur in acute *cholera* when the stage of rice-water stools has been reached ; here again the diagnosis depends upon the fact of residence in a part where cholera is endemic, or in a district in which cholera has recently broken out in epidemic form ; it is confirmed by the discovery of the comma bacilli of cholera in the stools.

In this country there are various types of *acute infective diarrhœa* which may simulate cholera to such an extent that, although not cholera at all, they have been grouped together under the heading of *cholera nostras*, and in such cases tenesmus may be extreme. Temporary acute diarrhœa with much tenesmus may arise in school-boys and others from the eating of unripe apples or other fruit ; after a brief but acute illness, and perhaps a drastic purge, rapid recovery is the rule. More serious are the acute attacks of vomiting and diarrhœa which are familiar under the name of *ptomaine poisoning*, the cause being bacilli allied to Gaertner's bacillus ingested along with some article of diet. Cases of ptomaine poisoning may be sporadic, but occasionally as the result of many persons eating the same cold pork pie perhaps, or something of that kind at a public function, acute epidemics are recorded, some of the cases ending fatally. The bacteriology of the condition is complex ; different micro-organisms, including not only Gaertner's bacillus, but also Morgan's bacillus A, Morgan's bacillus B, and probably others, are at the root of different outbreaks. The diarrhœa is at first painless though frequent, but severe tenesmus ensues after the bowel has become empty of practically everything but a little fluid together with mucus and exuded blood.

Chronic dysentery is less often associated with tenesmus than is the acute form, but a considerable degree of tenesmus may none the less be complained of by those who have suffered from dysentery in the tropics and, having returned home not yet cured, still suffer from repeated diarrhœa to the extent of perhaps twelve or fifteen motions a day. The same applies to cases of *colitis*, whether muco-membranous or ulcerative, arising at home. It may be very difficult in some such cases to exclude malignant disease of the bowel unless the history is too long for this. In cases of doubt much may be learned by passing the sigmoidoscope and actually seeing the inflamed or ulcerated mucous membrane of the lower part of the bowel.

Intussusception will only cause tenesmus when the lower end of the intussusceptum has reached the pelvic colon or the anus. The symptoms will be those of intestinal obstruction, and when the intussusception is felt per rectum or seen protruding per anum, the only difficulty will be to distinguish it from a rectal polypus, or prolapse of the rectum. The condition is very much commoner in infants about nine months old than in any other class of patient, and at this tender age tenesmus will not as a rule be obvious. In older patients a subacute or chronic intussusception is fortunately very rare, and it is seldom diagnosed accurately previous to operation.

Acute summer diarrhœa and vomiting of infants is allied to ptomaine poisoning and is similarly due to one or more of the enteritic micro-organisms ; tenesmus may be very severe in infants as well as in adults.

Another malady allied both to ptomaine poisoning, to tropical dysentery, and to acute summer diarrhœa and vomiting of infants, is so-called *asylum dysentery*, of which the symptoms and results are very similar indeed to those of tropical dysentery ; asylum dysentery also has a bacterial cause, and the micro-organism producing it has been the subject of considerable investigation. The difficulty of deciding the precise nature of the infecting organism in all these cases depends upon the similarity between the different possible bacilli and the ordinary bacillus coli which always abounds in the evacuations.

Acute tenesmus may be a marked feature in some cases of *poisoning by arsenic* ; the diagnosis of this cause may be obvious either on account of the patient having taken an over-dose with intent to commit suicide, when choleraic diarrhœa and much tenesmus may come on subsequent to the initial vomiting and collapse ; or because, in less acute cases, the patient is known to be taking large doses of arsenic in his medicine—for instance, in the treatment of chorea or of pernicious anæmia ; indeed, the occurrence of diarrhœa with griping rectal pains and tenesmus is one of the difficulties that presents itself in continuing the arsenical treatment of pernicious anæmia and other blood diseases to the extent that one would like. On the other hand, even when arsenic is the cause of diarrhœa and tenesmus, it may sometimes be very difficult indeed to make certain of the fact, although some suspicion of it may have arisen in the mind of the physician. Accidental contamination of the water or of some food may have occurred ; or, still more important, some member of the household may be administering arsenic surreptitiously, either with a view to getting rid of the individual concerned, or occasionally even without any particular object,—for instance, in the case of some hysterical servant girls, who have been known to administer poisons in a household in this way apparently without any material object at all. The circumstances of the case may make one suspicious and lead to a careful watch being kept, or perhaps an analysis of the water-supply or of some suspected food will lead to the detection of the arsenic. In case of doubt one might have to resort to the expedient of taking the patient entirely away from the house in which she has been living, and from amongst the individuals with whom she has been associated, in order to see whether the symptom persists when she is secluded in a nursing-home or elsewhere, or whether it disappears there to return again when she goes home. Analysis of the hair for arsenic in cases of this kind will seldom be available as a test of the diagnosis, because it is only in the hair that is growing during the time arsenic is being administered that excess of arsenic is stored, and when sufficient doses to produce tenesmus have been administered they will generally have been large, and therefore have been given over only a short period.

Besides arsenic, other irritant drugs may produce tenesmus, especially perhaps *cantharides*, *calomel* in repeated doses, *colocynth*, *gamboge*, and indeed most of the powerful purgatives. The diagnosis depends upon a knowledge of the drugs that are being administered.

There remain for discussion a number of other conditions which may produce painful and frequent but fruitless straining at stool, the cause being either irritation of the rectum or obstruction to it or within it. These may be enumerated as follows :—

1. **Causes within the Lumen of the Rectum :**

Impacted fæces
A foreign body that has been inserted
Concretions
Worms.

2. **Things in the Wall of the Rectum :**

Carcinoma
Rectal prolapse
Polypus or polypi
Adenoma
Hæmorrhoids, especially if thrombosed
Fissure
Proctitis.

3. **Things Outside the Rectum :**

Enlarged prostate
Periprostatic abscess
Periproctal abscess
Ischio-rectal abscess
Vesical calculus
Ovarian cyst
Uterine fibroid
Retroverted gravid uterus
Pelvic hæmatocele
Ectopic gestation.

The diagnosis of all the above depends upon careful examination of the anal region, the rectum, and the vagina, by inspection, direct or through a speculum, proctoscope or sigmoidoscope, or with the finger.

Impacted fæces may, from their symptoms, simulate rectal carcinoma very closely ; but when there is carcinoma of the rectum, one's finger when inserted seldom comes upon a mass of fæces, whereas with fæcal impaction the mass is generally well within reach of the finger. The diagnosis will be confirmed by removing the mass itself piecemeal with a spoon, followed by enemata or other local measures, after which the patient recovers completely.

Rectal concretions differ from impacted fæces only in the material of which they are composed ; for instance, instead of being ordinary fæcal material, they may consist of hard lumps of *bismuth*, *magnesium*, *chalk*, or other drug that has been given by the mouth, or of the husks or products of some unusual meal, as an example of which one may mention the case of a boy who having stolen a bundle of cinnamon sticks, chewed them up and swallowed them, and a day or two afterwards suffered extremely from tenesmus as the result of a mass of undigested bits of cinnamon stick that had been impacted in his rectum. *Hair balls* have caused similar trouble, though they are even rarer in the rectum than they are in the stomach.

Adenomata, or long finger-shaped non-malignant polypi of the rectum, occur sometimes in such a way as to produce a kind of entanglement, in which fæces become impacted higher up than the finger can reach, and the patient will be thought to be suffering from carcinoma of the sigmoid colon or of the pelvic colon ; there will, however, as a rule be much less wasting than there would be with malignant disease, though both conditions produce their symptoms at a similar age. The diagnosis will depend upon examination with the procto-scope or the sigmoidoscope, and perhaps upon excision of such polypi as can be reached, followed by microscopical examination.

Another condition which may simulate rectal carcinoma very closely is *periproctal inflammation* followed by the formation of an abscess round the pelvic colon. The tenesmus which results, the constant painful straining, and the unsatisfactory evacuations, may be associated with obvious ill-health ; anæmia from loss of sleep ; and loss of weight ; so that malignant disease will frequently be thought the almost certain diagnosis. The condition is by no means so easy to detect as might be supposed ; sometimes it is not until the patient has been anæsthetized with a view to further examination with the proctoscope and sigmoidoscope that the nature of the condition is indicated by a sudden gush of pus escaping past the instrument as the result of the bursting of the abscess. The cause of such a condition is generally some previous local inflammation in the rectum, associated, for instance, with piles, or a polypus which has hitherto produced no symptoms at all ; in some cases there may be both carcinoma and abscess, the latter the result of the former.

A *vesical calculus* causing tenesmus will generally be one situated in a pocket of the bladder posteriorly ; it is a very rare cause of tenesmus, and generally there is or has been hæmaturia to indicate the need for examination of the bladder either with the sound or the cystoscope.

The remaining conditions in the above list need not be discussed in detail ; they will be diagnosed by rectal or vaginal examination, or a combination of the two.

There remains still one other very important cause of tenesmus, namely, *tabes dorsalis with rectal crises.* The symptom is not so uncommon as might be supposed. Not a few cases of tabes dorsalis complain that they are unable to go about their duties as early in the morning as they would like, because after going to the closet in the ordinary way after breakfast, they find that at intervals of perhaps half an hour they suffer from repeated urgent and painful re-calls, extending over perhaps two or three hours before the bowels settle down to comparative comfort for the rest of the day. Not much is passed after the first one or two visits to the closet, but the patient dares not be far away whilst the recurrent crises continue, for the call is urgent and very painful ; but as a rule, after two or three hours of suffering of this kind he finds that he is free until the next day. The condition is quite distinct from the incontinence of fæces which may result at a later stage of the malady ; it is an example of purely nervous tenesmus, which, however, may simulate carcinoma of the bowel very closely. It may last for months or years, and then cease to trouble the patient spontaneously, just as the lightning pains of tabes may. The patient's complaint is generally of diarrhœa, but upon careful inquiry it is found that the diarrhœa in the sense of fluid evacuations is much less pronounced than the tenesmus—that is to say, the recurrent painful call to stool without material evacuation. The diagnosis depends upon detecting the absence of knee-jerks and the existence of Argyll Robertson pupils.

Herbert French.

TESTES, ATROPHY OF.—(See ATROPHY, TESTICULAR, p. 66.)

TETANIC CONTRACTIONS.—(See CONTRACTIONS, p. 137.)

THERMO-ANÆSTHESIA.—(See SENSATION, SOME ABNORMALITIES OF, p. 604.)

THIRST, EXTREME.—Cases of extreme thirst may be subdivided into two main groups ; namely, those with and those without polyuria. To the former belong such conditions as diabetes mellitus, diabetes insipidus, hysteria, and so on, which are discussed under POLYURIA (p. 534). To the other group belong such conditions as are for the most part so obvious as to require no more than simple enumeration under main headings, as follows :—

1. Prolonged abstention from drinking : purposeful, or the result of necessity.
2. Fevers and febrile states.

3. Excessive loss of fluid: (*a*) From the skin by profuse perspirations, natural or pathological; (*b*) From the stomach, from repeated vomiting; (*c*) From the bowel, from excessive diarrhœa; (*d*) Into serous membranes, as in acute peritonitis.

4. After severe hæmorrhage: (*a*) External, e.g., post-partum; (*b*) Internal, e.g., from duodenal ulcer, ruptured tubal gestation, leaking aneurysm.

5. Gastrectasis due to pyloric stenosis, owing to the fact that the stomach absorbs little fluid as compared with the intestines.

6. Poisoning by such drugs as dry up the secretions of the mouth, notably belladonna and its allies, or astringents such as alum, gallic acid, tannic acid, or perchloride of iron.

7. The exhibition of excess of various salts, particularly sodium chloride, either as such, or incorporated in various food-stuffs.

It is clear that in some cases more than one factor at a time may be causing extreme thirst.

Herbert French.

THRILLS, PRECORDIAL.—In order to arrive at a diagnosis of the cause of any thrill which is felt over the præcordia, two facts must first be ascertained, namely (1) *The situation of the thrill*; and (2) *Its rhythm*. Having discovered a thrill over the mitral area, that is, in the region of the apex beat, and found that it is presystolic in rhythm, it is obvious that it is due to *mitral stenosis*. The valvular lesion will be confirmed by the presence of a presystolic bruit, as it is rare to find the thrill without a bruit being associated with it. On the other hand, if the thrill be systolic in time, and *mitral regurgitation* be present, the thrill is due to this valvular lesion. A systolic thrill at the cardiac apex may also be caused by *pericardial friction fremitus*, or *pleuritic fremitus*. A pericardial friction fremitus can be distinguished from an endocardial thrill by being more rubbing in character, usually occurring both during systole and diastole, while an endocardial thrill is a more purring vibration, and it is confined to either systole or diastole. The pericardial friction is confirmed by auscultation. *Pleuritic fremitus* in this region may be distinguished from an endocardial thrill and pericardial friction fremitus by asking the patient to hold his breath, when the fremitus will disappear unless it is due to pleuro-pericarditis. These distinguishing features between endocardial thrill, pericardial friction fremitus, and pleuritic fremitus, apply to any area in which thrills are detected. A pericardial friction fremitus may be present over the whole or any part of the præcordia, but the most common situation is to the left of the sternum near the base of the heart.

A systolic thrill in the second right intercostal space close to the sternum may be due to *aortic stenosis*, *thickening of the aortic valve*, *atheroma*, or *dilatation* or *aneurysm* of the ascending portion of the thoracic aorta, and the diagnosis of the cause of the thrill can only be made by the other physical signs which indicate the morbid condition present. Thus, if there be dullness in the second right intercostal space, over which the thrill is felt, there is dilatation or aneurysm of the arch of the aorta. There is no dullness in this situation when the thrill is due to aortic obstruction or to atheroma of the aorta. Not only may dullness on percussion accompany the thrill, but there may be pulsation, and even a pulsating tumour, in this region, showing that there is an aneurysm; an *x*-ray examination (*Fig.* 100, p. 209) helps in confirming the diagnosis.

A diastolic thrill may also be felt in the second right intercostal space close to the sternum, but it is rare; when present, it is due to *aortic regurgitation*, and is accompanied by the characteristic diastolic bruit and waterhammer pulse. Sometimes the thrill, like the bruit, is most marked in the third left space close to the sternum.

In the pulmonary area, viz., in the second left intercostal space near the sternum, systolic thrills are due to *congenital affections* of the heart, especially pulmonary stenosis and patent ductus arteriosus. An extensive thrill over the base of the heart in young children is nearly always due to congenital malformation. The apex beat is generally near its normal position. The cardiac dullness usually extends to the right of the sternum as the result of the enlargement of the right ventricle, and there is commonly a loud universal systolic bruit, having its point of maximum intensity over the base of the heart. The following signs of congenital malformation of the heart, other than patent ductus arteriosus, are also to be expected: cyanosis, either continuously present or occurring at intervals, dyspnœa, especially upon exertion, clubbing of the fingers and toes, and polycythæmia; with patent ductus arteriosus (p. 156), there may be no symptoms accompanying the abnormal physical signs.

A thrill occurs very occasionally in the second left intercostal space close to the sternum, in association with a *functional pulmonary bruit*. In such a case the functional origin of the thrill may be distinguished by the general condition of the patient, who will be suffering from anæmia or some debilitating condition ; and the signs of congenital heart disease, just mentioned, will be absent.

Presystolic and systolic thrills sometimes, but very rarely, occur to the right of the sternum in the *tricuspid area*, due to *stenosis* and *incompetence* of this valve.

J. E. H. Sawyer.

THROAT, SORE.—(See SORE THROAT, p. 613.)

THYROID GLAND ENLARGEMENT.—An enlarged thyroid gland gives rise to a swelling in the front of the neck, internal to the sternomastoid muscles and the carotid vessels, which, if the swelling is large enough, are pushed outwards. The gland is connected intimately with the larynx ; hence the most important sign of a thyroid tumour is that it rises and falls with the larynx and trachea during deglutition. In the great majority of cases the presence of this sign alone is sufficient to make a correct diagnosis. There are two sources of fallacy : (1) A swelling not thyroid in origin but lying in front of it, such as a sub-hyoid bursa or sebaceous cyst, or a suppurative or syphilitic perichondritis of the thyroid cartilage, may present the above sign ; (2) A thyroid swelling, if fixed, as it may be by inflammation or malignant growth, may not present it. In the vast majority of cases, however, a swelling in the position of the thyroid gland which moves on deglutition indicates an enlargement of that gland.

Varieties of Enlargement and their Differential Diagnosis.—During *menstruation* and *pregnancy* the thyroid becomes enlarged, but seldom sufficiently so to cause symptoms ; if the gland happens to be the seat of pre-existing disease the increase of swelling may be sufficient to induce respiratory difficulty.

Parenchymatous Goitre, or a general hypertrophy of the whole gland, is the commonest form of enlargement. All parts of the gland are affected more or less equally ; the tumour being bilateral, the normal shape is preserved. The swelling is freely movable, painless and soft. It is rarely congenital, and more often appears about puberty. Its rate of growth is usually very slow, and it may attain an enormous size without causing any other symptoms.

Cystic Goitre is a loose term used to cover any form of enlargement of the thyroid which is caused chiefly by the presence of one or more cysts. If the cyst is large and lax, fluctuation may be made out. The cysts, however, are often small and tense, and cannot be distinguished from solid adenomata. Cysts are rarely present without some enlargement of the rest of the gland.

Adenomatous Goitre.—The common cause of unilateral enlargement is the presence of an adenoma, a definite encapsuled tumour which may contain cysts and grow to a large size. A hæmorrhage into one of these cysts may cause a very rapid enlargement, and so give rise to a suspicion of malignancy ; cases have also been reported in which an intrathyroid hæmorrhage has been followed by acute dyspnœa and death. Adenomata may be single or multiple ; when present in both lobes, the enlargement may be difficult to distinguish from the parenchymatous form.

Fibrous or **Ligneous Goitre** is a rare condition, due to a primary chronic inflammation of the whole gland, and resulting in a firm dense enlargement.

Malignant Disease is seldom met with. It occurs with equal frequency in both sexes, and is rarely seen before the age of forty. In the early stages, while still confined within the capsule of the gland, it may be difficult to differentiate from the other forms of goitre. It should be recognized by its rapid growth, its hardness, and irregular bossy outline. When the neoplasm has penetrated the capsule and invaded surrounding structures, the diagnosis is made with more ease. The tumour may become fixed, no longer moving on deglutition ; often one or other vocal cord is paralyzed, a condition rarely seen with innocent goitre ; and involvement and ulceration of the trachea is common. The lymphatic glands may be enlarged, but as those first implicated are placed deeply, defying detection, not much help is gained from this source. There is a special liability for metastatic secondary deposits to occur in bones, particularly in the vertebræ and cranium.

Exophthalmic Goitre (Graves's disease, von Basedow's disease) is far more common in women than in men (*Fig.* 113, p. 236), and rarely occurs before puberty or after middle life. The most prominent features of the disease are: (1) Exophthalmos; (2) Tachycardia with palpitation; (3) Enlargement of the thyroid gland, often pulsatile; (4) Tremulousness of the hands and general nervous excitability; (5) Breathlessness on exertion. The vision is normal, but when the eyeball is moved downwards the upper lid does not follow as in health (von Graefe's sign). The palpebral aperture is wider than in health, owing to retraction of the upper and lower lids (Stellwag's or Dalrymple's sign). Pigmentation of the skin may be intense (*Fig.* 222, p. 527) and simulate Addison's disease, but the mucous membrane of the mouth is not affected as in the latter malady.

A well-marked case is quite characteristic, but there are others extremely hard to separate from simple parenchymatous enlargement, for with this, especially in young girls, anæmia is often associated, and with it the symptoms of tachycardia, palpitation, and breathlessness. It often becomes a matter of opinion whether a given case should be styled simple parenchymatous goitre or incipient Graves's disease.

The above are the commonest forms of enlargement. Others much rarer are: *Enlargement due to pyogenic infection*, either acute or chronic. In pyæmia it is not uncommon to find the thyroid the seat of multiple abscesses. *Tuberculous* and *gummatous disease* may also cause enlargement, and a slight degree of goitre has been noted in *typhoid fever*, *acute rheumatism*, *malaria*, *variola*, *cholera*, and *secondary syphilis*. *Hydatid cysts* of the thyroid gland have been noted on a few occasions. It having been ascertained that the swelling in the neck is definitely thyroid in origin and its nature defined, it remains to see whether there are any pressure signs on the surrounding structures.

Pressure on the Trachea.—Dyspnœa is by far the most important of all the symptoms that may be produced by enlargement of the thyroid gland. It may be the only thing complained of by a patient not even aware of the presence of a goitre. The size apparently is not so important as the shape and situation, for one reaching to the waist may cause no obstruction, and one the size of a cherry, if situated between the sternum and trachea, may give rise to intense dyspnœa. If the goitre is unilateral, the trachea is pushed over towards the opposite side and flattened; if bilateral, as in the parenchymatous form, it is compressed laterally. The dyspnœa may be constant and distressing, or only noticeable on exercise or on lying down. Most such patients like to lie high in bed, propped up on pillows. An idea as to the amount of pressure on the trachea may sometimes be gained by a question on this point.

Pressure on Nerves.—Unless malignant, a goitre rarely causes much pressure on nerves. Those that may be involved are: (*a*) The recurrent laryngeal, resulting in paralysis of a vocal cord; (*b*) The cervical sympathetic, shown by contraction of the pupil on the affected side and ptosis; (*c*) The vagus; (*d*) Rarely the nerves of the brachial and cervical plexuses. If any of these nerves are involved, suspicion must arise as to the malignancy of the tumour.

Pressure on the Œsophagus.—Being placed behind the trachea, the œsophagus generally escapes pressure by a goitre, though this is to be remembered as a rare cause of dysphagia.

Pressure on Veins is common, particularly on the internal, external, and anterior jugulars. The pressure is rarely more than sufficient to make them stand out prominently.

George E. Gask.

TINEA, VARIETIES OF.—(See FUNGOUS AFFECTIONS OF THE SKIN, p. 246.)

TINNITUS is a symptom which occurs in a large proportion of cases of disease of the ear, and occasionally when there is no obvious lesion of the auditory mechanism. The sounds complained of are usually subjective, but they may occasionally have an objective origin. Tinnitus may be continuous or intermittent. Its intensity and character vary greatly in different patients; to some it is an intolerable annoyance, and occasionally has even been the cause of suicide. The character of the sound may give some clue to the cause. Thus a pulsatile or rhythmical sound may be produced by the flow of blood through the internal carotid artery, which in its course through the carotid canal is separated from the tympanum only by a thin plate of bone, which may be deficient. A creaking noise may be produced by cerumen, or a foreign body, in the external auditory meatus. A bubbling noise may be due to the presence of catarrhal exudation in the middle ear.

A cracking or clicking sound may be caused by spasmodic contraction of the dilatator tubæ and salpingopharyngeus muscles which are attached to the Eustachian tube. When the character of the sound is described as humming, hissing, roaring, whistling, or musical, it is practically always subjective, and due to some irritation of the auditory nerve, rarely cerebral or in its course, but usually at its terminations in the labyrinth. In rare cases the tinnitus may be associated with an intracranial murmur which can be detected on examination of the head with the stethoscope. An audible intracranial murmur associated with tinnitus may be due to the following causes : (1) Possibly venous murmurs associated with increased intravenous pressure due to excessive pulsation of the brain ; (2) A *bruit de diable* in the jugular bulb which may occur in anæmia, plumbism, syphilis, and scrofula ; (3) Intracranial aneurysms. The sudden explosive sounds in the insane and others may be due to similar causes.

A distinction must be made between tinnitus and hallucinations of hearing, the latter usually taking the form of hearing voices, and indicating mental trouble, usually of a serious nature. Tinnitus, however caused, is usually influenced markedly by the general health and environment of the patient. Thus, sometimes the noises are less marked when the patient is in the open air, when his attention is occupied by other matters, or when the sense of hearing is occupied by listening to objective noises. Similarly, the trouble may be present only at night, but may appear in the day-time if the patient closes the external auditory meatus with his finger. Generally speaking, tinnitus becomes less marked and more bearable when the general health of the patient is good, and increases when the sufferer is out of health or overworked, either mentally or physically. Working in close, stuffy rooms, or in proximity to noisy machinery, over-indulgence in alcohol, and excessive smoking, have a bad effect ; in women the trouble may be increased during pregnancy, menstruation, or the menopause.

Though tinnitus is very common in diseases of the ear, yet serious lesions of the middle ear, internal ear, or auditory nerve, may be present without this symptom. There is no constant relation between tinnitus and deafness. The former may be present with perfect hearing, but when long continued the hearing nearly always becomes impaired. The sounds, too, may persist when the patient has become totally deaf.

Tinnitus may occur in the following diseases of the ear :—

1. The presence of *cerumen, aural polypi,* or *a foreign body* in the external auditory meatus. Removal of the offending body will in this case probably lead to the cessation of the tinnitus.

2. In any *inflammatory disease, acute or chronic, suppurative or non-suppurative, of the middle ear.* In catarrhal inflammation of the middle ear, the noise frequently has the character of bursting bubbles, and is due to movements of the viscid exudation in the ear itself. In otosclerosis, tinnitus is a very prominent and usually early symptom. It may occur before any alteration in hearing is present.

3. In diseases of the internal ear, tinnitus is especially liable to occur in a severe and intractable form. Thus, it is especially likely to be present in *Ménière's disease, syphilitic disease* of the internal ear, and in those lesions of the internal ear which may arise in the course of *typhoid* and other *specific fevers. Extension of suppuration to the labyrinth* from the middle ear is also an important cause ; and it may be present, usually associated with deafness, after a *fracture of the base of the skull.*

Tinnitus has been recorded as resulting from a *cerebral tumour* involving the roots of the auditory nerve, but this is a very unusual condition.

'Noises in the ears' may be complained of in a considerable number of general diseases, either with or without a lesion of the ear. Thus, they are frequently present in *anæmia,* and in diseases such as *leukæmia* or *pernicious anæmia,* in which anæmia is a prominent symptom.

Some *cardiac lesion,* especially aortic regurgitation, may be found in the pulsatile variety of tinnitus. *Gout, chronic nephritis, uræmia,* and *arteriosclerosis* with high blood-pressure, may also be responsible for tinnitus, and it may occur during attacks of *migraine.* Sometimes it has apparently a reflex origin, being associated with neuralgia or digestive disturbances, especially in gouty dyspepsia. *Malaria* may also be a cause, though here the trouble is likely to be the result of large doses of *quinine.* Other drugs likely to cause the trouble are *salicylates, antipyrin,* the excessive use of *tobacco,* and after *anæsthetics* such as chloroform or ether.

In persons who constantly use the telephone tinnitus may occur, associated with pain and some deafness—a condition known as 'telephone ear.' *Philip Turner.*

TONGUE, SWELLING OF.—(See Swelling of the Tongue, p. 698.)

TONGUE, ULCERATION OF.—(See Ulceration of the Tongue, p. 738.)

TREMOR occurs when the normally continuous contractions of a muscle at work. or the normally uniform tone of a muscle at rest, are replaced by a succession of separately perceptible muscular twitches. In these circumstances, a movement which is normally uniform becomes tremulous ; a position that can be maintained steadily under normal conditions is now kept unsteadily or shakily.

The normal muscular contraction is due to the discharge of a rhythmic series of nervous impulses from the motor neurons that govern the muscle. It is estimated that, in health, from five to fifty such nervous impulses leave the motor nerve-cells and reach the contracting muscle-fibres every second, the actual number per second varying in different motor neurons, and also in the same neuron according to its temporary condition of nutrition or fatigue. It is when the number of nervous impulses received per second by the contracting muscle-fibres becomes low, that steady and apparently uniform muscular contractions are likely to be replaced by tremors.

Tremors are of very various periods, amplitudes, and general characters in different cases. Their physiology and pathology are not at present fully understood, so that it is not yet possible to classify them etiologically. From a clinical point of view they may be classified roughly in accordance with their more obvious physical characteristics—their fineness, periodicity, regularity, and the circumstances that favour or inhibit their production ; but an unbroken series of graduated tremors can be traced in various diseases, passing by imperceptible degrees from the rapid and minute oscillations observed in paralysis agitans to the extremely coarse and irregular movements composing the intention-tremor of disseminated sclerosis. A similar variety of regular tremors may be observed sometimes in a normal person, as the temperature of his body falls from exposure to cold, or during the occurrence of a rigor. Hence a rigid clinical classification of tremors is impossible.

Classification.

Fine Tremor.

Exposure to cold
Nervousness, emotion
Muscular fatigue or weakness
Convalescence
Congenital and familial tremor

Senile tremor
Paralysis agitans
General paralysis of the insane
Graves's disease
Occupation neurosis
Chronic intoxications, e.g., by
Alcohol Cocaine
Tobacco Mercury
Absinthe Lead
Morphia

High pyrexia
Hysteria
Neurasthenia
Railway spine
Uræmia.

Unilateral Fine Tremor.

Cerebral tumour
Chronic hemiplegia
Hysteria
Chorea.

Coarse Tremor.

Exaggerated degrees of fine tremors
Familial and hereditary ataxias
Chronic hemiplegia.

Intention Tremor.

Disseminated sclerosis
Hysteria
Congenital cerebral diplegia
Some cerebral or cerebellar lesions.

FINE TREMOR.

Fine tremor consists of regular oscillations of small amplitude due to alternating contractions in antagonistic muscles or groups of muscles, repeated from three to nine times per second. It is usually most marked in the extremities, but may also—as in old age and in paralysis agitans—affect the head and neck. It may occur only when some movement is attempted ; or it may continue also when the patient is at rest. Fine tremors cease almost invariably during sleep ; emotion habitually increases them. They are not purposive, and should be distinguished from the fibrillar contractions (see CONTRACTIONS, p. 134) of individual muscle-fibres or muscle-bundles, seen in some muscular degenerations.

Fine tremors occurring in consequence of *cold, nervousness, excessive emotion, convalescence*, and *muscular fatigue* or *weakness*, are matters of common daily observation. They tend to pass off as the patient's condition of mind or body improves, and should occasion little trouble in diagnosis.

Congenital and *familial fine tremors* occur mostly in children or young adults, mainly in the hands and arms, face, or tongue. The oscillations are often absent while the patient is at rest, but make their appearance whenever movement is attempted, and are increased by nervousness or emotion. They can often be suppressed for a time by a strong effort of the will, and in many instances they disappear as the patient grows older. They cause practically no inconvenience, and are not accompanied by any other abnormalities in the neuro-muscular apparatus. Their diagnosis should be made plain by the patient's personal and family histories.

Senile tremor and *paralysis agitans* may be considered together. Senile tremor comes on with old age in the form of fine regular or irregular oscillations affecting the arms and the head. Both sides of the body are involved, the head early, and the tremor ceases during repose and in sleep. The muscles of the affected parts are neither rigid nor weak. These senile tremors must be carefully distinguished from the tremors of paralysis agitans, or Parkinson's disease, a progressive and more serious disorder ; in this the tremors are of several varieties. A fine oscillatory to-and-fro tremor, with from three to six excursions per second, affects the extremities and head in some cases. In others, the tremor is coarser, rhythmical, slower, and to some extent purposive ; in typical instances it produces the alternating movements in the thumb and index finger described as 'bread-crumbling' or 'cigarette-rolling.' These may be combined with more irregular movements of flexion and extension at the wrist, pronation and supination of the forearm. The progressive development of these tremors and movements is often characteristic ; beginning in one hand, the fine tremor may spread to the other after some months or years, and ultimately the lower limbs, the head and neck, the lips and tongue, and even the muscles of the trunk, may become involved. As a rule, the movements continue when the patient is sitting, or lying at rest ; in severe cases they may persist even during sleep. In most instances they can be lessened by an effort of the will, and diminish also on passive or voluntary movement. If, on the other hand, they increase on voluntary movement, a tolerable imitation of an intention tremor may result. They are augmented by emotion or excitement. In cases of some duration a well-marked coarse shaking of the arms may be a noticeable feature ; while contractions alternating in the flexor and extensor muscles of the legs may cause the feet to chatter when they are placed on the floor ; the thighs are commonly held in adduction.

Paralysis agitans is characterized by other signs that facilitate its diagnosis, and the chief of these are :—(*a*) *Muscular rigidity*, causing a fixed, expressionless facial aspect (see FACIES, p. 236), a monotonous voice, a bent and rigid carriage, and a shuffling, hesitating gait, with festination, propulsion, and retropulsion. Festination implies that the patient in walking from one point to another, starts with slowness and difficulty, but accelerates as he goes along, much as if he were running after his own centre of gravity, and at the end he may even fall forwards unless there is some object at hand for him to catch hold of. Propulsion, retropulsion, and the rarely observed lateropulsion, are terms meaning that the patient, sent off with a vigorous push to walk forwards, backwards, or sideways, exhibits the same acceleration and proclivity to fall down at the end—forwards, backwards, or sideways. (*b*) *Muscular weakness* of the tremulous or rigid parts. (*c*) *Paræsthesiæ*, such as aching pains about the limbs or body, flushes of heat or cold. The sphincters and mental faculties are not affected, while the deep reflexes are usually normal, but may be increased. Cases of Parkinson's disease occur in which tremor is absent ; the presence of the other signs mentioned above, however, should suffice for its recognition. *Bilateral cortical degeneration*, with its slowly increasing rigidity and muscular weakness, and its set facial expression, may resemble paralysis agitans ; but it is accompanied by progressive mental failure, increase of the deep reflexes, sphincter troubles, and the other evidences of cortical degeneration.

The tremor of certain types of *general paralysis of the insane* is a fine, irregular oscillation, often with a twitching character. It first appears in the hands and arms, and may spread till it is conspicuous in the tongue, lips, and face, when it is associated with the characteristic changes—slowness and blurring—in the speech. For the most part this tremor occurs on exertion ; it varies in extent, and may almost disappear during periods of general improvement. In the later stages of the disease a coarse universal tremor sets in, combined, perhaps, with grinding of the teeth. In these patients the moral and intellectual changes, tottering gait, alterations in the tendon- and pupil-reflexes, and other phenomena of general paralysis, will probably not escape attention.

In *Graves's disease* a fine, regular, and rapid tremor, about eight to the second, on exertion, is one of the cardinal signs. It affects the hands most, the arms much less, and least often the legs ; it is increased by movement or by excitement. In addition, attacks of trembling that affect the whole body may occur. It is only in aggravated cases, however, that these tremors are so severe as to interfere with the customary employments of the hands. The tremors of Graves's disease are likely to be of comparatively sudden onset, and the patient will generally exhibit many of the other prominent signs or symptoms of hyperthyroidism, such as thyroid enlargement, exophthalmos and the other ocular phenomena, tachycardia, attacks of palpitation, wasting, sweating, or mental changes, so that the diagnosis ought not to be difficult.

Fine tremors are observed frequently in many of the *chronic intoxications*, particularly those due to alcohol, absinthe, lead, mercury, nicotine, carbon disulphide, morphine, cocaine, and many other organic compounds that may be taken in excess in rare cases, whether intentionally or by accident. *Alcoholic tremor* is fine, regular, and rapid ; it is well shown in the outspread fingers of the extended hand and in the feet. In many cases it can be felt by the observer's hand more readily than it can be perceived by his eye ; or it may be rendered more conspicuous by laying a sheet of paper on the backs of the outspread pronated fingers and hand. It is absent during rest, and is increased by movement, excitement, or fatigue. It may also affect the tongue, lips, and facial muscles, taking the form of a rapid and rather irregular twitching, increased on exertion. This tremor is an early sign of alcoholism, and is often more marked in the morning hours, when it is due, perhaps, to fatigue and want of alcoholic stimulation ; it can be controlled to some extent by the will. Associated with it is a certain general nervousness and jumpiness ; in addition, the patient will no doubt exhibit some of the other signs of chronic alcoholism—venous stigmata or acne rosacea on the nose and face, restlessness, insomnia, gastric disturbances—particularly the morning vomiting of mucus on an empty stomach —paræsthesia and weakness of the extremities, mental and moral deterioration. If the main facts of the case can be made out, tremors due to alcoholism should not be hard to diagnose. A history of chronic alcoholism should always be inquired after most carefully, both from the patient, who may deny it *in toto*, and from the patient's friends, who may hasten to admit more than the facts warrant. This is of importance, because mere *nervousness* at the prospect or realization of an interview with a medical man, will often bring on a fine but temporary tremor, indistinguishable, for the time being, from the lasting fine tremor of the drinker. If such a tremulous, but teetotal, patient has indigestion and acne rosacea, and repudiates any veiled suggestion of alcoholism with apparently unnecessary warmth, there is some danger lest these evidences be taken as confirming the erroneous diagnosis of alcoholism.

The tremor of *mercurial poisoning*, a very rare complaint nowadays, is at first fine, but later coarse and even choreiform. It is met with amongst workers in furs, hatmakers, and others who use skins that have been cured with mercurial compounds. It begins in the face, hands, and arms, and may spread to all parts of the body. At first it is brought out only by excitement, or on attempted movement. Later, it may persist even during sleep, and speech may be interfered with from involvement of the muscles of the tongue, pharynx, and larynx. Other prominent symptoms of mercurialism that should not be absent are profuse salivation, stomatitis, anæmia, and cerebral symptoms of various kinds. Mercurial tremors may have to be diagnosed from those of paralysis agitans or disseminated sclerosis.

In *lead poisoning* a fine tremor of the affected limb is sometimes met with in cases marked by paralysis. The oscillations may also be seen in the tongue and lips, particularly in the rarer instances of chronic plumbism that exhibit cerebral symptoms and simulate general paralysis of the insane. The diagnosis of these unusual cases would be difficult unless a suspicion of lead-poisoning were aroused, either by a history of exposure to the intoxication, or by the occurrence of other signs and symptoms of plumbism (see p. 34)

In *hysteria* the clinical picture of any or every disorder of movement or sensation may be more or less closely reproduced; and tremors of every variety may be met with in hysterical patients. The diagnosis may be extremely difficult until hysteria is suspected, when it may be confirmed by the discovery of signs and symptoms that, singly or together, are pathognomonic (p. 465). The diagnosis of hysteria should never be made lightly; but only after a careful consideration of the history, signs, and symptoms, and when all evidences of organic disease have been looked for and found wanting. Unless a careful examination be made, the tremor of intracranial tumour, for example, or of disseminated sclerosis, may be wrongly diagnosed as hysterical.

Unilateral Fine Tremor is but rarely seen. It may be a *hysterical* manifestation, functional, and significant of no underlying lesion of the central nervous system. Unilateral tremor may occur in *tumour of the frontal region of the brain*; if present, it occurs in both arm and leg, and only on the same side of the body as the tumour. The patients will often exhibit mental changes, such as inattention, incoherence, loss of memory, alterations in character; sometimes, too, irritative phenomena occur.

Unilateral fine tremor may develop on either the same or the opposite side of the body in *tumour of the mid-brain and sub-thalamic region*. The general symptoms of cerebral tumour will be present, and in addition certain localizing signs may make their appearance. The chief of these would be paralysis of the third nerve, loss of sensibility over the area supplied by the fifth nerve, eccentric position of the pupil, defective reaction of the pupil to light, and weakness of the upward movements of the eyeballs.

It may be added that fine tremors occasionally occur in the paretic limbs after *hemiplegia*. The history of the case and the presence of other signs characteristic of hemiplegia should make the diagnosis here a comparatively straightforward matter. Fine tremor may be seen in *chorea*, and may be unilateral in such cases.

COARSE TREMOR.

Coarse tremors may develop as temporary exaggerations or later developments of the fine tremors occurring in several of the morbid states already considered. Thus, when the body is thoroughly chilled or fatigued, or when a patient is in a rigor, the initial fine tremor will often pass on into a very coarse tremor, as the amplitude of the involuntary muscular contractions increases, their rhythm remaining much the same. The fine tremor of paralysis agitans or general paralysis may similarly grow into a coarse tremor; coarse tremors are not infrequently seen in hysteria. The diagnosis in all these cases must be made on the lines already indicated.

Coarse tremor is met with sometimes in the various forms of familial and hereditary ataxia. Thus in ***Friedreich's disease*** (p. 512), in addition to the intention tremor considered below, irregular involuntary motions, described as coarse tremors in some cases, as choreiform in others, take place in the arms while the patient is at rest. Irregular nodding or tremulous movements of the head and trunk, also occur in advanced cases; the muscles of articulation and of the face may exhibit irregular purposeless contractions or quiverings when conversation is attempted. In *spino-cerebellar ataxia*, irregular choreiform movements, or constant tremors, large and small, may be seen in the head, trunk, and limbs whenever the attempt is made to hold them steady, but unsupported. Similar disturbances have been recorded in *cerebellar ataxia* and in the *olivo-ponto-cerebellar atrophy* of Dejerine and Thomas. In all these conditions the ataxia is the prominent symptom, the coarse tremor being no more than an occasional epiphenomenon; the diagnosis between them must be sought in special manuals, and also under the heading ATAXY (p. 55).

The coarse tremor of the affected limbs seen in patients with chronic or spastic *hemiplegia* or *diplegia*, and in some other cerebral disorders, is a variant of the athetoid or choreiform movements that are characteristic of those conditions. They are considered under the heading CONTRACTIONS (*Paramyoclonus multiplex*), p. 137. As has been mentioned already, it is practically impossible—were it, indeed, desirable—to draw any hard-and-fast line between the grosser fine tremors and the finer coarse tremors. In the same way, coarse tremors merge insensibly into the lesser degrees of athetotic and choreiform convulsions.

INTENTION TREMOR.

Intention tremor—known also as *action* or *volitional tremor*—has been defined as tremor produced, or if not produced at least exaggerated, by voluntary movements. These tremors affect the upper extremities, and sometimes the head and trunk also; the limb is quiet when not in actual use, but as soon as voluntary movement is attempted, irregular and involuntary to-and-fro motions begin in it, and are superadded to the intended movement. These to-and-fro motions become more marked, and sometimes more rapid the more nearly achievement of the desired movement is reached. The greater the amount of precision demanded by the voluntary action, the greater becomes the amplitude of these involuntary excursions. Wishing to drink, the patient may lift the cup from the table steadily enough; but as the cup approaches his lip, the involuntary movements appear and rapidly increase till its contents are jerked wildly in all directions as it reaches his mouth. The tremor may spread from the muscles that are being put into action, and cause extensive jerky movements of the head and trunk. The pathogeny of intention tremor is obscure. Very possibly it is due to destruction of the short intersegmental nerve-fibres in the substance of the spinal cord. The destruction of these fibres would render impossible the accurate mutual adjustment of the contractions of antagonistic muscle-groups that is essential for the smooth performance of even the simplest willed movements.

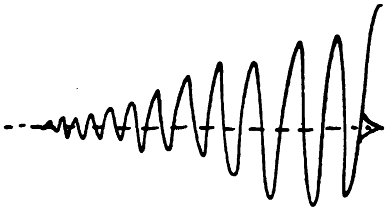
Fig. 295.—Movements in intention tremor.

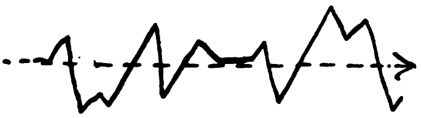
Fig. 296.—Movements in ataxy.
The dotted lines show the direction of the movement attempted.

Intention tremor is one of the most characteristic features of *disseminated sclerosis*. The arms are affected most often and most markedly, but careful observation will often show that none of the voluntary muscles escape. The head may oscillate when the patient is holding it up; the trunk may exhibit jerky movements when he sits or stands; the legs when he stands or walks, after the disease has made some progress. Disseminated sclerosis is a protean disorder. Typical examples, however, may be recognized by the occurrence of intention tremor (*Fig.* 295), muscular rigidity, nystagmus, pallor of the optic discs, and staccato or scanning speech. The deep reflexes are increased; the gait is spastic or ataxic; Babinski's sign is present; subjective sensory signs are far commoner than objective; and control over the sphincters may sometimes be impaired fairly early in the disease. In many instances, however, some, or even many, of these characteristics are absent; the diagnosis of disseminated sclerosis may then be far from easy, especially in its early stages. In *hysteria*, for example, intention tremor may occur in just the same way as a fine or a coarse tremor may; and other points of correspondence between hysteria and disseminated sclerosis may often be found in the age and sex of the patient, in the remittent course pursued by either of the disorders, in the frequent occurrence and partial recovery of various paralyses and of amblyopia with contraction of the visual fields, and in exaggeration of the deep reflexes. But distinct differences between the two are, fortunately, not wanting. In hysteria, the objective sensory signs are well marked, the optic discs are not affected, nystagmus is absent, Babinski's sign probably never occurs, and control over the sphincters is not lost. Attention to these points should suffice to clear up the diagnosis between hysteria and disseminated sclerosis; but in the earliest stages of the latter it may be necessary to keep the patient under observation for some little time before a definite opinion can be pronounced. Intention tremor has also been described as an exceptional feature in patients suffering from *neurasthenia*.

An intention tremor is not very rare in the *familial* and *hereditary ataxias*, especially Friedreich's disease and cerebellar ataxy; but the disturbance of movement in these disorders is characteristically an ATAXY (p. 65). It also occurs in some cases of *congenital* or *acquired cerebral diplegia* of backward or mentally defective children, occasionally appearing as a familial disease, and characterized by bilateral spastic paralysis affecting the limbs, or limbs and body. It is athetosis that is characteristic of these cases; but disordered movements of all sorts occur in them. In addition, the sphincters are commonly affected, the deep reflexes are increased, optic atrophy or inequality of the pupils are frequent; and if the patient is able to get about, a spastic or 'scissor' cross-legged gait is to be seen. Congenital cerebral diplegia in which the spastic weakness is most marked in the legs is described as *Little's disease*. The intention tremor occurring in birth palsy or in infantile hemiplegia has, unfortunately enough, been described under the name *chorea spastica*.

Intention tremor has also been recorded in a few instances of lesion of the *superior cerebellar peduncle*, *corpora quadrigemina*, or *optic thalamus*, particularly when the tegmentum, red nucleus, and rubro-spinal tract are involved. It may be noted in patients with *extra-cerebellar tumours* growing in connection with the eighth nerve, and occupying the posterior fossa of the skull between the pons and cerebellum. It is seen in a certain proportion of the cases of cerebellar atrophy, whether the degeneration is primary parenchymatous, progressive and due to interstitial and vascular lesions, or acute and following some acute specific fever. Intention tremor is also present in some patients with *olivo-ponto-cerebellar atrophy*. The diagnosis of these rare instances will naturally depend upon the development of other general and localizing signs of intracranial disease.

A, J. Jex-Blake.

TRISMUS, or lockjaw, signifies a maintained closure of the jaws by tonic muscular spasm, so that the mouth cannot be opened. It is seen best in tetanus. The term does not include mechanical inability to open the jaws owing to such affections as mumps, alveolar abscess with surrounding inflammatory œdema, angina ludovici, quinsy or severe tonsillitis, an odontoma, epithelioma of the mouth, myositis ossificans, and so forth; but there are at least two mechanical conditions that may not at first sight be obvious, but which may lock the jaws together and simulate true trismus—*impaction of a wisdom tooth*, and *arthritic changes in the temporo-maxillary joint*. These will be diagnosed as the result of a careful local examination of the teeth and of the joint respectively, and in the latter case there may be osteo-arthritic changes in other joints also.

Circumstantial evidence will generally serve to distinguish trismus due to *hysteria* or to *facial neuralgia*; and if there is any doubt at first, this will disappear if the patient can be watched for a while. If there are convulsive seizures in a hysterical patient with trismus, they can generally be distinguished from those due to tetanus or to strychnine poisoning by their polymorphous character, and by the fact that touching the patient, and other similar stimulation, does not bring them on so certainly as would be the case with strychnine or tetanus.

The rigidity of the face muscles in certain cases of *tuberculous* or *posterior basal* or *cerebrospinal meningitis* never occurs by itself, and is a minor symptom amongst others that point to the correct diagnosis. The same applies to *epilepsy* and to *uræmia*.

Malingering may sometimes take the form of lockjaw, and it may be a little while before the fraud can be detected; sleep is sure to come in time, and as the result of fatigue the malingerer's muscles relax completely.

Catalepsy may include trismus amongst its varieties of maintained muscular contractions; the general mental symptoms will assist the diagnosis. and as a rule there are no convulsive seizures.

Trichinosis is very rare nowadays, but if infected pork is eaten raw, or insufficiently cooked, the larvæ of the parasites find their way to many different muscles, and they show some predilection for those of the tongue, mouth, and jaws. The resultant irritation, pain, and stiffness cause trismus, whose nature may be difficult to determine unless the history points to pork as the origin. The patient is very ill in the earlier stages, with high fever, and the condition is often fatal. There may be an epidemic of the malady. The blood exhibits eosinophilia. The final criterion of the diagnosis is the discovery of the typical parasites coiled up in their little oval cysts amongst the affected muscle fibres. In a certain proportion of acute cases the embryos can be detected in films prepared from the centrifugalized deposit from 5 c.c. of the patient's blood after the red corpuscles have been laked.

Hydrophobia and **tetany** seldom exhibit trismus as a prominent symptom. The former, though it is almost unknown in Great Britain now, would suggest itself if any convulsive illness developed after a definite bite by a dog, wolf, or other similar animal, particularly if the spasmodic muscular difficulty was markedly increased by efforts at swallowing. The symptoms may not develop for weeks or months after the bite, so that the patient may fall ill when he has come home after being bitten abroad. *Tetany*, also rare, is at once distinguished by its typical carpo-pedal contractions (*Fig.* 1, p. 3).

Strychnine poisoning gives rise to generalized twitchings and convulsions long before trismus, the lateness of the development of the latter serving to distinguish it from tetanus. There may be evidence of strychnine having been taken or administered, either by the mouth or hypodermically ; the symptoms develop very acutely, and are apt to be rapidly fatal.

Tetanus is the cause par excellence of trismus ; the diagnosis is often obvious if the illness develops steadily in an otherwise healthy person or new-born infant, starting with stiffness of the neck muscles, spreading to those of the face and jaw, and thence to the rest of the trunk and limbs, with a tendency to extremely painful exacerbations on the slightest stimulation, even by a stroke with a feather or the banging of a door ; risus sardonicus ; opisthotonos ; no complete relaxation of the stiffening muscles unless chloroform is given ; a duration of days rather than hours, and a termination in death more often than in recovery ; especially if all these things follow a few days, or a week or more, after infection of the umbilical cord or a small penetrating wound with a rusty nail, or a piece of stick or other similar body that may have been contaminated with tetanus bacilli from the soil. It may be possible to demonstrate the drum-stick bacilli in films prepared from the deeper parts of the wound (see *Plate XXVIII*, *Fig. T*, p. 614). The chief difficulty arises when there is no clear history, or when the wound has been so small that it has healed or cannot be found. Even then, most cases are so typical that they can be diagnosed as tetanus without much difficulty. Unnecessary anxiety arises chiefly in cases of impacted wisdom teeth, or of hysteria, where tetanus may be suspected at first ; the subsequent course of the malady soon serves to exclude this.

Herbert French.

TUMOURS.—(See Swelling, pp. 655 to 702.)

TUMOURS OF THE SKIN.—The malignant tumours which affect the skin include carcinoma, epithelioma, Paget's disease, sarcoma, mycosis fungoides, and xerodermia pigmentosum.

As a rule, *cancer en cuirasse* and *nodular* (lenticular) *cancer* are secondary to cancer of the breast or other parts, and their diagnosis is self-evident. In *melanotic carcinoma* the tumours differ greatly in size, and also in colour, varying from a slate tint to bluish-black ; they appear more frequently on the genitalia and the extremities than elsewhere. The only condition from which melanotic cancer requires to be distinguished is *pigmented sarcoma*, and for this histological examination is necessary.

Paget's disease, occurring chiefly in women after the age of forty, begins as a reddening of a patch of skin, usually on or around the nipple, followed by branny desquamation. Infiltration soon produces a bright-red, granular, indurated surface, with a sticky, yellowish discharge, which by forming crusts may obscure the nature of the lesions, save at the border, which continues to be characteristic—sharply defined, indurated, and sometimes distinctly raised. After a period, which is usually about two years, but may be much longer, deep-seated parts may become affected, this extension of the disease showing itself on the breast by retraction and induration of the nipple and the formation of a tumour in the substance of the gland. In the early stage Paget's disease has to be distinguished from *chronic eczema*, which it resembles closely. Its differentiating features are the bright-red, granular surface exposed after removal of the crusts, the induration at the well-defined edge, the intractability, the age of the patient, and (later) the retraction of the nipple. The diagnosis may be made certain by microscopic examination of scrapings in iodized serum or liquor potassæ, when the bright, oval, nucleated bodies styled psorosperms will be seen, some still contained within the host-cells, others surrounded by distinct capsules.

Epithelioma begins usually as a single growth, superficial, deep-seated, or papillary, but all the forms alike are marked by peripheral extension, infiltration and destruction of neighbouring parts, central ulceration, and (except in rodent ulcer, for which see ULCERATION OF FACE, p. 735) a tendency to the formation of secondary growths in lymphatic glands, in viscera and elsewhere. Epitheliomata have a predilection for the natural orifices, for such moist parts as the glans penis, for exposed regions, and parts exposed to friction and trivial injuries. A wart, a mole, an ulcer, lupus vulgaris lesions, or an *x*-ray cicatrix, may be the starting-point. If the tumour begins in the skin, it appears first as a papule ; if in a gland, as a nodule. In the former, the more frequent case, the papule becomes firmer and extends laterally ; infiltration is evidenced by the hard, raised, pearly border. Ulceration occurs in the centre of the growth while extension is proceeding in the depths and at the sides. If the necrotic process involves the vascular tissue, there is more or less hæmorrhage. If the lateral extension predominates, the discoid type of epithelioma, as in sweep's cancer of the scrotum, is the result ; the surface is raised, with a steep border, and is bright-red, with a firm, granular surface. If the granulations are of large size, the growth is of the papillary type. The chief diagnostic features of epithelioma are : the origin as, usually, a single growth, the site, the starting-point, the slight discharge, the characteristic border, the secondary growths in glands and elsewhere. From a wart or a mole, epithelioma can be distinguished conclusively only by microscopical examination or by long-continued observation, and should signs of ulceration or crustation appear in such growths, epithelioma should be suspected. The so-called tubercular ulcerating syphilides are, as a rule, multiple, and not rounded, but rather segmental. (For the diagnosis of epithelioma from lupus vulgaris, see under NODULES, p. 402).

Sarcoma of the skin is most frequently secondary to growths commencing in the lymphatic glands or the deeper structures. Sarcomata vary considerably in colour, from reddish to brown or bluish-black, and also in consistence ; those of the spindle-celled type are fairly firm, the small-celled ones soft, with all intervening grades of density. They may appear in any part of the body, but are often found on moles, warts, and ulcers. The diagnosis usually depends upon histological examination, and it must suffice to say that a tumour which arises in previously healthy skin, or in a mole or wart, or at the site of an injury, which is soft and reddish from the vascularity that is a marked feature of this kind of tumour, or bluish from pigment, and which, after a period of slow growth, enlarges rapidly, projects above the surface, and readily ulcerates and bleeds, is probably a sarcoma.

In the early stage of *mycosis fungoides* the lesions are dull-red or livid patches, sometimes slightly tinged with yellow, varying in size from the area of a finger-nail to that of the palm of the hand, with border sometimes well marked, sometimes fading off, most frequently raised or thickened, but occasionally flat. At first the patches are smooth and dry, afterwards they become scaly, and later still they may be moist or covered with crusts. Presently the surface becomes infiltrated, and tumours as small as a pea or as large as an apple, firm and lobulated, broader at the free than at the attached end, and somewhat resembling tomatoes, project above the level of the skin ('fungoides'). As a rule, progressive thickening occurs, leading on to fungation. The lymphatic glands may be enlarged throughout the body. In the premycotic or eczematous stage—which is sometimes absent—the diagnosis may hesitate between mycosis fungoides and an eczematous or urticario-eczematous condition, and in some cases it may be impossible at this stage to distinguish definitely between the two ; but in mycosis fungoides the lesions will make little response to therapeutic measures, the red of the patches may be slightly tinged with yellow, and they are more persistent than those of eczema. The only malignant condition which mycosis fungoides in the mycotic stage at all resembles is sarcoma, but there is seldom any difficulty in distinguishing between the two.

The initial lesions of *xeroderma pigmentosum* (Kaposi's disease) are small spots resembling freckles, but rather darker, which appear chiefly on the face, neck, arms and legs, and generally begin within the first two years of life. Usually they disappear in winter and return in summer ; but after a time they become permanent, and often quite black. At first the condition suggests nothing but excessive freckling, but presently amid the 'freckles' appear white, glazed, atrophic spots, telangiectases, and superficial ulcers discharging pus which dries into yellow crusts. After some years, small, warty-looking growths develop on the 'freckles.' Tumours now form and ulcerate, producing fungous masses, and the process extends both widely and deeply, and destroys every tissue it encounters, not excepting bone. It is only in the earliest stage that there can be any difficulty in recognizing this very distinctive disease. In that stage it may be mistaken, as is suggested above, for simple lentigo, from which there may be nothing but the more extensive distribution to distinguish it. With the appearance of the later lesions lentigo will be dismissed from consideration, and it should be not less easy to rule out sclerodermia.

The *benign* tumours of which the diagnosis may be, in very different degrees, open to doubt, are sebaceous and dermoid cysts, fibroma molluscum, von Recklinghausen's disease, neurofibromata, myoma cutis, myxoma, the xanthomas, rhinoscleroma, molluscum contagiosum, colloid milium, benign adenoides cysticum, and keratosis follicularis.

Sebaceous cysts, seen most frequently on the scalp, the face, and the back, rounded, often somewhat flattened on the top, and sometimes as large as an orange, are distinguished from *fatty tumours* by the absence of lobulation and the fact that the sebaceous contents can be squeezed out when there is an opening ; in *Dercum's disease* (p. 410) the deposits of subcutaneous fat may be less well defined, but the diagnosis is generally obvious from the alcoholic history. *Dermoid cysts* may resemble *fibromata*, but if they are incised a sebaceous-looking material escapes. *Fibroma molluscum*, a pear-shaped or rounded fibrous tumour, usually covered by smooth skin and pedunculated, varying in size from a pin's head to an orange, and nearly always multiple (*Fig.* 291, p. 711), differs from a sebaceous cyst by its solid structure, and from a fatty tumour by its usual pedunculation and the absence of lobulation. *Von Recklinghausen's disease*, of which the lesions consist of nodular tumours, on and around which there is coffee-coloured pigmentation, is differentiated from ordinary fibroma in that the tumours are composed of fibrous and nervous, and not simply of fibrous, tissue. There is also a perceptible thickening of the nerves of the arms. *Neuro-fibromata*, which have their origin in the tendon-sheaths or the sheaths of the nerve fibres, and range in size from a pin's head to very large dimensions, are sometimes mistaken for rheumatic nodules, but instead of specially affecting the region of the elbows and the scalp, they occur on the trunk and extremities generally, nor is there (except from coincidence) a history of rheumatism.

Superficial *myoma cutis* occurs in the form of nodular tumours on the arms, back, chest, and cheek ; the deeper kind, originating in the subcutaneous muscular structures, occurs as a solitary tumour, commonly on the breasts and genitals. The former growths are soft and elastic, and, like the latter, are often painful. The distinctive clinical feature of myoma cutis generally is that it contracts under the influence of cold. This, with the pain, the absence of any tendency to ulceration, and the aspect and slow course of the growth or growths, should enable the affection to be identified.

Myxoma, when it arises in the skin—most frequently in the loose skin of the scrotum and labia—usually forms rounded, pedunculated, translucent tumours which tend to enlarge slowly. They have to be distinguished from *molluscum contagiosum*. This begins by the formation of small growths that have been likened to tiny mother-of-pearl shirt-buttons. They are usually flattened at the top, where as a rule there is a depression in which can be seen a small aperture leading into the interior of the tumour. Through this orifice a whitish material, or sometimes a milky fluid, can be squeezed out. When they are very small the tumours resemble the vesicles of *varicella*, but a microscopic examination of the contents will obviate the confusion. A small molluscum body on the genitals may resemble a hard chancre, but similar growths will be found elsewhere.

Xanthoma planum, often associated with jaundice and migraine, and characterized by the formation of yellow or yellowish-white plaques (rarely nodules), usually in the upper eyelid and sometimes affecting also the lower lid, is easy of recognition, the appearance of the yellow patches embedded in the corium, and almost imperceptible to the touch, being absolutely distinctive. *Xanthoma multiplex*, however, is not identified quite so easily. Here the lesions are nearly always nodular, and often observe a linear grouping, and the colour varies, a blackish or reddish pigment being mixed sometimes with the yellow. Usually the nodules occur in connection with hepatic disease. The condition has been confounded with *urticaria pigmentosa*, but there is no itching, there are no wheals, and it is impossible to produce factitious lesions. The tumours may be indistinguishable from multiple dermoids of the skin until microscopic examination is made. *Xanthoma diabeticorum* differs from other forms of xanthoma, *inter alia*, in the presence of a raised red area around the yellow spots. This feature has led, in the early stages of the affection, to confusion with *acne*, but if the lesions are punctured they will prove to be solid. In the same stage the lesions may simulate those of *lichen planus*, but the resemblance soon disappears.

Rhinoscleroma begins, usually before the age of forty, in and around the nostrils as nodules in the cutis, and in the deeper layers of the mucous membrane. These coalesce to form a hard, smooth, glistening growth which spreads inwards from the lip and downwards to the pharynx from the posterior nares. The growth does not break down spontaneously, but is generally slowly progressive. It is not likely to be mistaken for anything but *epithelioma*, which is prone to ulcerate, generally has infiltrated edges, seldom attacks the upper lip, and usually begins later in life. In *rhinophyma*, pustules are often present, the growth is soft, and there is vascular dilatation.

In *colloid milium*, small, yellow, cyst-like formations containing a gelatinous substance appear in the skin, chiefly on the upper part of the face. They may become depressed in the centre and be absorbed slowly, or may inflame and dry up. The only condition from which colloid milium needs to be distinguished is *epithelioma adenoides cysticum*, in which the tumours, shining and translucent, contain one or more white, brightly refracting, milium-like bodies. The face is the part attacked most frequently, but the growths may appear on almost any part of the body. They are not yellow, like the growths in colloid milium, nor are they soft.

Keratosis follicularis begins as small brown or yellow crusts, hard and dry, which, when detached from the underlying tissue, are found to present on their under surface a softish prolongation which dips into a follicle. At first discrete, the lesions may become confluent, and there is thickening of the affected parts until nodular masses are formed, from which oozes an offensive discharge. The affection is slowly progressive. At the outset the condition may be mistaken for *keratosis pilaris*, but it is not confined to the situations affected by that disease. The prolongation into a follicle gives it some resemblance to molluscum contagiosum, but it has a less limited distribution, nor have the growths the pearly appearance of the molluscum bodies, while the aperture in the individual lesions is larger. *Malcolm Morris.*

TYMPANITES.—(See METEORISM, p. 388).

ULCERATION OF THE CORNEA.—The course of all corneal ulcers conforms to a general type, though the clinical varieties may vary. The process begins with an infiltration in the substance of the cornea, either central or peripheral; the result of this infiltration is a local loss of transparency, though in early stages the surface of the cornea may still retain its polish. The infiltration proceeds to suppuration, which is followed by a loss of substance, the corneal surface being dull and irregular, and, in the centre of the ulcer, depressed below the surrounding level. The base of the ulcer is grey or yellowish, and the surrounding portion of the cornea may be opaque with more or less grey infiltration.

The suppuration is followed, in cases which have a favourable termination, by vascularization, superficial vessels from the surrounding conjunctiva encroaching on the cornea and invading the suppurating area. The vascularization is followed by cicatrization, the surface of the cornea again becoming polished but flattened and opaque. The opacities resulting from corneal ulcers are localized, well defined and opaque, in contrast to the diffuse indefinite haze which follows such non-suppurative forms of inflammation as interstitial keratitis. Corneal ulcers may not heal, but occasionally lead to perforation of the cornea, prolapse and adhesion of the iris, anterior polar cataract, or panophthalmitis. Iritis, iridocyclitis, and pus in the anterior chamber (hypopyon) may also be associated conditions. The usual subjective symptoms are pain, photophobia, and lachrymation. The presence of corneal ulcers is demonstrated most satisfactorily by the instillation of a drop or two of fluorescein, which stains necrotic corneal epithelium or exposed corneal substance, green. The brightly-stained ulcer shows up in marked contrast to the surrounding clear cornea.

Corneal ulcers may occur in the following clinical varieties:—

Catarrhal, or simple infective ulcers. These usually occur as minute grey infiltrated spots in the centre or periphery of the cornea. They heal very rapidly as a rule. They may follow injury to the corneal epithelium by foreign bodies, or may be associated with acute conjunctivitis or rhinitis.

Phlyctenular ulcers are associated with phlyctenular conjunctivitis, the ulcer forming after the epithelium on the top of a phlyctenule has been rubbed off. They are usually marginal, but may occasionally make their way on to the cornea, a leash of conjunctival blood-vessels trailing after them. Similar ulcers may be associated with *acne rosacea.*

Hypopyon ulcer, or ulcus serpens. This is a shallow ulcer affecting chiefly the superficial layers of the cornea in or about its centre. The middle layers of the cornea are comparatively unaffected, but at the posterior surface the infiltration again becomes dense, with much fibrin and débris, associated with the formation of more or less pus in the anterior chamber. The ulcers often perforate ; they are usually due to infection with the pneumococcus (*Plate XII*, p. 232). The pus in the anterior chamber is always sterile, unless there is perforation of Descemet's membrane. These ulcers do not react to ordinary methods of treatment as a rule, but require cauterization, either by pure carbolic acid or the galvano-cautery. The hypopyon then disappears rapidly.

Mooren's ulcer, or rodent ulcer of the cornea, is a chronic serpiginous ulcer, usually affecting the eyes of elderly people. It begins at the margin of the cornea and spreads slowly over the whole surface, the advancing edge being much undermined. The ulcer is always shallow, and perforation never occurs ; the ulcer may heal in places, but this is seldom permanent, and the ulceration usually spreads over the whole surface of the cornea whatever method of treatment may be employed to arrest its progress, though recently radium has been used with some success. No specific organism has yet been discovered.

A *dendritic ulcer* is characterized by its peculiar shape—a long central stem with small linear ramifications. It is not really an ulcer, but an infiltration under the corneal epithelium, which in the later stages may become necrotic and break down. It is best treated by rubbing off the affected corneal epithelium with a pointed stick dipped in absolute alcohol.

Corneal ulcers may occur in association with pannus in *trachoma*, their usual site being at the margin of the vascular area. Occasionally they penetrate more deeply into the corneal substance.

Corneal ulcers frequently follow *gonorrhœal* and *diphtheritic conjunctivitis.* They spread rapidly, and often lead to perforation of the cornea and panophthalmitis. The diagnosis depends on bacteriological methods and the discovery of the causative micro-organisms.

Keratomalacia, a disease of childhood, is associated with night blindness and xerosis or dryness of the conjunctiva. Characteristic foamy white patches are seen on both sides of the cornea (*Plate XII*, p. 232). The cornea becomes dull, grey, and cloudy, and ultimately disintegrates from purulent infiltration, associated with very slight signs of ocular inflammation. The ocular condition is associated with marasmus and malnutrition. The prognosis, both as to eye and vision, is bad.

Keratitis e lagophthalmo is associated with paralysis of the seventh nerve. Owing to the failure of the orbicularis palpebrarum the eye cannot be closed, and does not remain closed during sleep. The lower part of the cornea is exposed, becomes dry, and the corneal epithelium dies, with consequent ulceration of the cornea. The condition can be cured by diminishing the palpebral aperture by sewing the eyelids partially or completely together.

Similar exposure of the cornea and consequent ulceration is seen occasionally in cases of EXOPHTHALMOS (p. 229), for instance in severe *Graves's disease.*

Keratitis neuroparalytica. In paralysis of the fifth nerve, or as a result of excision of the Gasserian ganglion, the cornea becomes dull and cloudy and necroses in the centre, only the periphery remaining clear. A hypopyon forms, and in some cases the whole eye is destroyed, though occasionally the keratitis may lead only to a permanent opacity. The condition is due to arrest of lachrymal secretion and absence of corneal sensation, which is followed by abolition of the winking reflex. Foreign bodies lodge on the cornea and are not removed. The prognosis is bad, and is little affected by treatment.

Corneal ulcers may be associated with *herpes frontalis*, vesicles forming on the cornea simultaneously with the vesicles on the skin, especially along the course of the supra-orbital nerve. The ulceration is often severe and may lead to perforation and destruction of the eye, and is in any case followed by considerable corneal opacity. The cornea is usually insensitive, and the intra-ocular tension may be raised.

Tuberculous ulceration is not common fortunately, but it should be borne in mind as a possibility in chronic or resistant cases. The diagnosis depends on the history, the presence of tuberculous glands or other similar lesions, positive reaction to the various tuberculin tests, and, most conclusively of all, upon the detection of tubercle bacilli in the discharge from the ulcer itself. *Herbert L. Eason.*

ULCERATION OF THE FACE.—The ulcers most often met with on the face are lupous, scrofulous, syphilitic, or malignant. In *lupus vulgaris*, the ulceration is extremely chronic. The lesion begins as a papule, develops into a nodule, and after a while, in the majority of cases, the lupous tissue breaks down and forms a granular sore covered with greenish-black crusts; but around the ragged edge will still be seen the characteristic 'apple-jelly' nodules in different stages of development. The ulceration may extend through the whole thickness of the skin and may become the seat of warty vegetations. In the nose, where the integument is thin, it may cause necrosis of cartilage. The course the pathological process runs, from the papule onwards, as here described, and the frequent presence of the different lesions simultaneously, shed sufficient light on the character of the ulceration. I need only say further that the ulcer of lupus, however deeply it may extend, never erodes bone. This alone is sufficient to differentiate lupus from the ulcers of syphilis and cancer. It nearly always begins before the age of twenty.

In the ulcers of *scrofula*, though they have no absolutely distinctive characters, it will often be noticed that the edge is undermined and the surrounding skin blue and of low vitality. Their occurrence in children of strumous aspect, or in elderly persons who bear the stigmata of scrofulous lesions dating from childhood, and their tendency to become chronic owing to the feeble resistance offered by the tissues to morbid processes, leave no room for doubt as to their true nature.

It is in the late secondary and the tertiary stages of *syphilis* that cutaneous lesions on the face, as elsewhere, are prone to ulceration, instead of to the resolution to which typical secondary syphilides tend. The whole structure of the skin, or mucous membrane, is frequently involved, the ulceration is deep, and the ulcers, while healing in the centre, are prone to extend at the margins, and so assume the characteristic circinate or serpiginous form. The appearance of the ulcers, with the history, and the marks of earlier syphilitic lesions, will supply all the guidance the diagnostician needs; Wassermann's serum test and the effects of mercury and iodide of potassium or salvarsan may serve to clinch the diagnosis.

As a rule *rodent ulcer* occurs in persons of more than middle age, and its favourite points of attack are the outer edge of the orbit and the side of the nose. It begins as a small, circumscribed nodule, dull brownish-red in colour, flat, depressed in the centre, and firm to the touch. After, it may be, years, the cuticle covering it is broken, and an ulcer is formed with depressed granular centre and infiltrated border. Very slowly this extends, both in circumference and in depth, infiltrating and destroying the subjacent tissues, including bone. Usually the destruction of the underlying parts is more marked in the centre, so that the ulcer becomes crateriform. Its invariable features—the inconsiderable suffering it inflicts, the singular slowness of its progress, its depressed centre, and the firm, raised, rolled edge, its failure to affect neighbouring glands, and its incurability except by extirpation or by physiotherapy—are so characteristic as to leave little scope for diagnosis. Epitheliomatous in structure, it differs from epithelioma in that the latter growth has a very hard and everted edge, and a foul base roughened with granulation, is often attended by severe pain, is much more rapid in its course, and infects the glands in its vicinity. It differs from lupus vulgaris in its mode of onset, in the absence of the 'apple-jelly' nodules, and in not being a disease that starts in childhood. It may be diagnosed from tertiary syphilitic ulcers by the characters described above, and also by its usually solitary character and its resistance to treatment. *Malcolm Morris.*

ULCERATION OF THE FOOT.—The ulcer which attacks the foot specially, though not exclusively, for the hand may be affected in the same way, is that known as perforating ulcer (*Fig.* 297). The exciting cause is pressure upon or injury to a foot in which there is interference with the nerve supply, either from peripheral lesion, as in peripheral neuritis, or from damage to the nerve-trunk, as in leprosy, syphilis, or diabetes mellitus, or to the nerve-centre, as in tabes dorsalis and general paralysis. The commonest situation of the ulcer is at the point of greatest pressure—the under aspect of the metatarso-phalangeal joint of the big or little toe. The ulcer, which is more a sinus than a true ulcer, usually painless, may be simple or multiple, and both feet may be affected. It often begins by suppuration under a corn. When the horny covering is cast off, a track is seen which extends downwards until the bone is exposed. The process is usually very slow, and if the pressure from walking is continued the thickened epidermis forms a kind of corn-shield around the opening. The more essential symptoms of the disorder of which perforating ulcer is but an incidental manifestation will disclose the true nature of the lesion. The only malady with which it can be confused is a suppurating corn. From this it is distinguished by the absence or small degree of pain, and by its irresponsiveness to the simple surgical treatment to which a suppurating corn yields readily.

Ulceration of the foot is also apt to arise as the result of rubbing, irritation, or other injury to parts whose nutrition is impaired, for example in cases of talipes from nerve disorders, or when sensation is impaired, as in cases of syringomyelia (p. 608), or paraplegia (p. 510).

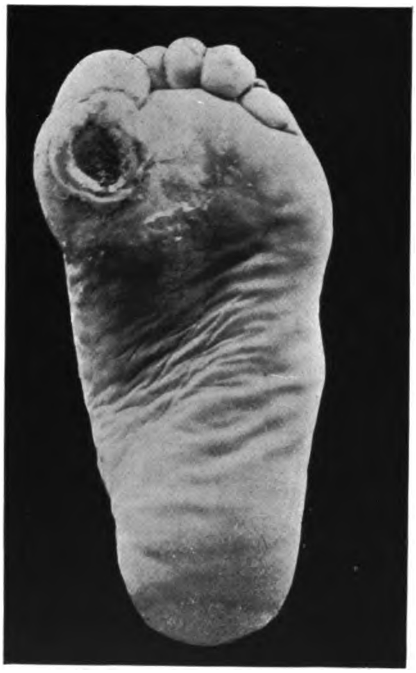

Fig. 297.—Perforating ulcer of foot; from a case of tabes dorsalis.

Mycetoma is a fungous disease that is known alternatively as Madura foot, because, endemic in Madura and other parts of India, it usually affects the foot or the leg, though sometimes the hand, and in rare cases the shoulders or the scrotum. The affection appears in several forms, according as they are due to different species of discomyces and aspergillus. The lesions may be black ('melanoid') or pink ('ochroid'). The disease begins with slight swelling and redness or local induration, and as it progresses the foot swells and the surface becomes dotted with small nodules, each containing the opening of a sinus which discharges a viscid, syrupy, slightly purulent, sometimes blood-streaked fluid, in which are suspended rounded greyish, yellowish, or black granules. As the foot enlarges, the leg, from disuse, atrophies. The only condition from which mycetoma needs to be discriminated is *actinomycosis*. This affection usually begins in the bone or other deep structures of the jaw, face, or neck, may thence spread to the surface, and may involve the viscera. In the discharge the ray fungus may be found in the form of tiny, friable, yellowish or greyish bodies, though microscopical methods and the discovery of the characteristic ray fungi (*Plate XXVIII, Fig. S*, p. 614) will generally be required before the nature of the case can be confirmed.

Malcolm Morris.

ULCERATION OF THE LEG may be classified under three headings:—(1) *Non-infective Ulcers.*—These include those that are not due to any specific infection, but which are caused by various factors which interfere with the vitality of the part by injury, lack of circulation, or deficient innervation of the tissue. (2) *Infective Ulcers* resulting from the direct action of a definite specific infection, e.g., tuberculosis or syphilis. (3) *Ulcerating Tumours.*—These are malignant tumours, which have originated in or invaded the skin.

Non-infective Ulcers.—*Varieties and Causes.*

Varicose Ulcer.—The presence of varicosity in the veins of the leg diminishes the free return of blood, and so leads to congestion and interference with nutrition, and thus to ulceration. In the majority of cases the ulcer is situated on the inner side of the leg about three inches above the ankle. It may be small, or may encircle the limb. For some distance round the ulcer the skin suffers from the effects of passive congestion; it becomes indurated and of a purplish-brown colour, and numerous small varicose veins may be seen in it. Any slight injury may cause abrasion of this weakened skin and thus another ulcer.

The presence of varicose veins associated with an ulcer will usually lead to the conclusion that the latter is dependent on the former, and that view will probably be correct, but it may not be the whole truth, for syphilitic and varicose ulcerations may be present at the same time. Before the introduction of Wassermann's test for syphilis it was common practice to give antisyphilitic remedies in order to clear up the diagnosis, and this may still be done when the serum test cannot be carried out.

Lymphatic Obstruction also leads to loss of nutrition, and ulceration may result. The best instance is seen in elephantiasis due to *Filaria sanguinis hominis*. In this country elephantiasis is rare. Other instances that may be cited are swellings of the leg following a badly united fracture ; the cicatricial contractions of extensive burns ; phlegmasia alba dolens, or white leg, during pregnancy or after labour.

Atheroma of the Arteries leads to a feeble or imperfect circulation of the blood, and so to loss of nutrition. Ulcerative conditions of the lower part of the leg are therefore common in such cases, and even gangrene may result.

Old Age.—Owing to a weaker condition of the tissues, ulcers are much more frequent in old people than in the young.

Cold.—A similar condition is brought about by exposure to cold, especially in persons whose nutrition is imperfect, whether from bad or insufficient food. The first effect of cold is to produce a chilblain ; this if rubbed or irritated may degenerate into an ulcer.

Trauma.—In a normal individual, any lesion of the skin of the leg, such as that caused by a kick, a scratch, or a cut, will heal quickly, and no ulcer result. Circumstances may arise which interfere with the healing process. Perhaps the most frequent cause which leads to the formation of an ulcer is infection with pyogenic organisms, and the prevention of the discharge from the wound. Occasionally there is also accidental contamination of the wound with some specific organism, such as that of diphtheria or phagedæna.

An important cause of want of healing of an ulcer is interference with its contraction. If contraction is impossible, as when a sore is situated over and adherent to a bone, healing may come to a standstill.

Deficient Innervation leads to loss of nutrition. Examples are seen in infantile palsy; rubbing of the boot or pressure of an instrument is prone to be followed by an obstinate ulcer. In cases of hemiplegia, even when the patient is lying on a water-bed, ulceration in the form of bedsores will occur much more rapidly on the paralyzed side than on the other. Perforating ulcer of the foot is a well-known sequel of *tabes dorsalis*, its other common cause being *diabetes mellitus*; ulceration and Gangrene (p. 255) are prone to occur because the resistance of a diabetic individual to micro-organisms is lowered, also because the arteries are often atheromatous, and possibly because the innervation of the whole body is interfered with.

Infective Ulcers.—The legs may be attacked by any form of acute infective ulcer, such as *anthrax* or *glanders*, but such an event is rare. The chief ulcers that belong to this group are chronic, and due to syphilis or tuberculosis.

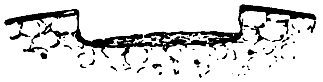

Fig. 298.—Diagram of a gummatous ulcer. Cleanly punched out. Slough on base.

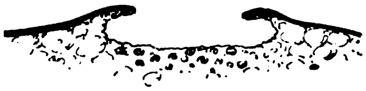

Fig. 299.—Diagram of a tuberculous ulcer. Undermined edges with perforations and tags.

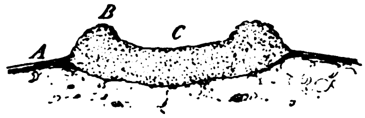

Fig. 300.—Diagram of an epitheliomatous ulcer. Growth in excess of destruction. *A*, Normal skin ; *B*, Heaped-up edges ; *C* Ulcerated portion.

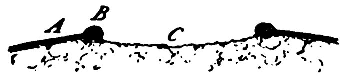

Fig. 301.—Diagram of a rodent ulcer. *A*, Normal skin ; *B*, Smooth, wire-like edges *C*, Shallow cavity.
(*From Introduction to Surgery, by Prof. Rutherford Morison.*)

Syphilitic Ulcers are the result of gummata which have formed in the subcutaneous tissues. These ulcerated gummata are almost always circular, and present a punched-out appearance (*Fig.* 298) ; they are generally multiple and tend to run into each other, so that the ulcer has a serpiginous outline. They tend to heal at one side while they progress at another. The scars are thin and supple, and if in the lower part of the leg, usually pigmented round the edges, white and slightly depressed in the centre. Gummata are often found with varicose veins or ulcers, and it seems probable that the low state of nutrition of the tissues caused by the obstruction of venous return is favourable to their formation. Diagnosis can in most cases be made on the distribution and shape of the ulcer, especially if they are on the outer aspect of the lower third of the leg; on the presence of other signs of syphilis; and on the effect of giving iodide of potassium or salvarsan. In cases of doubt, a Wassermann's reaction is of service.

Tuberculous Ulcer usually follows the formation and bursting of a tuberculous abscess, starting either in the subcutaneous tissue or in a bone, and the history may help materially in diagnosis. The ulcer is very chronic, and is characterized by undermining of the skin for a considerable distance from the edge (*Fig.* 299). The surface is pale, and the granulations are very small, with here and there small areas of caseation. Primary tuberculosis of the skin, or lupus, is not often found on the leg, though it may occur there as in any other part of the skin. A useful guiding rule is that lupus never starts later than the age of twenty and lasts for years, whereas a gumma starts at a later period and tends to heal spontaneously. In lupus the chief characteristic is the presence of minute, semi-transparent nodules at the margin of the ulcer and in the skin around, resembling apple jelly. If further methods of diagnosis are required, a diagnostic injection of Koch's old tuberculin may be used, or von Pirquet's skin test applied. A particular variety of tuberculous ulcer of the legs is described on p. 404 under the heading of Bazin's disease, or erythema induratum scrofulosorum.

Ulcerating Tumours.—*Epithelioma* may develop in a simple varicose ulcer that has existed for many years. The change may be very slow, or rapid. The ulcer spreads, the edges become heaped-up, everted, and indurated (*Fig.* 300). The femoral lymphatic glands become enlarged, and if the disease is allowed to progress, the bone is attacked. If any doubt arises as to a change in the character of an ulcer, a piece from the edge should be removed for histological examination.

Rodent Ulcer (*Fig.* 301) usually attacks the face (p. 735), though it may be found on any part of the body.

Sarcoma, starting in the deeper tissues, may fungate through the skin, which has given way before the pressure of the tumour.

George E. Gask.

ULCERATION OF THE THROAT.—(See Sore Throat, p. 613.)

ULCERATION OF THE TONGUE.—To enable a good view to be obtained of the affected part, the patient should be seated in a strong light and the protruded tongue gently wiped with a piece of soft linen to remove moisture. The presence of an ulcer being ascertained, its nature may be considered under the following heads :—

1. Carcinomatous
2. Syphilitic
3. Dental
4. Tuberculous
5. Dyspeptic
6. Ulcer in connection with stomatitis.

Carcinomatous Ulcer is much commoner in men than in women, probably owing to the fact that chronic glossitis due to smoking and syphilis is more common in the male sex. It is practically unknown before the age of thirty, and rarely starts before forty-five. The ill and wearied expression of the patient may awaken suspicion before the tongue is seen, for the pain and trouble caused by an epithelioma have a rapid and marked effect. The tongue in a normal individual can be protruded from one to one and a half inches beyond the teeth ; if the protrusion is limited, or if the tongue is not protruded straight, it can generally be inferred (except in cases of paralysis) that there is some tumour binding it down. The position of the ulcer is to be studied, and its relation to any sharp and carious tooth. Usually an epithelioma is on the side of the tongue, but there is no rule ; it may be anywhere on the upper, lateral, or under surfaces, or on the floor of the mouth.

As regards the ulcer itself, the typical appearance, when fairly developed, may be described as irregular, deep, foul, sloughy, with raised nodular everted edges, and a surrounding area of induration. The lymphatic glands are enlarged and hard, and they may be fixed. The submaxillary set is generally the first affected, but the disease sometimes misses these and infects the carotid and even the supraclavicular glands. Examination, therefore, should not be concluded before the whole of the neck had been palpated. The diagnosis should have been made, however, before the disease had developed thus far ; in its earliest stages an epithelioma may be represented by a superficial ulcer no more than

PLATE XXXII

CANCER OF THE TONGUE: VERY EARLY CONDITIONS

(From drawings kindly lent by the late Sir HENRY T. BUTLIN, F.R.C.S.)

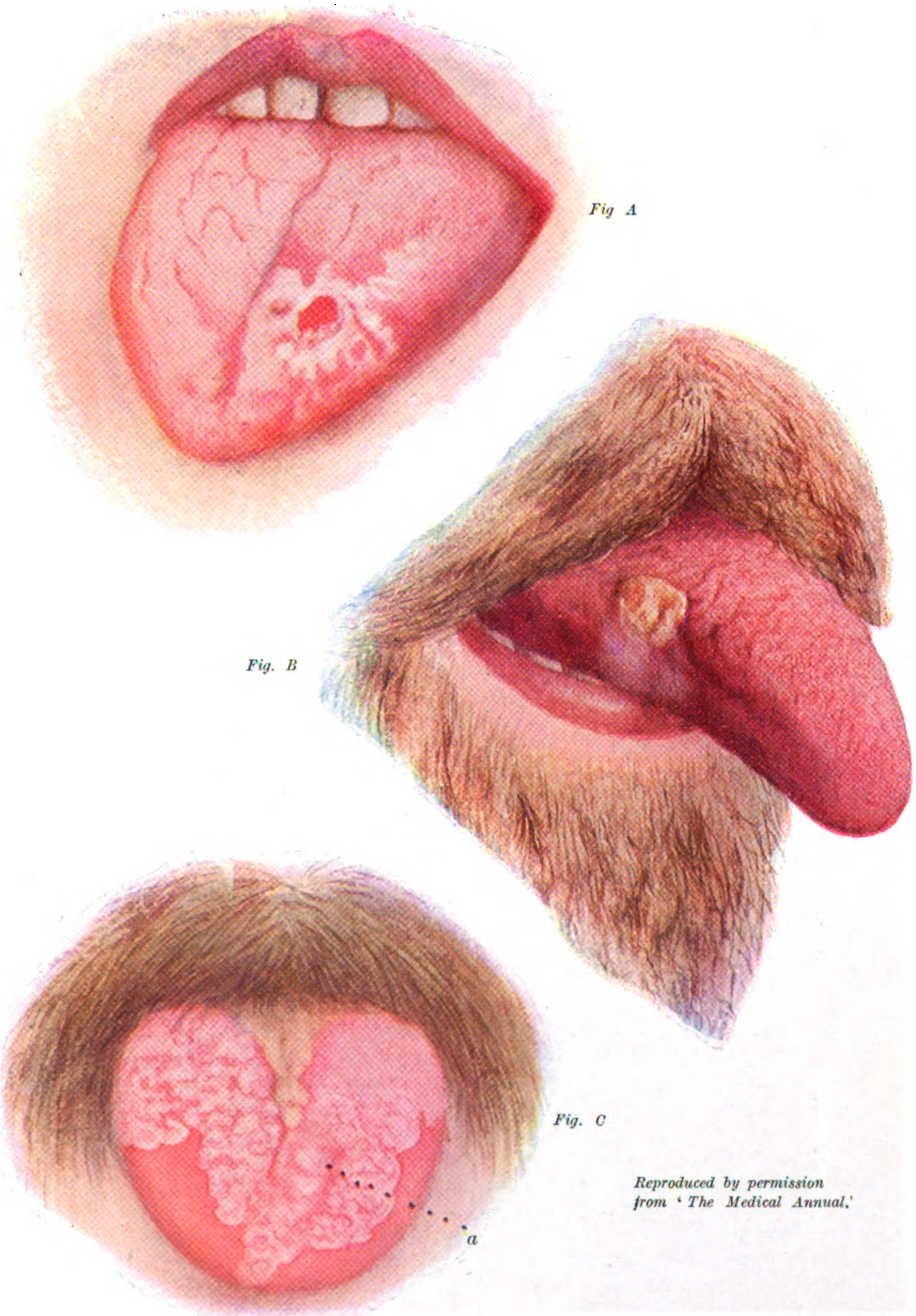

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Fig. A.—Old leukoplakia of many years' duration. Quite recent development of epithelioma, in the form of a very slightly raised smooth red plaque, feeling about as thick as a sixpence. Between it and the middle line is a tiny nodule resembling a pimple.

Fig. B.—Area of thin leukoplakia on the right border of the tongue, with a small epithelioma, which had developed in the site of a bite received several months previously.

Fig. C.—Leukoplakia of many years' duration, with the very earliest condition of epithelioma to the left of the middle line in the form of a very small area of leukoplakia (*a*), slightly more raised and a little firmer than the rest. The diagnosis depended as much on the slight hardening as on the appearance.

a sixteenth of an inch in diameter, by a crack or a small lump, without any enlargement of the glands. In all these conditions, however, the ulcer is already hard, and very resistant to any form of treatment. Any ulcer of the tongue occurring in a middle-aged man, and lasting for more than two or three weeks, should awaken suspicion.

Diagnosis from Syphilitic Ulcer.—This may be a very real difficulty, owing to the fact that the two conditions may exist side by side, and that the syphilitic leucoplakia or leucomic wart may be the actual precursor of a cancer. A positive Wassermann's reaction, therefore, is not proof that an epithelioma is not present. If a well-formed gumma is present, antisyphilitic remedies soon make a great change in its appearance, and a diagnosis may be made in this way, but not more than ten or fourteen days should be allowed to pass in uncertainty. There are many cases in which the cleverest surgeon is in doubt, and seeing the rapid course this disease runs, and the vital importance of securing an early diagnosis, it is urged here with the greatest possible insistence that the only certain method, and the one to be employed early, is that of taking out a piece of the ulcer, or, if small, the whole ulcer, and submitting it to histological examination.

Diagnosis from Dental Ulcer.—The ulcer in this case is caused by a bad tooth, and therefore is in a position on the tongue corresponding to the latter. Further, the ulcer is soft to the touch, and heals rapidly when the offending tooth is stopped or extracted. There is seldom difficulty in differentiation except when the ulcer is of very long standing.

Syphilitic Ulcer.—This may be primary, secondary, or tertiary. *Primary Syphilis* or *Chancre* is certainly rare on the tongue and, owing partly to its rarity and partly to the fact that it is unexpected, it is frequently missed. It is more common in men than in women, but it may occur even in children. It starts as a small pimple, which ulcerates and becomes indurated, though the induration is not so marked as when it is situate on the glans penis. The appearance of a secondary rash with general enlargement of the lymphatic glands would indicate the diagnosis with certainty, which might be confirmed by Wassermann's serum reaction, and the detection of spirochætæ (*Plate XXVIII, Fig. J*, p. 614) in serum from the sore. Furthermore, the sore heals rapidly under the influence of mercury.

Secondary Syphilis manifests itself by the formation of mucous patches and superficial ulcers. The latter are almost always multiple, and situated along the edges and tip of the tongue, and with them are also found similar sores on the mucous membrane of the cheek, lips, palate, and tonsil, and at the edges of the mouth. The ulcers are small, round, painful, with sharply cut edges and a greyish floor. Other secondary symptoms will be present to make the diagnosis clear.

Tertiary Syphilis or *Gummatous Ulcerations.*—These are divided into superficial and deep. *Superficial* gummata begin as small round-celled infiltrations in the mucous and submucous tissue. The ulcers are usually shallow, often irregular and associated with chronic glossitis, fissures, and leucoplakia. They are extremely important, for, as stated above, such a condition is often followed by an epithelioma. They are also very resistant to antisyphilitic remedies other than salvarsan. The ulcers themselves are not at first indurated, but if surrounded by interstitial fibrosis may appear hard; therefore a histological examination is eminently desirable if there is the least doubt. A *deep* gumma starts as a hard swelling in the substance of the tongue; later it softens, breaks down, and shows itself, generally in the middle line, as a deep cavity with irregular, soft, undermined walls, and a wash-leather-like slough at its base. It is not painful, and does not increase progressively in size. The important thing is to distinguish it from epithelioma and tuberculous disease. Unlike epithelioma, it is not hard, and its history is short. Furthermore, it yields very rapidly to potassium iodide or salvarsan.

Dental Ulcer is due to repeated small injuries from the sharp edge of a decayed tooth. It is therefore situated in such a position, generally on the side of the tongue, that it is opposite the tooth. The ulcer is single, small, superficial, and not indurated unless it is of long standing. It is therefore not easily mistaken for any other kind of ulcer, or if doubt arises it is allayed by the healing of the ulcer on stopping or extracting the tooth.

There is a form of dental ulcer which is found on the frænum of the tongue in children suffering from whooping-cough; during the violent expiratory spasms peculiar to the illness, the under surface of the tongue may suffer from rubbing over the lower incisor teeth.

Tuberculous Ulcer of the Tongue is rare, but it occurs at that period of life during which tuberculous disease of the lung is common, that is to say, between the ages of fifteen and thirty-five. It is due to infection with tubercle bacilli brought up into the mouth, and if a patient is found to be suffering from tuberculous disease of the lungs or larynx and also from an ulceration of the tongue, there is a strong probability that the latter is of the same nature as the former. The ulcer itself may be situated on the tip or side of the tongue ; it has an irregular outline, and the base is nodular, sloughy, or caseous. It has often been mistaken for epithelioma or gumma. The fact that it is not hard, and that phthisis is present, should put one on one's guard. As against gumma, a Wassermann's reaction would be negative ; moreover, the ulcers are often small and multiple, more nearly resembling dyspeptic ulcers. A von Pirquet's test or a diagnostic injection of Koch's old tuberculin might be employed, but a more reliable method is the removal and microscopical examination of a piece of the ulcer, when the histological appearances of tubercle will be seen. The tubercle bacillus (*Plate XXVIII*, *Fig. K*, p. 614) is not always found.

Dyspeptic Ulcer, as the name implies, is connected with disorders of digestion. The ulceration is often multiple, each ulcer being round, small, often covered with a greyish slough, and with a bright ring of inflammation round it. They are situated on the dorsum and edges of the tongue near the tip. The mouth, too, is very foul, there may be similar ulcers on the inner aspects of the lips and cheeks, and the cervical glands may be enlarged.

Ulcers in connection with Stomatitis (Ulcerative Stomatitis).—Septic infection of the mouth due to a variety of causes, such as irritation from decayed teeth, alkalies, acids, or mercury, may be accompanied by the formation of small vesicles which, on bursting, give rise to superficial ulcers. They are not limited to the tongue, but appear on the mucous membrane of the cheeks and gums as well. Aphthous stomatitis commonly occurs in conjunction with the febrile diseases of childhood. It is characterized by the formation of whitish spots on the buccal mucous membrane, and by the shedding of epithelium small superficial ulcers may be formed. The ulcers of the tongue are here, so to speak, accidental, occurring in the course of a general inflammation of the mouth, and will hardly be confounded with any other form of ulcer except so-called dyspeptic ulcers, and there is no real line of demarcation between the latter and the stomatitic variety.

George E. Gask.

UNCONSCIOUSNESS.—(See Coma, p. 117.)

UNEQUAL PULSES.—(See Pulses, Unequal, p. 550.)

UNEQUAL PUPILS.—(See Pupil, Abnormalities of the, p. 551.)

URATE DEPOSIT IN THE URINE.—A precipitate of urates is often recognizable at once by its pink colour, due to their carrying down with them the uroerythrin pigment of the urine. Urates themselves are white, however, and if, as is sometimes the case, there is no uroerythrin present for them to carry down, they form a white precipitate which may be mistaken for mucus, phosphates, or pus. They may be distinguished at once, however, by warming the urine back to body temperature ; they re-dissolve long before boiling-point is reached. They are also soluble in liquor potassæ, unlike phosphates. Microscopically they are nearly always amorphous, though in rare cases they assume the form of small spheres with irregular projecting spicules—the so-called 'thorn-apple' or 'hedgehog' crystals.

Their only significance from a clinical point of view is that they indicate a concentrated urine. It does not follow that a urine is not concentrated if no precipitate of urates occurs, but the fact that the urates re-dissolve on warming serves to show that, although there may be enough water to keep them in solution at body temperature, the urine becomes supersaturated with them as it cools, and precipitates them out. The reason for the urinary concentration has to be learned from collateral evidence. It may be that there has been much sweating, and in hot weather a precipitation of pink urates is a very common physiological condition, which is apt to alarm some patients when they first notice it. On the other hand, the concentration may be due to pathological conditions, of which the commonest are fevers, chronic valvular disease with heart failure, and maladies which lead to loss of fluid by vomiting, sweating, or diarrhœa. The urates themselves afford hardly any clue to the cause of the concentration, and their appearance is just the same whether their deposition is due to physiological or pathological changes.

The most marked examples of uratic deposits are to be seen in cases of acute rheumatic fever, lobar pneumonia, and chronic heart disease with failing compensation. It is a general rule, moreover, that when the kidneys are themselves affected there is decidedly less tendency for uratic deposits to form than when the primary disease is in the heart or lungs; thus when one may be in doubt as to whether a given case of chronic heart failure is due to primary renal disease or primary heart disease, an abundant urate deposit affords some evidence in favour of the latter and against the former. It is no absolute rule, however, and almost any concentrated urine may precipitate urates.

Students are a little apt to confuse the significance of urates with that of uric acid, though the two are entirely independent from a clinical standpoint. *Herbert French.*

URETHRA, DISCHARGE FROM.—(See DISCHARGE, URETHRAL, p. 181.)

URETHRA, FÆCES PASSED THROUGH.—(See FÆCES PASSED PER URETHRAM. p. 238.)

URIC ACID DEPOSIT IN THE URINE.—The most typical form taken by a precipitate of uric acid in a urine is the cayenne-pepper deposit, seldom voluminous, of characteristic light-brown, prismatic crystals (*Fig.* 302), arranged either as separate 'whetstones,' or in overlapping bundles, or 'rosettes'; occasionally, crystallization is imperfect, and they appear as 'dumb-bells.' Intrinsically, they are colourless; but they differ from all other urinary deposits in that they carry down with them the ordinary yellowish-brown urochrome pigment of the urine. For clinical purposes the best test for them is the microscope.

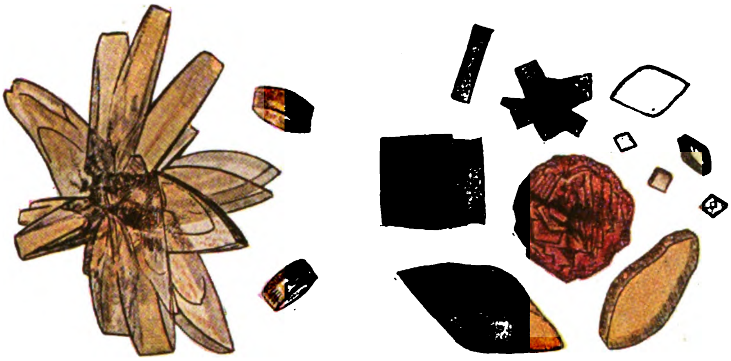


Fig. 302.—Uric acid crystals of various types.

Besides the cayenne-pepper deposit, uric acid crystals may be present in considerable numbers in the midst of other precipitates, such as mucus, or oxalate of lime; in which case they may not be discernible without the use of the microscope; or, again, they may become aggregated together into small pellets or calculi, which the patient may be conscious of as 'gravel' on micturition.

A deposit of uric acid is generally found in a decidedly acid urine of high specific gravity; but it may occur in urines of almost any reaction or specific gravity. A great deal more importance than it deserves has been attached to the supposed relationship between this uric acid and gout. *A cayenne-pepper deposit by no means indicates gout*; indeed, it may be perfectly physiological, occurring abundantly sometimes in healthy young persons, particularly boys. It does not even follow from its occurrence that there is excess of uric acid, either in the urine or in the tissues; for the precipitation depends nearly as much upon the relative proportions of phosphates, chlorides, and inorganic sulphates to uric acid, and upon the absolute and relative amounts of sodium, potassium, and other bases in the urine, as upon the absolute amount of uric acid. The greater the tendency of the bases to form phosphates, by mass action or otherwise (see PHOSPHATURIA, p. 523), the less the tendency for the soluble quadriurates, and the greater the liability for less soluble biurates, to be produced, the relatively insoluble uric acid being liberated from the latter and deposited in crystalline form.

Considerable care has to be exercised, therefore, before any useful clinical deductions can be drawn from the fact that a urine contains a deposit of uric acid. It is true that a persistent tendency to it is often associated with gout; but the latter should be diagnosed from the collateral evidence rather than upon the uric acid crystals in the urine. Many gouty subjects precipitate no uric acid in their urine at all. Naturally, there will be a greater tendency to such deposition when the total amount of uric acid present is greater than normal. Uric acid in the urine is derived from two sources—exogenous and endogenous. The exogenous are such foodstuffs as are rich in nucleo-proteid, and in the so-called xanthin bases, or purin, or alloxuric bodies, xanthin, guanin, hypoxanthin, adenin, heteroxanthin, paraxanthin, episarkin, epiguanin, methylxanthin, and carnin, which are mainly derived from nuclein. Analyses of the various foodstuffs as to purin bases need not be given here, for it is easy to remember that, broadly speaking, these substances are contained in largest quantities in the richest food. A considerable proportion of the xanthin bases are excreted as uric acid, and it is common knowledge that rich foods tend to increase uric acid in the urine. Endogenous uric acid, on the other hand, is derived from the patient's own tissue metabolism. Birds excrete nearly all their nitrogenous waste as uric acid; man excretes his mainly as urea, and only to a minor extent as uric acid. Sometimes, however, too much of his nitrogenous metabolism stops short at the stage of uric acid, instead of the latter being nearly all converted into urea; he then excretes an abnormal total quantity of uric acid, with the result that it may be precipitated in crystalline form. One repeats, that this does not necessarily constitute gout, however; it occurs in certain healthy subjects, in leukæmia, in pernicious anæmia, during the course of certain fevers, and in some cases of chronic heart disease. Perhaps one of the best ways of avoiding too narrow a conception in regard to this uric acid is to remember that in some respects the human body is a fire; fires may burn their coal well or badly; if well, the residue is but a little ash; if badly, the residue is not ash, but clinker; uric acid is the clinker of the human body, and many different things that make human nitrogenous metabolism incomplete may cause a deposition of this clinker in the urine. Gout is one such thing; but excessive eating, deficiency of exercise, biliousness, and various chronic imperfections of the circulation, or digestion, may do so; and the same may occur in apparently healthy subjects who have never had any untoward symptoms at all. Oxalate of lime (see OXALURIA, p. 423) is possibly derived in part from similar imperfect combustion of carbohydrates or fats, and it is noteworthy how often crystals of uric acid and of oxalate of lime occur, either together, or alternating with one another. Still further, error of metabolism may produce glycosuria in association with uric acid crystals, so-called gouty glycosuria.

Besides being evidence of overloading, or of imperfect combustion in a general sense, the occurrence of a uric-acid deposit may be of particular clinical importance in certain cases of *frequency of micturition*; of *urethritis*; and of *renal calculus*. Necessity to micturate frequently, only small quantities of urine being passed at a time, is a symptom that in young people suggests cystitis, possibly tuberculous; enlargement of the prostate in men over sixty; or some uterine or other pelvic malady in women. It is important to remember, however, that undue acidity of the urine, with a tendency to deposit crystals of uric acid, or oxalate of lime, may produce the same symptom in considerable degree. It is sometimes spoken of as irritability of the bladder; the highly acid urine irritates the vesical mucosa, and it may produce actual cystitis. The same irritation may inflame the urethral mucosa, and produce a 'gouty' urethritis; and, perhaps, epididymo-orchitis, which may be mistaken for the gonococcal form, unless pus films can be shown to contain no gonococci.

If the patient has suffered from renal colic, hæmaturia, or vesical pain, suggestive of calculus in the kidney, ureter, or bladder, the discovery of abundant uric acid crystals in the urine affords confirmation of the diagnosis of a uric-acid stone, particularly if they are obviously aggregated together into tiny calculi; there are generally red corpuscles, excess of leucocytes, and tailed epithelial cells from the renal pelvis, or pyriform cells from the deeper layers of the bladder mucosa, at the same time.

The danger of diagnosing glycosuria in the absence of sugar when uric acid is abundant in a urine needs special mention. Uric acid has considerable power of reducing Fehling's solution. It seldom gives the copious brick-red or orange-yellow precipitate that is characteristic of abundance of sugar, but it may give just enough reduction or change of colour to make it doubtful whether sugar is present or not. More than a few proposers for life insurance have suffered unfairly on this account; no such partial reduction should be regarded as due to sugar until the presence of glucose has been confirmed by other means, particularly the phenylhydrazine and the fermentation tests. *Herbert French.*

URINE, ABNORMAL COLORATION OF.—This may be due to: (1) The presence in abnormally large quantities of certain urinary pigments, such as uroerythrin or hæmatoporphyrin; (2) The presence of pigments formed in the organism, but which are not normally excreted in the urine, such as hæmoglobin and the pigments of the bile; (3) The presence of pigmentary substances derived from drugs or foods, or administered directly by the mouth.

Urines of unusual tints may be classified conveniently according to the colours which they exhibit, as follows: (I) *Yellow and orange urines*; (II) *Pink and red urines*; (III) *Brown and black urines, including such as are of normal tint when passed, but darken on exposure to air*; (IV) *Green and blue urines.*

Yellow and Orange-coloured Urines.—The normal yellow tint is wholly due to urochrome, for other urinary pigments are present in traces so minute that their presence has no obvious effect. However much it be diluted, normal urine remains yellow as long as any tint is visible. In some cases of diabetes insipidus the urine is almost colourless, and the abundant urine of diabetes mellitus usually exhibits a peculiar pale bright greenish-yellow tint (*Plate XXXIV*, *Fig.* 11, p. 748), which has not yet been explained.

Urobilin, when present in large amount, imparts a rich orange-yellow colour; and when seen in very thin layers, as near the apex of a conical glass, urines rich in urobilin have a pinkish tint, due to selective absorption in the middle of the spectrum. Such urines, when examined with the spectroscope, show a dark absorption band near the solar F line (*Fig.* 36, p. 80).

Urobilinuria—the excretion of excess of urobilin—may result from widely different causes, and as a consequence, its clinical significance is not so clear as might be expected. The symptom is met with in connection with hæmolytic diseases, such as *pernicious anæmia*, in *diseases of the liver*, such as *cirrhosis*, and in cases in which *excessive bacterial action* is going on *in the intestine.* The bulk, if not the whole, of the urobilin of urine is derived from the intestine, where it is formed by the action of the bacteria present upon bilirubin. It is present in abundance in normal fæces, and in traces in normal urine. The test for it is given on page 324.

Uroerythrin—the highly unstable pigment to which the colour of pink urate sediments is due—when abundantly present in solution in the urine imparts to it a rich orange-red colour, which may even be mistaken for that due to blood. The colour is changed to a pale greenish-yellow by addition of an alkali. Hepatic derangements of almost all kinds, including the most trifling functional disturbances, may lead to the appearance of uroerythrin in the urine; but the most intensely pink urate sediments are seen in cases in which the liver is the seat of pronounced morbid changes, such as cirrhosis, or the passive congestion due to cardiac disease.

Choluria.—Urine which contains bilirubin has a rich orange colour with a greenish tint at the edge of the meniscus. The foam formed by shaking it has a yellow colour, whereas that of bile-free urine, even when deeply pigmented, is colourless. The colour of the urine may be much modified by the presence of biliverdin, in addition to bilirubin, and may approach to black or dark green.

The presence of bile pigment may be demonstrated by *Gmelin's test.* This is best carried out by allowing the urine to flow gently on to the surface of some nitric acid in a test tube; on gently shaking, the familiar play of colours is seen at the junction of the liquids, and the urinary layer often retains the green tint of biliverdin for a considerable time. Again, a green ring is observed when diluted tincture of iodine is allowed to flow on to the surface of the urine in a test tube (*Plate XXXV*, p. 750).

When the quantity of bile pigment present is very small, the above tests may fail to reveal its presence, and *Huppert's test* may then be resorted to. A precipitate is formed by the addition, to a much larger volume of urine, of a solution of barium chloride and baryta water, or of calcium chloride and lime-water. The precipitate, which carries down any bile pigment which may be present, is filtered off and washed into a test tube with alcohol. Dilute sulphuric acid is then added, and the test tube is heated in a beaker of boiling water. If bile pigment be present, the acidulated alcohol acquires a rich green tint, due to biliverdin.

Choluria is merely a symptom of jaundice, but the appearance of bile pigment in the urine may precede any yellow coloration of the conjunctivæ or skin, or, as in cases of acholuric family jaundice (*Plate XVIII*, p. 332), the skin may be tinted although the urine is free from bile pigment. In the very rare cases in which a fistula exists between the biliary and urinary tracts, choluria of pronounced degree has been observed, apart from any jaundice.

Certain *drugs* impart to urine a tint which, although yellow, is abnormal. This is seen when *santonin* is administered, or *chrysophanic acid*, which is a constituent of rhubarb and senna. In either case, the urine turns pink on addition of an alkali, but the pink colour is far more brilliant after santonin than after chrysophanic acid has been taken.

Pink and Red Urines.—The conditions which lead to the excretion of a pink or red urine may be classified as follows: (1) Hæmaturia, in cases in which the blood pigment appears in the urine mainly as oxyhæmoglobin; (2) Hæmoglobinuria—usually in cases which do not belong to the paroxysmal class; (3) Hæmatoporphyrinuria; (4) Administration of rosaniline as a drug; (5) Eating of sweetmeats coloured with eosin; (6) Presence of chrysophanic acid in an alkaline urine.

Hæmaturia and *hæmoglobinuria.*—For the significance of these symptoms, and the detection of blood pigment in urine, the special articles dealing with them may be referred to (p. 275 and p. 284).

Hæmatoporphyrinuria is a condition in which urine is passed which has a pink, port-wine, or nearly black colour, and which contains considerable quantities of the hæmoglobin derivative, hæmatoporphyrin. In the darker specimens the colour is mainly due to other little-known pigments which accompany the hæmatoporphyrin. For the recognition of the condition spectroscopic examination is necessary. It is most liable to be mistaken for hæmoglobinuria, but when the urine contains no albumin the distinction is not difficult; when albumin is present in a pink urine, the diagnosis is more difficult, because the spectrum of hæmatoporphyrin in the combination in which it usually occurs in such cases resembles that of oxyhæmoglobin somewhat closely. However, the addition of hydrochloric acid changes the spectrum to that of acid hæmatoporphyrin instead of to that of acid hæmatin (*Fig.* 34, p. 80).

If a mixture of 10 per cent calcium chloride solution and lime-water be added to the urine, the precipitate formed carries down all the abnormal pigments, and the filtrate is yellow. From the precipitate the hæmatoporphyrin may be extracted with acidified alcohol, and its highly characteristic spectra may be observed and identified. The trace of hæmatoporphyrin present in normal urine escapes detection by such means, but the increased quantities present in a variety of morbid conditions may be revealed by faint bands, even in cases which do not fall into the class under consideration, and in which the urine shows no obvious anomaly of pigmentation.

In the great majority of cases, hæmatoporphyrinuria results from prolonged administration of *sulphonal* in medicinal doses, and forms one of a group of toxic symptoms of much gravity, which often usher in a fatal ending. These symptoms may only develop after the drug has been taken for months or even years, and even some days after its administration has been stopped. Their development calls for the free administration of sodium bicarbonate. It is a remarkable fact that such toxic effects of sulphonal are seldom seen except in women, and the few male cases on record have mostly been of a mild kind.

Much more rarely, hæmatoporphyrinuria, with deep red urine, is met with in cases in which it cannot be ascribed to any drug. Several patients have been sufferers from *hydroa æstivale*, others from tuberculosis, and others from maladies so different from each other that no definite diagnostic significance can yet be assigned to the symptom. In such cases the hæmatoporphyrinuria does not appear to have the grave import which it has in sulphonal cases, nor is there manifested any special liability of the female sex.

Coloration by constituents of foods and drugs.—*Rosaniline*, which was at one time employed in the treatment of albuminuria, imparts a pink colour to the urine which, provided that it be known that the drug is being taken, offers no diagnostic difficulty. Aniline dyes have also, ere now, been deliberately added to the urine for the purpose of simulating hæmaturia.

Eosin has been employed extensively for the coloration of pink sweetmeats and lozenges, and the urine of those who eat such sweetmeats in considerable quantities acquires a rich pink colour, and shows a brilliant green fluorescence. The nature of such pigmentation can hardly be mistaken by anyone who is aware of the fact that eosin is so employed.

Drugs which contain *chrysophanic acid* are used so frequently as aperients that this compound may rank as a common constituent of urine ; and if, from any cause, the urine be alkaline, it acquires a pink or red colour, which may easily be misinterpreted. However, the history of the taking of rhubarb or senna, and the fact that the addition of an acid changes the colour of the urine to a bright yellow, renders the diagnosis easy. The pink colour which alkalies impart to the urine of patients taking santonin is so fugitive that it does not call for consideration here.

Brown and Black Urines.—The urine may be brown or black in the following conditions : (1) Jaundice ; (2) Hæmaturia ; (3) Hæmoglobinuria ; (4) Hæmatoporphyrinuria ; (5) Indicanuria ; (6) Melanuria ; (7) Alkaptonuria ; (8) Carboluria ; and after the administration of certain other drugs, such as salol, salicylates, resorcin, gallic acid, and uva ursi.

In some of the above conditions the urine has such coloration when passed ; but in others, such as melanuria and alkaptonuria, the urine is usually of normal tint when freshly passed, and only darkens on standing in contact with the air.

Brown and black *jaundiced urine* is met with chiefly in cases of long-standing icterus, in which the skin has acquired a dull greenish tint, and the urine contains biliverdin as well as bilirubin.

In some of the early recorded cases of black urine, the colour was certainly due to *blood pigment*, and the smoky colour of many urines which contain blood pigment in the form of methæmoglobin is familiar to all. In *paroxysmal hæmoglobinuria* also, the urine is not infrequently almost black. The ordinary tests for hæmoglobin, together with microscopic and spectroscopic examination, serve to reveal the nature of such cases (*Figs.* 30 *et seq.*, p. 80).

That the urine of *hæmatoporphyrinuria* may approach to actual blackness, owing to the abundant presence of purple pigments which have no characteristic spectra, has already been mentioned in the account of that symptom above.

Indicanuria.—Urines which contain much indican may show no abnormality of tint ; but occasionally, and especially in extreme cases, there are present in the urine, in association with the colourless indoxyl sulphate, other and higher oxidation products of indol, which impart to it a brown colour, intensified or developed on exposure to air. This variety of brown or black urine is recognized less than it should be, and it is probable that the condition has been mistaken not infrequently for melanuria. Such urine is not blackened, as that of melanuria is, by the addition of ferric chloride, nor by nitric acid in the cold, but does blacken when heated with nitric acid. The ordinary tests for indican reveal its presence in large amount. Thus, if a specimen of the urine be heated with an equal volume of hydrochloric acid, and a drop of a dilute solution of bleaching powder, or a drop of nitric acid, it becomes black. If, after cooling, the dark-coloured liquid be shaken with chloroform, the latter takes up indigo-blue or -red and acquires a deep purple colour ; but the supernatant liquid remains black. If the chloroform extract be separated and evaporated to dryness, the indigo-red may be dissolved out of the residue with alcohol, whereas the indigo-blue, which is insoluble in alcohol, may be taken up afterwards with chloroform.

Indicanuria signifies abnormal amount of protein decomposition in the alimentary canal, brought about by intestinal bacteria ; but it is stated that it may also have its origin in collections of putrid pus, such as putrid empyemata. In such a case recently under the writer's care, the abundant indican disappeared from the urine when a dose of calomel was given, and was probably of intestinal origin.

Melanuria.—This is a symptom which is met with in some cases of *melanotic sarcoma.* The urine, when freshly passed, contains a colourless chromogen, melanogen, and usually has a normal tint. On exposure to air, it darkens quickly, owing to oxidation of the melanogen to melanin, becomes brown, and eventually quite black. When nitric acid is added to such a urine, it causes prompt blackening, even in the cold, and immediate blackening also follows the addition of a solution of ferric chloride. This is the most valuable and characteristic of the tests for melanuria. Bromine water produces a yellow or brown precipitate which quickly blackens.

As a rule, melanuric urines, when treated with liquor potassæ and sodium nitroprusside, yield a deep Prussian blue on acidification with acetic acid, but this reaction is not due to the melanogen as such, is yielded by some other urines, and cannot be taken as diagnostic of melanuria.

It is stated frequently that melanuria may be met with apart from melanotic growths, in cases of wasting and other diseases. There is little doubt that some of the cases quoted in support of this contention, and which were recorded before the more distinctive tests for the condition were known, were, in reality, examples of indicanuria, such as have been described above, and the writer has never met with true melanuria save in cases of melanotic sarcoma. Even in such cases it is not seen so long as the tumour is confined to its primary seat, but only when it has invaded the viscera, and especially the liver. Indeed, the quantity of melanogen excreted is apparently dependent upon the extent to which the liver has been invaded, and the amount of pigmentation in the growths of which it is the seat. Hence it happens usually that the diagnosis of the case has already been established before the peculiar pigmentation of the urine is developed.

Alkaptonuria is the outward sign of a very rare anomaly of metabolism which is almost always congenital, and persists through life without any serious detriment to the health of its subjects. The peculiar properties of the urine are due to the excretion in it of an aromatic acid, homogentisic or hydroquinoneacetic acid, a product of katabolism of tyrosin and phenylalanin. It is, in all probability, a product of normal metabolism, which in normal individuals undergoes complete destruction.

Alkapton urine seldom exhibits any abnormality of tint when passed ; but darkens quickly on exposure to air, undergoing changes through brown to black, which resemble in the closest manner those seen in melanuric urines. However, the two conditions are distinguished readily by means of simple tests. When a dilute solution of ferric chloride is added to alkapton urine, a deep blue colour appears for a moment, and reappears after each subsequent addition of the reagent, until oxidation of the homogentisic acid is completed. Unless the reagent be very dilute, oxidation occurs too rapidly, and the blue colour is missed.

The addition of an alkali causes very rapid darkening, with absorption of oxygen, and heat increases the rate of blackening.

As homogentisic acid is a powerful reducing agent, alkapton urines give some of the reactions of glycosuria. Fehling's solution is reduced freely with the aid of heat, but the blackening effect of the alkaline reagent gives a peculiar appearance to the reaction. No black precipitate is obtained with Nylander's reagent, but the alkali therein causes conspicuous darkening. The safranin reaction is not obtained, and alkapton urine is optically inactive. An ammoniacal solution of silver nitrate is reduced rapidly even in the cold, a reaction which is made use of for the quantitative estimation of homogentisic acid. It is because alkaptonuria is so rare, rather than because its recognition presents any special difficulty, that its properties are not widely known and not infrequently fail of recognition.

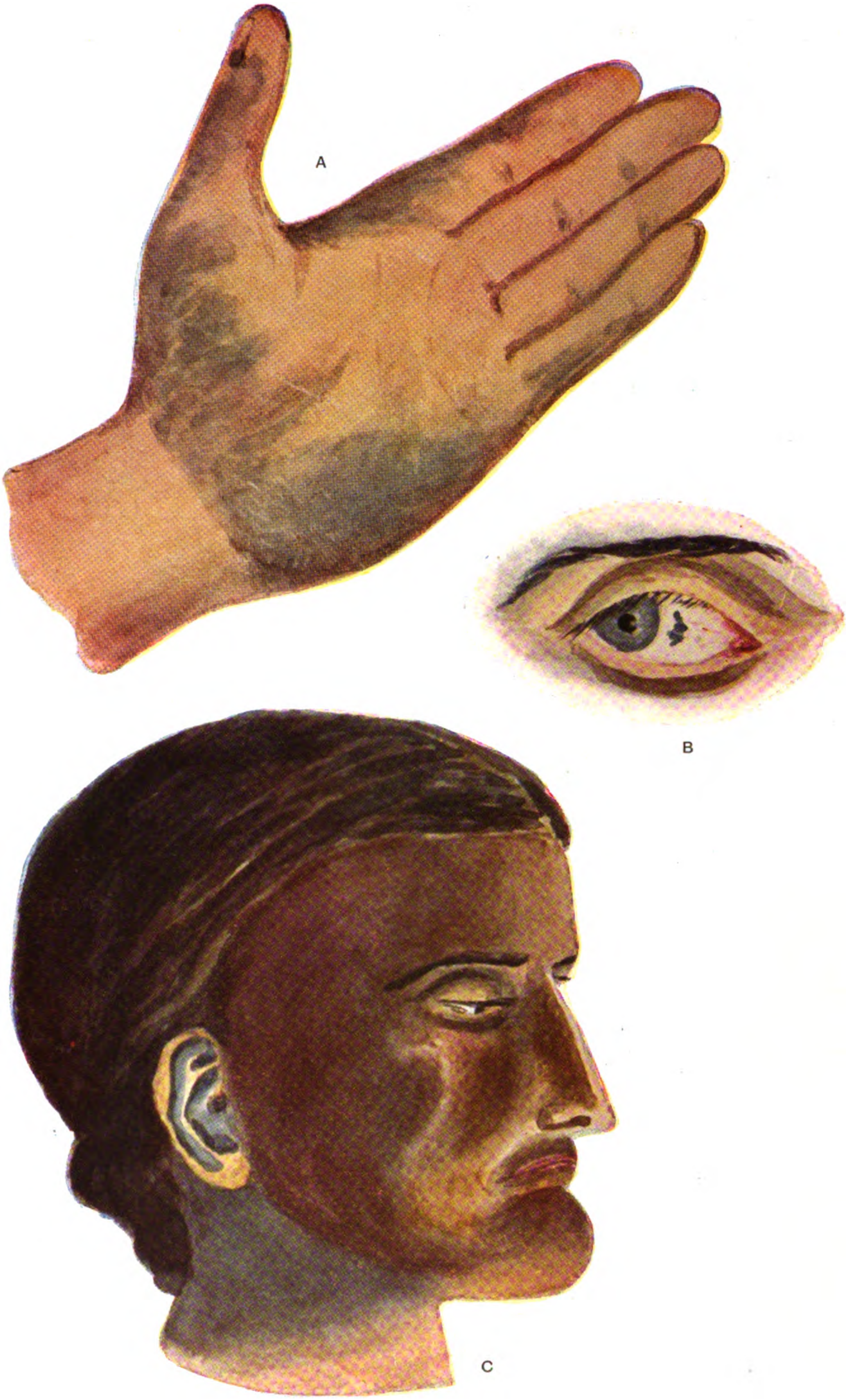
Ochronosis, i.e., a blackening of the cartilages, and deep pigmentation of regions of the skin, a bluish-black coloration of the ears, and pigment spots on the conjunctivæ (*Plate XXXIII*), is sometimes the outcome of alkaptonuria ; but there is a group of cases in which similar pigmentation results from the application of carbolic acid to chronic ulcers over long periods of years. There is also some reason to believe that the lesions of joints which sometimes accompany ochronosis may also be results of alkaptonuria.

Carboluria.—A darkening of the urine, increased by exposure to air, is seen frequently after the administration of certain drugs which contain phenol, in carbolic acid poisoning, and as the result of outward application of carbolic acid. A carbolic acid compress applied to the head of a child for the destruction of pediculi quickly induces carboluria, and the taking of salol is another common cause. The urine has a smoky tint, or in cases of carbolic acid poisoning may be actually black. In the slighter cases it is best described as brown with a greenish tinge, and the meniscus, when seen from the side, appears black.

There is no direct chemical test for carboluria, and the diagnosis is usually based upon the knowledge that phenol, or some derivative or compound thereof, has been administered or applied. After boiling the urine for some time with Fehling's solution, a slight reduction

PLATE XXXIII

OCHRONOSIS



The face is coloured dark brown (C), and bears a striking resemblance to a case of Addison's disease; the ears have a bluish appearance, and hands (A) show a similar discoloration. Note the small bluish patch which was present on each conjunctiva (B).

is observed ; but this is in no way comparable with that seen with alkapton urine. Indirect evidence is obtained by the addition of a solution of barium chloride, which in cases of carboluria produces a very slight precipitate or none at all. If, however, the urine be first boiled with hydrochloric acid, a precipitate is obtained such as is yielded by normal urines. This is due to the fact that, in the presence of abundant phenol and oxidation derivatives thereof, the sulphates of the urine are for the most part, or even wholly, combined as aromatic sulphates, which yield no precipitate with barium salts, whereas, when the aromatic sulphates are broken up by hydrochloric acid, a precipitate of barium sulphate is thrown down.

The diagnosis of the other varieties of brown or black urine which have their origin in the administration of drugs, is based upon the fact that salicylates, or other drugs capable of producing such pigmentation, have been taken.

Green and Blue Urines.—In some cases of jaundice, the bile pigment excreted is so largely in the form of biliverdin that the urine has a dark green colour : but with this exception, practically all green urines met with in practice owe their colour to the taking of methylene blue, either as a drug or in sweetmeats. When the dose is small, the tint may be a rich green ; but after larger doses, the urine is frankly blue. It is not always easy to account for the origin of such coloration of urine, for the patient may be quite unaware that he has taken methylene blue in any form, although examination of his urine may leave no doubt that he has done so. Sweetmeats are sometimes coloured with this pigment, as they are with eosin, and it is sometimes used to correct the colour of white sweetmeats. Again, a pill of methylene blue has before now found its way, either by accident or design, into a supply of pills of another kind. Absence of a known cause does not, therefore, by any means exclude this kind of pigmentation ; and experience shows that unless it can be shown, by careful examination, that the colour of the urine is not due to methylene blue, it is needless to search for any other causation.

Although the green urine which follows the taking of methylene blue may appear perfectly limpid, the blue pigment is not held in solution but in suspension, and is, to a large extent, removed even by a single filtration. The green colour of the filtrate is greatly reduced, and the filter paper shows a blue stain. The pigment upon the filter yields a blue solution in chloroform, and if the chloroform solution, or the blue extract obtained by shaking the urine with chloroform, be shaken with liquor potassæ in a test tube, the chloroform is decolorized, and the supernatant alkaline liquid acquires a pink tint. The original urine, or the chloroform extract, shows an absorption band in the red of the spectrum which may be mistaken for that of indigo-blue.

There is no reason to think that indigo-blue ever produces a green or blue coloration of urine similar to that due to methylene blue. By the spontaneous breaking down of indoxyl-glycuronic acid, usually in alkaline urines, indigo-blue may be set free, and may form a dark blue sediment, or may impart a blue colour to the phosphatic film upon the surface ; and when, in the earlier years of the last century, indigo-blue was employed somewhat frequently as a drug in the treatment of epilepsy, a dark purple colour of the urine of patients so treated was observed, but under no circumstances are indigo pigments formed spontaneously in quantities sufficient to bring about such a result. *A. E. Garrod.*

URINE, ACETONE IN.—(See Acetonuria, p. 3.)

URINE, ALBUMIN IN.—(See Albuminuria, p. 4.)

URINE, ALBUMOSE IN.—(See Albumosuria, p. 15.)

URINE, BACTERIA IN.—(See Bacteriuria, p. 69.)

URINE, BENCE-JONES BODY IN.—(See Albumosuria, p. 15.)

URINE, BILE-PIGMENT IN.—(See Urine, Abnormal Coloration of, p. 743.)

URINE, BLACK.—(See Urine, Abnormal Coloration of, p. 743.)

URINE, BLOOD IN.—(See Hæmaturia, p. 275.)

URINE, CASTS IN.—(See Albuminuria, p. 4.)

URINE, CHYLE IN.—(See Chyluria, p. 108.)

URINE, CYSTIN IN.—(See Cystinuria, p. 161.)

URINE, DIACETIC ACID IN.—(See Acetonuria, p. 3.)

URINE, DIAZO-REACTION IN.—(See Diazo-Reaction, p. 173.)

URINE, EXCESS OF.—(See Polyuria, p. 534.)

URINE, FÆCES IN.—(See Fæces Passed per Urethram, p. 238.)

URINE, FAT IN.—(See Chyluria, p. 108.)

URINE, GAS IN.—(See Pneumaturia, p. 529.)

URINE, GLUCOSE IN.—(See Glycosuria, p. 260.)

URINE, HÆMOGLOBIN IN.—(See Hæmoglobinuria, p. 284.)

URINE, INCONTINENCE OF.—(See Micturition, Abnormalities of, p. 393.)

URINE, INDICAN IN.—(See Indicanuria, p. 314.)

URINE, METHÆMOGLOBIN IN.—(See Hæmoglobinuria, p. 284.)

URINE, MUCUS IN.—(See Mucus in the Urine, p. 399.)

URINE, OXALATE DEPOSIT IN.—(See Oxaluria, p. 423.)

URINE, OXYBUTYRIC ACID IN.—(See Acetonuria, p. 3.)

URINE, PHOSPHATES IN.—(See Phosphaturia, p. 522.)

URINE, PUS IN.—(See Pyuria, p. 574.)

URINE, RETENTION OF.—(See Micturition, Abnormalities of, p. 393.)

URINE, SUGAR IN.—(See Glycosuria, p. 260.)

URINE, SUPPRESSION OF.—(See Anuria, p. 39.)

URINE, URATE DEPOSIT IN.—(See Urate Deposit in the Urine, p. 740.)

URINE, URIC ACID DEPOSIT IN.—(See Uric Acid Deposit in the Urine, p. 741.)

UTERUS, BLEEDING FROM.—(See Menorrhagia, p. 385; Metrorrhagia, p. 390; and Metrostaxis, p. 392.)

UTERUS, PROLAPSE OF.—(See Prolapse of the Uterus, p. 538.)

VAGINA, BLEEDING FROM.—(See Menorrhagia, p. 385; Metrorrhagia, p. 390; and Metrostaxis, p. 392.)

VAGINA, DISCHARGE FROM.—(See Discharge, Vaginal, p. 185.)

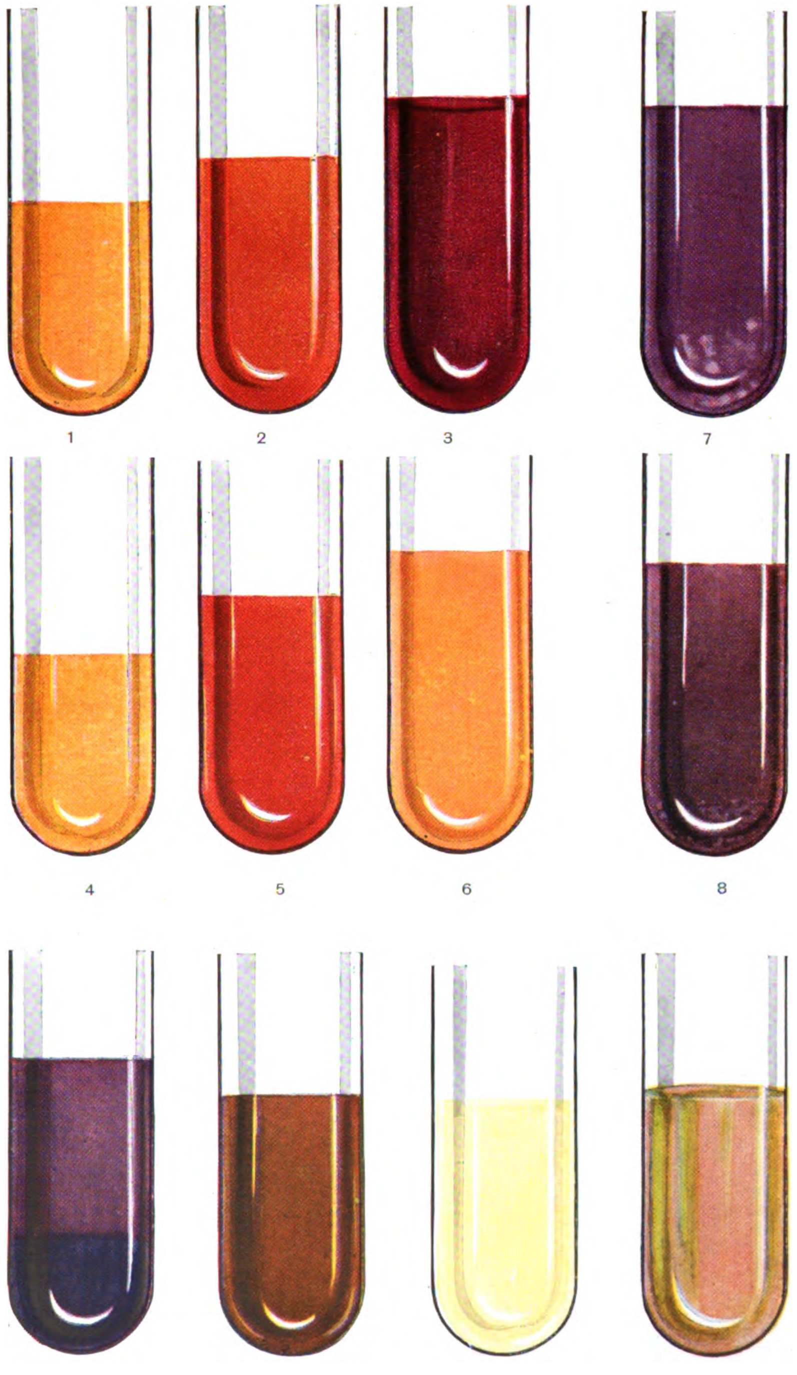
VARICOSE ABDOMINAL VEINS.—(See Veins, Varicose Abdominal.)

VARICOSE THORACIC VEINS.—(See Veins, Varicose Thoracic, p. 750.)

VEINS, VARICOSE ABDOMINAL.—The point at which distention of veins becomes varicosity is arbitrary; most conditions that produce undoubted varicosity of the veins of the abdominal wall in some cases, merely dilate them in others. When this dilatation is considerable (*Fig.* 303), it nearly always has much diagnostic significance, particularly if the direction of blood-flow is reversed. Veins, however, may seem to be dilated when they are but unduly visible owing to wasting of the subcutaneous fat; or they may, in very rare cases, be simply varicose, like veins in the leg, owing to idiosyncrasy or hereditary predisposition. In neither of these cases, however, is the blood-current in them reversed.

PLATE XXXIV

URINE TESTS



1, 2, 3. The three stages of the sodium nitroprusside test for acetone. **4, 5, 6.** The same in a urine containing no obvious acetone. (**1** and **4**, normal urine: **2** and **5**, the appearance after adding caustic soda and sodium nitroprusside; **3** and **6**, the appearance after adding acetic acid, **3** being positive, **6** negative, for acetone). **7.** Rothera's test for acetone. **8.** Ferric chloride reaction of diacetic acid. **9.** Indicanuria test. **10.** Melanuria. **11.** Diabetic urine. **12.** The fluorescent reaction of urobilin.

To test the direction of blood-flow, part of a vein should be chosen where there are no side branches, and the blood should be expressed from it by means of two fingers pressed down on the vein close together, and then drawn asunder, whilst pressure over the vein is maintained by each; when a length of the distended vein has been emptied in this way, one of the two fingers is taken off, and the time taken by the vein in refilling is noted; the procedure is repeated, the other finger being taken off this time; it is then generally easy to decide whether the vein fills from below upwards or from above downwards. Normally, the blood flows from above downwards in the veins of the lower two-thirds of the abdominal wall; when the blood-flow is from below upwards there is almost certainly obstruction to the inferior vena cava, the blood which is unable to return by it finding a collateral circulation via the superior vena cava.

Obstruction to the inferior vena cava is due to one or other of three main groups of conditions, namely:—

1. **Great general increase in the intra-abdominal tension,** owing to such conditions as: ascites; ovarian cyst; great splenic or hepatic enlargement.

2. **Thrombosis** without external obstruction.

3. **Obstruction by local compression,** especially by secondary growths in the retroperitoneal glands.

When the obstruction of the inferior vena cava is due, not to the vein itself being thrombosed or invaded by new growth, but to the *general intra-abdominal pressure* becoming so great that the vein is, so to speak, flattened out, the varicosity of the veins upon the abdominal wall is but a late symptom, and the diagnosis will be made from the cause of the great abdominal distention, generally Ascites (p. 43), or a big tumour. If there is marked varicosity of the superficial veins early in a case of ascites the probability is that both are due to malignant disease.

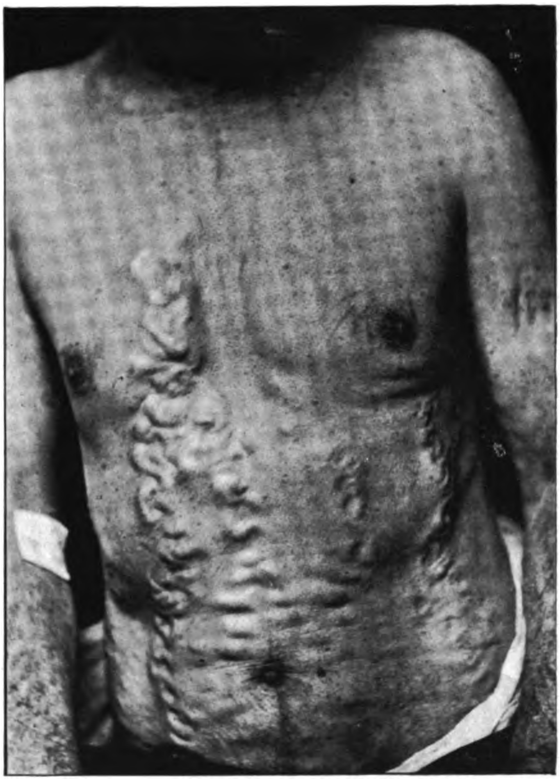


Fig. 303.—Varicose thoracic and abdominal veins in a case of syphilitic mediastinitis of some years' duration.

When the inferior vena cava is obstructed by '*simple*' *thrombosis*, the probability is that the clotting will not have started there, but will have extended to it from branches either in the legs or in the pelvis. Œdema of the legs will be a prominent symptom; and if a clear history is obtainable it may generally be ascertained that one leg became œdematous and painful before the other; when this is so it is always very suggestive of thrombosis starting in the saphenous or femoral veins, the other leg becoming affected later when the clot has spread up through the iliac veins of the one side to the inferior vena, and thence down the iliac veins of the other side. The higher the thrombus extends the higher up the back will the œdema spread; and when the renal veins have been reached, albuminuria, with tube casts, and even hæmaturia, may ensue. Ascites may also be present. Distention or varicosity of the veins of the abdominal wall will be of assistance in distinguishing such a case from one of acute or subacute nephritis, besides which there will be no œdema of the eyelids or face.

If there is no very tense distention of the abdomen; if the way the case began does not suggest thrombosis in one leg, or in the pelvis, extending upwards; and if, nevertheless, there is marked varicosity of the veins of the lower part of the abdominal wall, with the blood-flow in them reversed, so as to be from below upwards, the history being a relatively short one,—the probability is that the inferior vena cava is being obstructed by something that is in immediate contact with it. There will very likely be symmetrical œdema of the legs, and possibly albuminuria and hæmaturia. It is surprising how seldom an aortic aneurysm, or other non-malignant mass, obstructs a large vein sufficiently to produce this collateral varicosity; hence, the presumption is that such varicosity indicates *malignant disease*. It is worthy of note that carcinoma of the kidney is prone to extend into the renal veins, and thus into the inferior vena cava by a process of direct extension (*Fig.* 304)—sometimes the malignant clot reaches as far as the right auricle, and produces a pedunculated polypus in the latter. In such cases there has generally been hæmaturia or other renal symptom before evidence of inferior vena-caval obstruction arose, whereby cases of growth in the kidney invading the inferior vena cava may be distinguished from cases of secondary growth in the retroperitoneal glands, which if they produced hæmaturia at all, would do so by first obstructing the inferior vena cava, and thence involving the renal veins. In such cases there are often other symptoms pointing to primary growth in some organ whose lymphatics drain into the retroperitoneal glands; the testes and ovaries should not be overlooked in this respect.

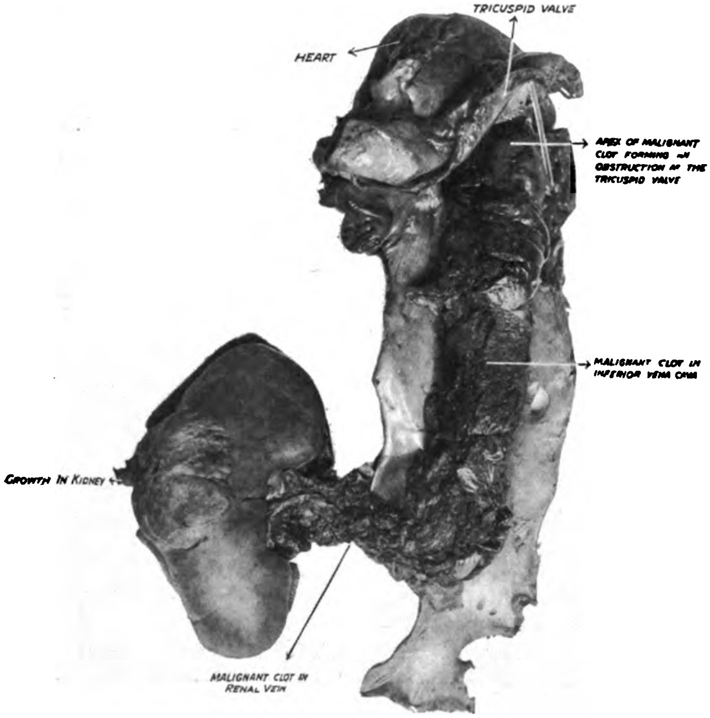

TRICUSPID VALVE
HEART
APEX OF MALIGNANT CLOT FORMING AN OBSTRUCTION OF THE TRICUSPID VALVE
MALIGNANT CLOT IN INFERIOR VENA CAVA
GROWTH IN KIDNEY
MALIGNANT CLOT IN RENAL VEIN

Fig. 304.—Renal growth extending into the inferior vena cava and right auricle and ventricle.

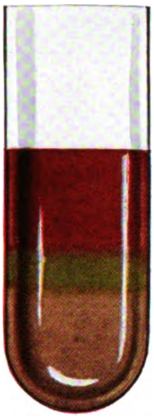
It is often said that *cirrhosis of the liver* leads to varicosity of the veins around the umbilicus—the so-called *caput medusæ*. It is a very rare condition indeed, the great majority of cases of cirrhosis of the liver causing no distention of the superficial abdominal veins until such time as the general intra-abdominal tension has been greatly increased by the tenseness of the ascites which occurs late. Not even the telangiectases that occur so commonly in men past middle age around the lower part of the chest, in a line with the attachments of the diaphragm, indicate cirrhosis; they are quite as common in cases of emphysema without cirrhosis.

In short, varicosity of the superficial abdominal veins generally indicates either thrombosis of the inferior vena cava, secondary to direct spread of thrombosis up to it from veins in the pelvis or in the leg, or else stenosis of the vena cava by secondary malignant disease. *Herbert French.*

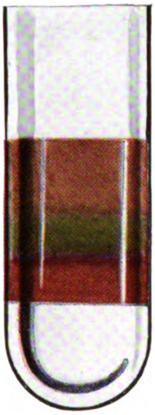
VEINS, VARICOSE THORACIC.—Much of what has been said above about varicose abdominal veins applies also to those of the thorax. The veins on the chest wall may merely be unduly visible; but if they are really distended, there is probably obstruction to one or other innominate vein or else to the superior vena cava; and the suspicion that this is so becomes a certainty if the blood current in the distended veins can be shown to be from above downwards instead of from below upwards. If the distention is bilateral, and

PLATE XXXV

URINE TESTS

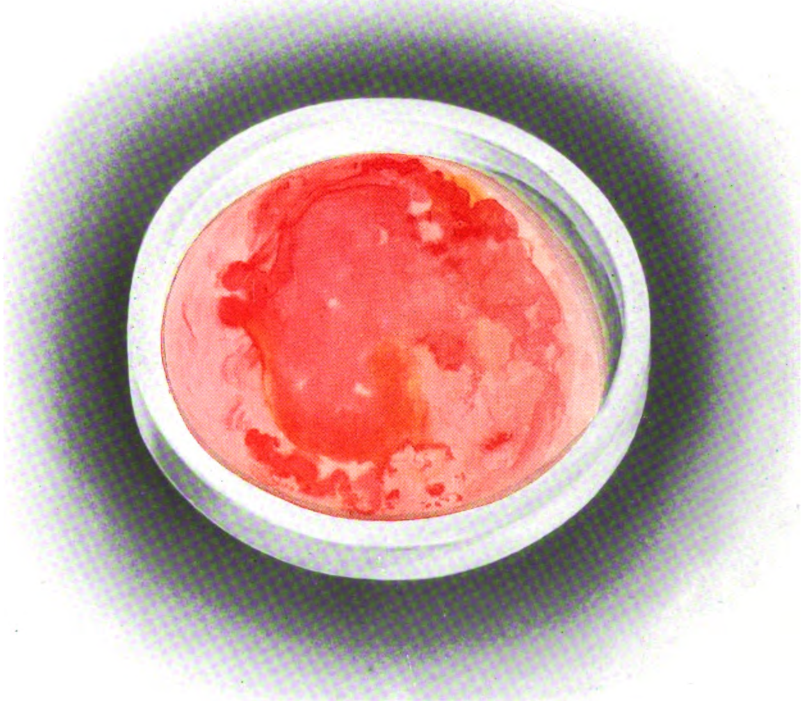


1



2

GUNSBERG'S TEST



3

1. The iodine test for bile pigment in urine. 2. Gmelin's reaction for bile pigment in urine.
3. Gunsberg's test for free HCl in gastric juice.

associated with œdema of both arms, and both sides of the neck, face and head, it is the vena cava that is obstructed; if the distention is unilateral, with œdema of the corresponding arm, but little if any of the neck or face, the obstructed vessel is probably one innominate vein. The superficial varicosity may be only slight (*Fig.* 99, p. 208), but sometimes it is extreme.

In arriving at a diagnosis of the cause of the venous obstruction, *malignant disease* within the thorax will be uppermost in one's mind—especially mediastinal sarcoma, starting in the thymus or in the lymphatic glands. It is only when the history and course are too long for primary or secondary malignant neoplasm that other causes of venous obstruction will be regarded as more likely, such as *thrombosis* extending to an innominate vein or to the superior vena cava from a whitlow, boil, or other inflammatory affection of the hand, arm, axilla, head, face, neck, shoulder, or front of chest; or *chronic fibrous mediastinitis*, sometimes tuberculous or gummatous (*Fig.* 303, p. 749) but often rheumatic in origin, and resulting from repeated attacks of pericarditis and pleurisy, with matting together, not only of the pleuræ to the diaphragm and pericardium, but also of all the structures in the superior, posterior, and anterior mediastina to one another; or, far less commonly, to *aneurysm* of the thoracic aorta or a *non-malignant mediastinal tumour*, such as a hydatid cyst (*Fig.* 137, p. 291), a dermoid cyst, or a large congenital fibroma, which may have been quiescent within the chest for many years before starting to enlarge and obstruct structures in its neighbourhood; the latter conditions, except aneurysm, are rarities, and although an aortic aneurysm does sometimes obstruct the superior vena cava sufficiently to cause distention or varicosity of the veins upon the chest wall, such varicosity is so much more marked in a case of intrathoracic malignant disease that one may say that when the diagnosis lies between neoplasm and aneurysm the presence of marked distention of the veins of the chest-wall indicates the former rather than the latter, though the converse of this is not true.

Herbert French.

VERTIGO, popularly known as dizziness or giddiness, depends upon a disturbance of the sense of equilibrium. In slight cases the trouble is perceptible to the sufferer either as the apparent movement of motionless objects (objective vertigo), or his own body may appear to be moving in relation to surrounding fixed structures (subjective vertigo). In more severe cases it may lead to reeling or staggering, and unless the patient can grasp some fixed support he may fall.

The equilibrium of the body is maintained by the co-ordinated action of various groups of muscles, and the nervous mechanism for this co-ordination is situated in the cerebellum. Afferent impulses are brought to the cerebellar centres from the muscles, skin, joints, eyes, and the semicircular canals. The cerebellum is also connected with the motor centres of the cerebral hemisphere, and thus the requisite contraction of the necessary muscles is ensured. Disturbances of equilibrium may therefore be the result of a lesion in the cerebellum itself or in one of the afferent tracts. True vertigo depends mainly upon interference with the afferent impulses from the semicircular canals or from the eyes, and it is often accompanied by nystagmus during attacks.

Occasionally vertigo may be the result of altered cutaneous impulses due to such causes as anæsthesia of the soles of the feet. Some people experience a slight feeling of giddiness on stepping on to some soft material such as turf or india-rubber pavement after walking on a hard road. Unusual cutaneous impulses are the probable explanation.

Interference with the afferent impulses from the muscles themselves, such as occurs in tabes dorsalis, leads to reeling and staggering rather than to true vertigo. The ocular impressions correct the false sensations from the muscles, and hence loss of equilibrium is more likely to occur in the dark, or when these impressions are cut off by covering the patient's eyes.

Vertigo is sometimes divided into 'general' vertigo and 'special' vertigo. In the latter, objects appear to move, or the patient tends to fall in a definite direction. Special vertigo points to a lesion of a particular semicircular canal. Thus, if there is a lesion of the external semicircular canal, objects appear to move in a horizontal plane, and the patient tends to fall towards the affected side. When the superior canal is the source of the trouble, objects rotate in a vertical plane, and the patient falls forward. Temporary vertigo, even of a very severe nature, may be produced readily in a healthy individual by prolonged movements of rotation or of swinging. In this case the cause of the giddiness is probably unequal pressure in the endolymph in the different semicircular canals. The dizziness with which many people are affected when near the edge of a high cliff is most likely ocular in origin, and depends upon the sudden cessation of visual impulses from near objects. Some persons are exceedingly susceptible to alterations in these impressions, and travelling by train or the movement of a boat may be sufficient to cause considerable dizziness.

Vertigo may also be of *toxic origin*. Alcohol and tobacco are familiar examples ; the dizziness associated with ptomaine poisoning, and in some cases of gastric disturbance, is also probably of this nature.

Vertigo is not infrequently of cerebral origin, either with or without some gross lesion. Thus it occurs in *migraine*, and is also a frequent *aura of an epileptic fit*. Vertigo may depend upon alterations in the blood-pressure, and this is the probable explanation of its occurrence in *arteriosclerosis* and *nephritis*, where the blood-pressure is increased. It may also occur in cases where the blood-pressure is diminished, as in *Addison's disease*, some *anæmic states*, and during *convalescence* from any prolonged illness. This may be due to defective nutrition of the central nuclei. Attacks of vertigo occurring in elderly people with atheromatous arteries or suffering from *chronic nephritis* or *arteriosclerosis* must always be regarded as of serious import, since they may be the precursor of cerebral hæmorrhage or thrombosis. Severe vertigo may be one of the symptoms of a *cerebral tumour*. It is more likely to be present when the growth is in the cerebellum, especially if the middle lobe is involved. A tumour involving the auditory nerve in its intracranial course will also give rise to this symptom. A *cerebral* or *cerebellar abscess* may also cause vertigo, but in this case there may also be suppuration in the middle ear, and the giddiness may be of labyrinthine origin. Optic neuritis should be looked for in every case of vertigo.

Vertigo may be of *ocular origin*. It is especially likely to occur when there is some lesion of the nerves or muscles leading to diplopia.

Laryngeal vertigo is a very rare condition ; spasm of the glottis is accompanied by severe giddiness which causes the patient to fall down, and he may lose consciousness for a few seconds. Complete recovery ensues in a short time, but the attacks are likely to recur. This trouble may be of an epileptic nature.

Vertigo is, however, most commonly of aural origin, and is a frequent symptom of diseases of the ear, especially of the internal ear or labyrinth. In its most intense form it is one of the symptoms of '*Ménière's disease*.' Its onset is then sudden, and so severe that the patient falls to the ground and even loses consciousness. Associated with the giddiness are severe tinnitus, and unilateral or bilateral deafness, while nausea, vomiting, and pallor of the face are frequently present. The vertigo passes off after a few hours or days, but impairment of hearing and tinnitus persist. The attacks tend to recur. Ménière's disease is probably caused by a sudden increase in pressure in the endolymph, and, in some cases at any rate, appears to be due to hæmorrhage into the semicircular canals. In some cases an embolism may be the cause. True Ménière's disease is very rare ; but the occurrence of the symptoms, viz., vertigo, deafness, and tinnitus, in a less acute and sudden form, is by no means uncommon. They may be present without any obvious lesion of the middle or external ear, though sometimes there is some abnormality in one of these portions of the auditory apparatus. Vertigo may be traumatic in origin, e.g., after a fracture of the base of the skull.

Syphilitic disease of the internal ear may produce symptoms closely resembling those of Ménière's disease, in that giddiness, tinnitus, and labyrinthine deafness are associated, the onset being quite sudden. Vertigo is, however, occasionally absent. The trouble is usually unilateral, and may occur in the secondary or tertiary stages. The diagnosis will depend on the history of syphilis or other evidences of the disease. Similar symptoms may occur in congenital syphilis, usually between the ages of ten and fifteen years, though occasionally much later. Eustachian-tube obstruction is usually present also, but treatment of this fails to improve the hearing, and other characteristic troubles, especially interstitial keratitis, may be found. Aural vertigo may also be associated with some lesion of the external or middle ear. Thus in the former case there may be a *foreign body*, or even a plug of *impacted cerumen*, as the exciting cause.

Vertigo is not infrequently present in chronic middle-ear suppuration. This may be due to labyrinthine inflammation or irritation, but a definite labyrinthine lesion is not necessarily present. In many cases the giddiness is caused by pressure on the stapes or the fenestra rotunda. Some patients with a perforation of the tympanic membrane become giddy whenever the ear is syringed. Occasionally the use of unduly hot or cold lotions produces this trouble. It is usually the result of pressure on the stapes, though occasionally the vertigo may have the special character associated with a lesion of the external semicircular canal (*vide supra*). When this is the case there is probably some erosion of the bony external canal. Similar giddiness may occur from a like cause on syringing the ear after a radical mastoid operation.

Vertigo may occur in *otosclerosis*, though it is not common in this disease, and is always of less importance than the DEAFNESS (p. 163) and TINNITUS (p. 722). Gout and gouty dyspepsia, with or without evidence of arteriosclerosis, may also be the cause of attacks of vertigo.

When a patient complains of vertigo, the ears should always be examined carefully. The hearing should be tested, and if the cause of the trouble is in the labyrinth, the deafness will have the characters of nerve deafness (p. 166). The onset of the trouble must be investigated carefully, and any associated symptoms ascertained. The eyes and ocular muscles must be examined, and if nystagmus is present the character of the movements should be observed. A general examination of the patient should also be made for some general constitutional cause such as gout, albuminuria, or arteriosclerosis. *Philip Turner.*

VESICLES.—One of the primary lesions, the vesicle may be defined as a circumscribed epidermal elevation varying in size from a pin's head to a small pea, and containing serous fluid, which may become sero-purulent or be mixed with blood. Serous elevations larger than a small pea are classified as BULLÆ (p. 96). To bullæ, therefore, vesicles bear the same relation as papules bear to tubercles. They differ from bullæ, however, not only in size, but in their mode of formation. They are always the result of an inflammatory process, whereas in the case of bullæ there is a veritable cleavage of the epidermis. Vesicles, again, often contain a number of chambers, at any rate in the beginning, whereas bullæ are from the outset unilocular. They may originate as vesicles, or may develop from papules. Vesiculation may be either parenchymatous or interstitial. In the one case, as in varicella, the plasma accumulates *within* the Malpighian cells, and the unicellular vesicles which are thus formed run into each other. In the other case, as in eczema, the plasma accumulates *between* the Malpighian cells.

In shape, vesicles are usually rounded, conical, or acuminate ; but they may tend to the oblong form, as in scabies, or they may be both oblong and irregular, as in dermatitis herpetiformis. The larger ones are occasionally umbilicated, as in variola, and instead of being tense, as is usual with vesicles, may be flaccid, as in herpes and dermatitis herpetiformis. At first the liquid they contain consists almost invariably of pure plasma, and is quite clear, or with the faintest tinge of yellow ; but exceptionally the fluid is from the beginning mixed with blood. After a time the clear fluid becomes turbid. In some conditions, as in varicella and miliaria, they remain discrete and few in number ; but usually, as in herpes, eczema, and dermatitis herpetiformis, there is a plentiful crop of them, forming groups or closely-set clusters. As a rule, they are of short duration : either they rupture and crust over, as in eczema, or they dry up and a crust is formed, as is usual in herpes ; or they enlarge into blebs, as frequently occurs in dermatitis herpetiformis ; or they are transformed into pustules, as in variola. On mucous membranes and the lips, and in folds of the skin, they break more quickly than in other situations, and leave excoriations. Since, as already stated, vesicles are the result of a more or less inflammatory process, they usually give rise to much burning and itching, though in some conditions, as in hidrocystoma, these symptoms are absent.

The most distinctively vesicular affections are herpes simplex and herpes zoster. In *simple herpes* the face and the genital organs are affected chiefly. The characteristic lesion is a cluster of transparent vesicles varying in number from two or three to twenty or more, seated on an erythematous patch, and surrounded by a narrow red zone. First, a slightly red spot appears on the skin ; effusion quickly takes place under the epidermis, and vesicles are formed ; these become opaque—sometimes purulent—shrivel up, and form yellowish-brown crusts, which after a few days become detached, usually leaving no scar, but a brownish stain that slowly fades and disappears. These four stages in the evolution of the lesion are styled the congestive, vesicating, desiccating, and macular stages. On mucous membranes the lesion runs a rather different course. Here the vesicles are quickly reduced to a whitish pulp, which presents the appearance of a false membrane. When this becomes detached, it reveals a number of roundish excoriations, either scattered about irregularly or running into each other and forming largish ulcers. The favourite situations of the vesicles in herpes genitalis are, in men, the prepuce, especially its inner surface, the meatus, the sulcus, and the glans ; in women, the labia and the cervix. In men, the vesicles are usually discrete, and the patient complains only of the itching and burning ; but if they are neglected, or irritated by the application of caustics, there may be severe and extensive ulceration, with swelling of the inguinal glands. In women, the vesicles tend to become confluent, and the perineum, the inside of the thighs, and the mons veneris may be invaded. There may be a great deal of swelling, excoriation, and discharge, with intense itching and burning, and, as in men, there may be enlargement of the neighbouring glands. The vesicles of genital herpes are too characteristic to be mistaken if they are seen before their real significance is obscured by ulceration. If, however, the ulceration is considerable, and especially if there is much suppuration, the herpes may be mistaken for *chancroids*. Generally, however, soft sores are multiple, have a fouler base, excavate more deeply, and the healing process is much slower. Soft sores, further, are flattened at the base, secrete very little liquid, and are auto-inoculable. In some cases there may be doubt as between herpes genitalis and true *chancre*, especially as, according to Fournier, a chancre not infrequently develops in the midst of a premonitory eruption of herpes. The points of differentiation are the absence, in herpes, of induration, the less considerable and more transitory gland-enlargement, the multiplicity, irregular form, and small size of the ulcers, and the intense burning and itching.

In the crusted stage, facial herpes may resemble *impetigo*, but the rapid course it runs, its limited distribution, the facts that it is not auto-inoculable, and that in impetigo the lips are seldom attacked, should suffice to obviate the confusion. The points which distinguish herpes facialis from vesicular eczema are touched upon below.

In *herpes zoster* (zona, shingles), clusters of vesicles seated on an erythematous base appear in the region of skin distribution of one or more of the posterior spinal nerve roots, preceded or accompanied by neuralgic pain and tenderness in the part. The erythematous patches, more or less oval, with the long axis parallel to the underlying nerve, come out in crops, the number of lesions varying from two or three to twenty or thirty. Soon the surface of the patches is studded with papules, which are quickly transformed into vesicles, from ten to twenty on each patch, sometimes discrete, sometimes running into each other to form bullæ. An important diagnostic feature of the eruption is, that in the great majority of cases it is unilateral, and appears much more frequently on the right side than on the left. In rare cases, however, it forms a complete girdle round the body. The usual limitation to one side of the body, the distribution in one or more nervous territories, and the preceding or accompanying neuralgia, usually suffice to distinguish herpes zoster from erythema multiforme and from dermatitis herpetiformis. Another important point in diagnosis is the history, for zoster is not a recurring disease. These various characters serve to distinguish it also from herpes simplex and herpes genitalis. The neuralgic pain may be mistaken at first for pleurisy, but the course the affection runs soon clears up the confusion. I should add that in herpes zoster the forehead, the conjunctiva, and the eyeball are frequently attacked, and sometimes the mouth, especially the tongue. In rare cases the lesions on the tongue are not associated with an eruption on the lips or the palate.

Although the vesicular stage is not, as some authorities consider, a necessary phase in the evolution of *eczema*, the vesicle is undoubtedly the most constant of all the primary lesions met with in that condition. Usually, following sensations of itching and burning, an erythematous blush appears, which is soon studded with numerous tiny vesicles. These grow larger and often coalesce, but they soon rupture or are broken by scratching, and a clear fluid exudes, the 'weeping' continuing as later vesicles break. In mild cases, the inflammation subsides gradually, and as the discharge ceases, scales or crusts are formed ; but much more frequently fresh crops of vesicles start up around the edge of the earlier patches, while new centres are formed in other parts, until nearly the whole cutaneous surface may be involved. In some cases papules are the predominant feature, in others erythematous lesions; in yet others pustules, and in extensive cases, the several kinds of lesions may be all present simultaneously. From herpes in general, eczema is distinguished by the characteristic exudation, by the crowds of tiny vesicles, which coalesce without forming distinct groups, by the slower evolution of the disease, and by the fact that as a rule there is some inflammatory thickening. From herpes zoster it is differentiated by the peculiar distribution of the vesicles in that affection (see above).

Acute vesicular dermatitis is precisely similar to acute eczema in its actual lesions, but differs from it in that a definite irritant cause exists and it does not recur, as does eczema, spontaneously, but only if the external cause is re-applied. The effects of certain plants, notably *primula obconica* and *rhus toxicodendron*, are familiar in this respect; so also is the vesicular eruption produced in some individuals by sugar (grocer's dermatitis), by the use of certain soaps (soap dermatitis), by the sawdust of satin-wood, by the use of lime in making mortar, by the hairs of certain caterpillars, and by various applications and lotions (application dermatitis), including tincture of arnica montana, mesotan, essence of vanilla; and of course all the well-known vesicating drugs such as cantharides, croton oil, capsicum, mineral acids, caustic alkalies, strong iodine or turpentine, and so on. The diagnosis is afforded by a knowledge of exposure to any of these, and in obscure cases is sometimes suggested by the fact that exposed parts only are affected, or that the patient is ill only when living in certain places or doing certain work.

Doubt can seldom arise as between eczema and *dermatitis herpetiformis*. It is true that the earliest and perhaps the most characteristic lesion of the latter disease is a vesicular eruption, appearing on an erythematous base; but the disposition of the vesicles in herpetiform groups should prevent confusion between the two conditions. The vesicles soon dry up and form scabs, but later they tend to coalesce into bullæ, which scarcely ever burst spontaneously but, as their contents thicken, slowly shrink, and finally, if left to themselves, shrivel up to a thick brown scab. The 'weeping' of eczema is therefore absent in dermatitis herpetiformis, of which, further, multiformity is a more pronounced feature—erythematous, vesicular, pustular, papular, and urticarial elements being mingled in all stages of evolution. Eosinophilia is more pronounced with dermatitis herpetiformis than it is with eczema (p. 99).

The vesicles of *impetigo contagiosa* are distinguishable from those of eczema by the larger size and discrete character of the former, and by their tendency to dry and form yellowish crusts without breaking, or as soon as they have broken. Even when the lesions run together and large crusts are formed, there will be discrete vesicles and papules which will point to the true nature of the affection.

Miliaria rubra may sometimes resemble the vesicular stage of eczema, but here again the lesions, though numerous, remain discrete: they do not run together to form patches, they do not rupture, and there is no 'weeping.' Miliaria of all forms is a very transitory affection, and instead of the intense itching of eczema, the patient describes his sensations as those rather of pricking and tingling. In the form of miliaria known as hidrocystoma, or dysidrosis of the face, small vesicles like those of miliaria appear on the face, but they are so grouped as to form patches, which show no tendency to spontaneous involution.

Scabies is another affection in which the vesicles, like those of impetigo and of miliaria, are discrete. Sometimes the lesions, usually consisting of papules and pustules as well as of vesicles, take on an eczematous character, but they are not localized as are those of eczema, and instead of being small, acuminated, or circular, they tend to be linear. In uncleanly persons, the burrows between the fingers and elsewhere which mark off scabies from all other affections, can hardly be overlooked. When they cannot be found, either because they have not yet been formed or because they have been laid open by scratching, the diagnosis of scabies must rest upon the irregularity of the lesions—vesicles, bullæ, and pustules being mingled with the marks of the finger-nails and the results of secondary inoculations—and upon the distribution, the parts most affected being those where the skin is least thick, namely, the webs between the fingers and toes, the front of the wrist, inside the umbilicus, on the lower abdomen, the genitalia, the nipples in women, and the axillary folds. The face nearly always escapes, except in infants in arms.

Another vesicular condition in which the hands are specially attacked is *cheiropom-*

pholyx, in which numerous minute vesicles deeply imbedded in the skin, and showing through the epidermis like boiled sago-grains, are distributed symmetrically on the extremities—always on the palms and fingers, and frequently also on the soles and toes. The general features of the affection—the limitation of the vesicles to the hands and feet and their proneness to unite and form bullæ which dry up, the tendency to recovery followed by repeated recurrence, and the constant association of the eruption with the summer season—are sufficiently distinctive, and the diagnosis is seldom in doubt. In some slight cases there is a general resemblance to certain subacute and limited cases of eczema in which the lesions may present the sago-grain aspect; but instead of rupturing and 'weeping,' the vesicles in cheiropompholyx tend to run together into bullæ, which shrink and crust over. This formation of bullæ by coalescence of vesicles differentiates the condition also from pemphigus.

In *erythema multiforme* the vesicle can seldom be difficult of interpretation, even in *erythema iris*, or as it is also styled, *erythema vesiculosum*. In one form of this affection a small red spot appears, upon which is formed a vesicle that is quickly surrounded by a zone of redness. When the central vesicle dries up it leaves a small scab, and a ring of secondary vesicles soon appears on the red zone. On the separation of the central scab, the skin beneath has a blue, congested appearance. The whole process may be repeated time after time until the concentric rings of vesicles and reddened skin suggest comparison with a target. In the form of erythema iris, which sometimes is infelicitously called *herpes iris*, a large central bulla is encircled by vesicles of considerable size. Outside the first ring of vesicles another circle may develop, and outside the second, sometimes a third. The symptoms of vesicular erythema multiforme are so characteristic that the affection can hardly be mistaken for anything else.

Lichen planus is so characteristically a papular affection that the absence of vesicles is one of the points which distinguish it from eczema. In some cases, however—very rarely in adults, and rather less infrequently in children—vesicles appear, but never so as to confuse the diagnosis. In *lichen urticatus* a vesicle appears on the summit of the small wheal, and the condition may offer some resemblance to eczema; but the individual lesions do not tend to run together nor to spread centrifugally, as in eczema, and the itching is usually more intense.

The vesicular form of *secondary syphilis* is so rare that by some authorities its existence is not recognized, and Stelwagon, who has never met with a case, justly points to the possibility of its being due, at any rate occasionally, to drug idiosyncrasy. The vesicles are reported as occurring in several forms: they may be minute, eczematoid, disseminated and grouped, or larger, irregularly scattered, or disposed in herpetiform groups; and cases have been reported which simulate herpes zoster. The vesicles in syphilis are usually associated with papules, and they have a papular base, the disappearance of which leaves a long-persisting dark stain. The papular base and the slow evolution are important diagnostic points; and usually other signs of syphilis will be present.

In *lymphangioma circumscriptum*, even more than in vesicular syphilis, the significance of the vesicles can hardly be missed. In circumscribed areas of the skin there is an eruption of clusters of small, deep-seated, thick-walled vesicles, straw-coloured, sometimes marked with red striæ, and filled with a clear alkaline fluid which contains a few lymph-corpuscles. The affection, which consists in the over-growth and dilatation of lymph-vessels and the formation of new ones, is probably congenital, though not generally noticed until early childhood.

Varicella—to come to the eruptive fevers—is an essentially vesicular affection: only occasionally do the vesicles develop into pustules. They are usually preceded by reddish spots of slight elevation, and the commonest situations are the face, chest, shoulders, back, and scalp, but they may also be found on the mucous membrane of the palate, mouth, or lips. As a rule the rash comes out within twenty-four hours. Often there is but trifling systemic disturbance. In the infrequent cases in which the varicellar eruption becomes pustular, it may possibly be confused with a pustular syphilide; but in the syphilide the lesions are pustular from the outset, or develop out of papules, and although the lesions of varicella start as papules, these are almost invariably small and evanescent. The absence of itching in syphilis is another point of difference. In exceptional cases of strophulus the vesicle on the summit may develop until it becomes visible macroscopically, and it may then be confused with varicella; but the cases are so rare as to be negligible.

It is with *small-pox* that chicken-pox is most often confused. In small-pox the vesicles are usually multilocular; in chicken-pox they are usually unilocular. In small-pox they are frequently umbilicated; in chicken-pox they are never umbilicated, and seldom even dimpled. The differences between the two exanthems in respect of the rash and the lesions generally, as summarized by Ricketts in "The Diagnosis of Small-pox," are these: In variola, the rash is most abundant on the face and limbs, and least abundant on the abdomen and chest (*Fig.* 305); in varicella, the abdomen and chest are covered as thickly as the face, if not more thickly. In variola, the rash is much more abundant on the back than on the abdomen; in varicella, the abdomen and the back receive equal attention. In variola, the rash is more abundant on the shoulders than across the loins, and on the chest than on the abdomen; in varicella, the distribution as between these parts is indifferent. In variola, the rash favours the limbs and is distributed centrifugally; in varicella it tends to avoid the limbs, and when it invades them is centripetal. In variola the rash, unlike that in varicella, favours prominences and surfaces exposed to irritation, and tends to avoid protected surfaces and depressions. As to the lesions generally, in variola they are deep-seated and have an infiltrated base; are generally circular in outline and homogeneous in character; whereas in varicella they are superficial and have no infiltrated base, are frequently irregular in outline, or else oval or elongated, and are not, as a rule, homogeneous.

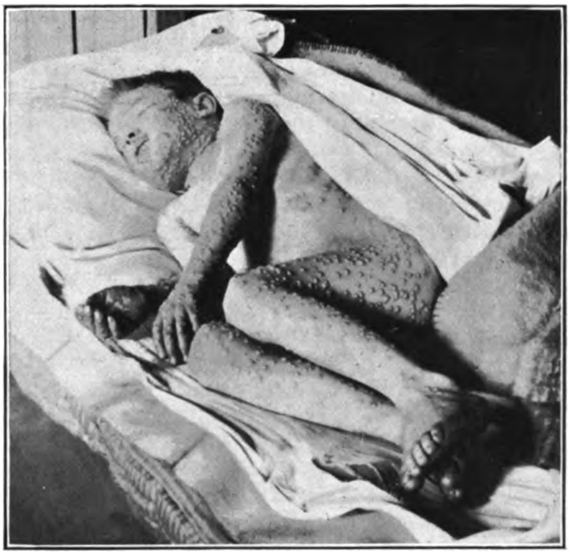


Fig. 305.—Small-pox: showing distribution of the eruption, particularly on the face and limbs rather than on the neck and trunk.

(*From a photograph by Dr. D. S. Davies.*)

Vaccinal eruptions may consist (1) of tiny vesicles or small superficial papules, or of a combination of those elements; or (2) of a small papule with a vesicular or pustular head. In the second case the eruption may simulate modified small-pox, but almost always the vaccinal lesions are more superficial than those of the mildest cases of small-pox, and show a preference for the trunk.

In eruptions due to the use of *bromides*, *iodides*, and other *drugs*, the vesicle is but one of the elements, and the nature of the affection is usually indicated with sufficient distinctness by the history of the case, the remission that occurs when the drug is withheld, and the recurrence that takes place when its administration is resumed. Vesicles that follow the *bites* or *stings* of *gnats*, *mosquitoes*, etc., are always easily recognizable from the history, and from the central punctum to be seen in the lesions; nor can there be any doubt as to the significance of those due to such accidental causes as *frostbite* and pressure from *splints*. *Malcolm Morris.*

VISION, DEFECTS OF.—This subject may be considered in the following order:—(*I*) *Normal vision*, (*II*) *Amblyopia*, (*III*) *Partial blindness*, (*IV*) *Complete blindness*, (*V*) *Colour blindness*, (*VI*) *Abnormal sensations of size*, (*VII*) *Day- and Night-blindness.*

NORMAL VISION.

1. **Visual Acuity.**—The act of vision comprises the perception of form, colour, and brightness; and, in vision with two eyes, the perception of space and distance. These faculties are possessed by all parts of the retina, though in varying degrees, and they are of

varying importance. It is necessary to distinguish between peripheral and central vision; or, in other words, between merely seeing a thing and looking at it. An object is seen by any portion of the retina that has visual perception; but an object is only looked at when its image falls upon a particular portion of the retina, the yellow spot, which is situated at the posterior pole of the globe on the outer side of the optic disc. The act of so directing the eye that the image of a given object shall fall upon the yellow spot, is termed 'fixation.' The vision obtained by the fixation of the eye is termed 'central vision,' and owing to the anatomical structure of the retina at the yellow spot, the vision here is the most acute of which the eye is capable, though its area is very limited. In the normal eye, central vision is capable of distinguishing two points or parallel lines, which are separated by a space which subtends an angle of 1′—approximately the diameter of a sixpenny-piece at 200 feet—and it is on this basis that ordinary test-types are constructed. Central vision, however, though acute, is very limited in extent, and it is estimated that the field of acute vision is only about the size of the thumb-nail held at arm's length, all vision outside this area being comparatively blurred and indistinct. This limitation of the field of acute central vision is barely appreciated under ordinary circumstances, owing to the rapidity with which the retina receives consecutive visual impressions, and the constant movements of the eyes. Compared with the visual acuity of the central portion of the field of vision, peripheral vision is relatively poor, though it is of extreme value in a different way. To appreciate the importance of peripheral vision it is only necessary to try to walk about looking through a roll of music; though central vision is unimpaired, and the smallest object can be seen distinctly, locomotion is almost impossible, owing to the inability to see where one is going or to ascertain one's position in relation to surrounding objects, the peripheral portion of the field of vision being responsible for the automatic appreciation of these. On the other hand, a person from some cause deprived of central vision can see to get about quite well, and has useful vision for many purposes, though he is unable to read or write, recognize people when looking directly at them, or do any work in which fine vision is required.

2. **Colour Vision.**—A person with normal colour vision can recognize six or seven distinct colours in the solar spectrum, and is able to appreciate many hundreds of varieties of colour caused by mixtures of them, and the colour perception of the normal person is most acute in the central portion of the field; but the field of vision for colours under equal illumination has by no means the same boundaries as the field of vision for white. The fields of vision for all colours are smaller than that for white, and the fields for red, green, and blue vary in extent among themselves. The field for blue is the largest, for red is next in point of size, and the field of vision for green is the smallest of all, being roughly only about half to a third the diameter of the field for white.

3. **Brightness Perception.**—The central and peripheral portions of the field of vision vary very much in their perception of brightness. In ordinary illumination the central portion of the field is the most efficient, but in a very weak illumination the peripheral portion has a higher efficiency than the central part: in other words, there is in very dim lights a relative central scotoma or loss of vision. This fact has long been known to astronomers, who have found that in counting stars of low magnitudes, vision is much better if the particular constellation or group of stars is not looked at directly, the Pleiades being a well-known example; more of these stars can be counted when the vision is directed to a point a little above or below them or to one side, whereas direct vision is comparatively dim and confused; and the same holds good of vision for any object in a dim light. Walking along a country road on a dark night, it will be found that a foot-path or track can be seen more easily if the gaze is directed forwards and not at the ground itself. These facts concerning vision may be correlated with the actual anatomical structure of the retina itself. It is found that in the region of the yellow spot—the area of the retina endowed with acute vision—the cones are very numerous, with few rods; towards the periphery of the retina the cones become fewer and the rods more numerous. It is now generally held that the function of the cones is to work in light of considerable brilliance, that they are capable of extremely acute vision for small objects, and are also concerned in the perception of colour. The rods, on the other hand, have no perception of colour; their perception of form is poor compared with that of the cones; but in very weak lights their visual acuity is greater than that of the cones. In support of this theory may be mentioned the fact that the retinæ of nocturnal animals are more fully provided with rods than cones.

AMBLYOPIA.

Amblyopia is the term applied to defective vision in which there is little or no evidence of any ocular condition which might account for the visual defect. It is not, therefore, employed where there is any obvious intra-ocular or intracranial lesion. The commoner forms of amblyopia are (1) Amblyopia ex anopsia ; (2) Uræmic ; (3) With nystagmus ; (4) Due to alcohol, tobacco, lead, quinine, organic forms of arsenic such as atoxyl ; (5) Migraine ; (6) Cortical or crossed amblyopia ; (7) With hysteria.

Amblyopia ex Anopsia is usually associated with a squinting eye, which may or may not have been put straight by operation. It is still open to discussion whether the amblyopia in such conditions is due to the loss of use by reason of the squint, or whether the squint is due to the visual defect ; but the fact remains, that in many squinting eyes the visual acuity is very much below normal, though objectively the eye exhibits no abnormal symptoms.

Uræmic Amblyopia, or **Amaurosis,** may be recognized by its association with renal disease, whether complicated by pregnancy or not. It is characterized by its comparatively sudden onset, vision failing either partially or completely within a few hours. The failure of vision may be accompanied by well-marked retinitis in both eyes ; but in many cases this is absent and the discs appear quite normal. The failure of vision lasts for from thirty-six to forty-eight hours, and then slowly disappears, the whole attack lasting, as a rule, under a week. In the majority of cases, unless there is some definite injury to the optic nerve or retina as the result of retinitis, the vision recovers entirely. In some cases of uræmia the blindness is complete.

Amblyopia with Nystagmus is usually associated with evidence of visual affections in very early life. At birth, fixation is not developed, and the higher visual acuity is only acquired after the first few months of infant life. Any affection of the eyes, therefore, that obscures the vision during the earlier weeks or months of life, prevents the due development of central vision and leads to a permanent amblyopia, as a rule associated with nystagmus. Such affections are ophthalmia neonatorum, which leaves more or less corneal opacity ; perforation of the cornea with anterior polar cataract as the result of this ophthalmia ; congenital defects, such as a persistent hyaloid artery or macular coloboma ; and any retinitis or choroiditis affecting the region of the yellow spot.

The **Amblyopia due to Lead, Alcohol, Tobacco, Quinine,** or **Atoxyl,** is usually described as a toxic amblyopia, and the symptoms are somewhat similar in all the varieties. In tobacco amblyopia there is a central loss of vision for colours, green only in the earlier stages, subsequently green and red, and in extreme cases even a central scotoma for white ; total blindness is practically unknown. The patient also states that he sees better in a dull than in a bright light, and that he is incapable of reading or writing, or distinguishing silver from gold coins. The ocular signs are usually very slight, being limited to some redness and blurring of the optic disc in early cases, or pallor of the outer side of the disc in later stages. Alcoholic amblyopia resembles in most of its symptoms the amblyopia due to tobacco, though vision for red is usually lost before vision for green. Amblyopia due to lead or atoxyl is rare, but it is also usually characterized by a central scotoma associated with some slight optic neuritis or atrophy. In quinine amblyopia the retinal vessels are extremely constricted, the disc is pale, and the field of vision is diminished peripherally.

The **Amblyopia of Migraine** is usually transitory, and may occur either in the form of a central scotoma, hemianopia, or monocular blindness. It is more rarely seen in the form of a quadrant hemianopia or a ring scotoma. In all cases the diagnosis is comparatively easy, as the amblyopia seldom lasts more than a few minutes, and is followed later by the characteristic headache and sickness of migraine.

Amblyopia has also been described as due to **disease of the Visual Cortex,** and rare cases have been described of a **Crossed Amblyopia** or defect of vision in one eye due to disease of the visual cortex of the other. The loss of vision is, however, usually associated with some slight defect of vision in the opposite eye, and hemianopia is much more commonly the symptom of disease of the visual cortex.

Hysterical Amblyopia may, like other hysterical affections, take various forms, such as loss of visual acuity, a loss of colour vision, or diminution in the visual field. The characteristic form of the visual field in hysteria is either a spiral contraction or an extreme concentric limitation. The symptoms, however, vary very much at different examinations, a point of much importance in diagnosis. In certain cases there may be a functional loss of vision in one or both eyes, which can be recognized as hysterical by the employment of Snellen's coloured types or some other device for deceiving the patient.

PARTIAL BLINDNESS.

This may be (1) *Definite*, or (2) *Indefinite*.

1. **Definite**: (*a*) *Hemianopia*; (*b*) *Central scotoma*; (*c*) *Peripheral constriction.*

(*a*). *Hemianopia* (see HEMIANOPSIA, p. 300).

(*b*). *Central Scotoma.*—A scotoma is a local defect in the visual field, and, from its position, may be either central or peripheral; it may also be negative or positive. A negative scotoma is one where the defect of vision exists, but where the patient notices nothing abnormal. The best example of a negative scotoma is the blind spot in the field of vision caused by the entrance of the optic nerve. This area is blind, but the individual is not conscious of any visual defect. Scotomata of this character exist where there is some injury of the visual layers of the retina itself, or of the optic nerve or tract.

A positive scotoma is one in which the visual defect is noticed as a black or coloured spot or cloud which obscures the vision in some part of the visual field. Such positive scotomata are due to lesions of the retina, such as hæmorrhages or patches of pigment which do not destroy the visual layers. Vision still remains; but it is obscured by some unusual opacity in the retina itself or in the adjacent portion of the vitreous.

Scotomata frequently exist in the peripheral portion of the field of vision without being noticed, as they are of little importance in direct vision, and are not discovered unless looked for carefully. A central scotoma, on the other hand, is noticed at once, however minute, because it affects direct vision and produces a considerable defect in the visual acuity. A central scotoma may be either relative or absolute, and may exist for colours only or for objects. Central loss of vision for colours, more particularly red and green, is associated with *tobacco* and *alcohol poisoning.* The colours cannot be recognized in small objects when looked at directly, though a red or green object in the peripheral portion of the field of vision will be recognized as such. This scotoma is associated with greater or less diminution of the general visual acuity, and vision in such cases is generally better in a dull than in a bright light.

Absolute central scotomata are met with in *disseminated sclerosis*, in certain forms of *hereditary optic atrophy*, and may persist after the acute affection of the optic nerve known as *retrobulbar neuritis*, or in rare cases after severe attacks of *migraine.* In nearly 25 per cent of all cases of disseminated sclerosis a central scotoma exists, and the diagnosis in such a case will be confirmed by its association with the general symptoms of the disease and with other ocular symptoms, such as optic atrophy, paralysis of accommodation, paralysis of the extrinsic ocular movements, or nystagmus. There is usually some pallor of the optic disc, though this is no indication as to the amount of visual defect. The diagnosis of a *hereditary optic atrophy* depends to a great extent upon the history of a similar affection among family relations and its usual period of incidence, namely, early adult life. It is associated with either neuritis or atrophy of the optic disc. *Retrobulbar neuritis* usually occurs in young adults, commonly attacks one eye only, and is sudden in its onset, vision failing from normal to no perception of light in a few hours. In the great majority of cases vision commences to return after the lapse of a day or two, and is ultimately restored in a fortnight or three weeks. If any defect remains it is usually central, and is due to some injury to those axial fibres of the optic nerve which supply the macular region. Central scotomata after *migraine* are rare, but may be ascribed to that cause when there is a definite history of sudden loss of sight associated with the characteristic hemicrania and vomiting. It is to be noted that central scotomata are not always easy to map out on a chart, owing to the patient's loss of power of fixation; a careful use of the perimeter by an experienced observer is necessary. A small central scotoma may cause considerable failure of vision, even though it is too small to chart on the ordinary perimeter. Scotomata may also be *paracentral*, in the immediate neighbourhood of the fixation point, but not actually upon it, or may take an *annular* or *ring form.*

(*c*). *Peripheral Constriction.*—Peripheral constriction of the visual field occurs commonly in affections such as acute or chronic *glaucoma*, *optic atrophy*, *disseminate choroido-retinitis*, *retinitis pigmentosa*, and *various functional conditions.* The constriction of the visual field in *glaucoma* is usually most marked on the nasal side, and can be recognized from its association with the acute symptoms of glaucoma, the circumcorneal injection, steamy insensitive cornea, dilated fixed pupil, hazy vitreous, and general symptoms such as trigeminal neuralgia, headache, and sickness. In chronic glaucoma it will also be associated with atrophy and cupping of the optic disc (*Plate XX, Fig. V*, p. 418). Central vision may remain quite good, even though the field of vision is extremely limited. The field of vision is, as a rule, most limited in *retinitis pigmentosa*, where the failure of sight will be found to be associated with night-blindness and characteristic ophthalmoscopic appearances, a small, ill-defined, waxy-looking disc, slender vessels, and diffuse superficial pigmentation of the periphery, the retina in patches resembling Haversian bone corpuscles. This condition often occurs in two or more members of the same family, and may exist where the parents have been first cousins. A limitation of the field similar to that of retinitis pigmentosa is often met with in cases of *disseminate choroido-retinitis* (*Plate XIX, Fig. G*, p. 416) and consequent optic atrophy; but may be distinguished from it by abundant evidence in the eye of deeper changes in the retina and choroid. Constriction of the field of vision may also occur in certain *functional states*, but may, as a rule, be recognized by its variable character and the absence of all evidence of organic ocular or general nervous disease.

2. **Indefinite.**—A defective visual acuity may exist with no evidence of any ocular or nervous disease (see AMBLYOPIA, p. 759). Defective vision may also be due to errors of refraction, to opacities of the cornea, aqueous, lens, or vitreous, to affections of the choroid and retina, and to lesions of the optic nerve. Opacities of the cornea can easily be recognized on illumination of the eye with a strong light concentrated by a lens, and intra-ocular causes of defective vision can be discovered by ophthalmoscopic examination. Detailed differential diagnosis of all the ocular causes of diminished vision requires a special knowledge of ophthalmology.

COMPLETE BLINDNESS.

Total loss of vision, blindness, or amaurosis, may be (1) *Bilateral*, (2) *Unilateral*.

Bilateral Blindness.—Total blindness in both eyes may be congenital or acquired. Congenital blindness may be due either to absence of the eyes themselves, *congenital anophthalmos*, or to *congenital defects* in the development of the eyes themselves. Nearly 90 per cent of all cases of total blindness in the United Kingdom are due to *ophthalmia neonatorum*. Such cases can be recognized by the history of profuse inflammation or discharge shortly after birth, by the diffuse opacity on the surface of the cornea, associated, in some cases, with thinning and protrusion of the anterior part of the eye, and more or less nystagmus. There is a peculiar congenital malady known as *amaurotic familial idiocy*, in which several members of a family suffer from complete blindness owing to bilateral optic atrophy associated with idiocy due to slow development of the brain. The diagnosis is made from the family history and from the presence of optic atrophy dating from infancy.

Total blindness may also be caused by *bilateral inflammatory affections* of the eyes, such as iritis with blockage of the pupils and consequent glaucoma, or ultimate shrinking of the eyes, bilateral primary glaucoma, optic atrophy, or lesions of the optic chiasma. It is seldom due to lesions of the optic tracts, as this would only be caused by a bilateral lesion totally destroying the optic tract on both sides.

Total blindness of a *transient* nature may also occur in renal disease, and is termed *uræmic amaurosis*. This condition is recognized by its association with the symptoms of renal disease, whether in pregnancy or not, and by its sudden onset and short duration, the whole attack as a rule lasting not more than four or five days. In the majority of cases there is some evidence of renal retinitis, though in others the eyes are normal. The pupils usually react to light, though occasionally the light reflex is absent.

Another form of transient blindness occasionally met with, is apparently due to *spasm of the retinal arteries*. In these cases the loss of vision may last only a few hours, and during its continuance it will be found that the retinal arteries are of a very slender calibre. It is to be noted that no cataract ever causes total blindness. Provided that the rest of the eye is normal, a patient with the densest cataract can always perceive light, and also has the power of projection, or the recognition of the direction from which the ray of light is coming.

Unilateral Blindness.—It is evident that unilateral blindness must be due to some lesion in the eye itself, or between the eye and the optic chiasma. Lesions of the optic tract above the chiasma do not cause monocular blindness, but HEMIANOPSIA (p. 300). Monocular blindness may be either sudden or gradual.

Gradual blindness may be due to any of the inflammatory affections of the eye mentioned above, or to such progressive diseases as optic atrophy or glaucoma.

Sudden blindness in one eye may be due to one of the following causes :—

Detachment of the retina (*Plate XX*, *Fig. T*, p. 418)
Embolism of the central artery (*Plate XX*, *Fig. S*)
Thrombosis of the central vein (*Plate XX*, *Fig. O*)
Vitreous hæmorrhage
Acute glaucoma (*Plate XX*, *Fig. V*)
Injury to the optic nerve due to an accident or fracture of the base of the skull
Compression of the optic nerve from hæmorrhage or dilatation of the nasal sinuses
Retrobulbar neuritis
Migraine.

The diagnosis of the majority of these causes is simple, owing to the characteristic ocular or ophthalmoscopic appearances. The only cases which present any obscurity are those in which there is sudden loss of vision without any visible ocular changes. These cases are usually due to retrobulbar neuritis, an acute affection of the optic nerve of obscure origin, characterized by sudden loss of sight, with some pain and tenderness on movement of the eye. The loss of vision as a rule lasts for not more than twenty-four or thirty-six hours, and coincidently with the return of vision, more or less definite neuritis appears at the optic disc (*Plate XIX*, *Fig. K*, p. 416). In the majority of cases vision returns entirely, but if there is a permanent defect it usually takes the form of a central scotoma.

Blindness due to compression of the optic nerve by *dilatation of the accessory nasal sinuses* can only be recognized after a thorough examination of the nasal passages ; sinus disease of any duration is always accompanied by certain well-defined appearances in the nose itself.

Monocular blindness may also occur in *migraine*, but in these cases it is of extremely short duration, seldom more than ten minutes or a quarter of an hour, and is followed by the characteristic headache and sickness.

COLOUR BLINDNESS.

Defects in colour vision may be either congenital or acquired. In congenital colour blindness thére is inability to recognize in the spectrum the six or seven definitely distinct colours which may be apparent to a normal eye. The commoner cases of colour blindness are those who can only see three colours in varying shades of black and white, or people who can only distinguish two colours, the spectrum being made up of yellow and blue, the one gradually passing into the other. Red, orange, yellow, and green are seen as one colour, blue and violet as the other. Scarlet and grass-green appear very similar to these persons.

Cases of congenital colour blindness can be recognized by examination with coloured wools, as in Holmgren's test, or with much more precision and certainty in a dark room by means of a lantern with properly coloured glasses.

Acquired loss of colour vision may also occur in *tobacco* blindness or in *optic atrophy*.

Colour Defects.

Rainbow Vision.—Objects, especially lights, may be seen surrounded by a ring containing the colours of the spectrum. The causes of this are, as a rule, either *conjunctivitis*—in which there is a thin film of mucus on the surface of the conjunctiva—or *glaucoma*. The diagnosis in the two cases should present no difficulty, because the rainbow vision of glaucoma will be associated with the other important symptoms of this disease, viz., steaminess or lack of brilliancy in the cornea, a shallow anterior chamber, dilatation of the pupil, and some limitation of the field of vision, especially on the nasal side.

Erythropsia, or red vision, occurs after prolonged exposure to white or violet light in conditions such as electric or snow blindness. It is accompanied by much inflammation and redness of the eyes, conjunctival discharge, and intolerance of light. It may also occur in slight vitreous or retinal hæmorrhages, though in severe vitreous hæmorrhages vision is abolished entirely. Erythropsia, and in some cases blue vision, may occur after cataract extraction, and appears to be due to some fatigue of the retina.

Xanthopsia, or yellow vision, has been said to occur in jaundice or in poisoning by santonin, amyl nitrite, cannabis indica, or picric acid, but it is hardly ever met with in practice.

ABNORMAL SENSATIONS OF SIZE.

Objects may appear rapidly to increase or diminish in size in the preliminary stages of an attack of epilepsy ; and this variation in size of objects is a fairly common symptom in the slight delirium of infantile febrile disorders. *Micropsia,* or abnormal diminution in the size of objects, also occurs to many normal people during the act of reading. The book appears suddenly to recede to a great distance, and it and the type appear extremely minute, though absolutely clear. No satisfactory cause has yet been adduced for this phenomenon. It may be relieved by a momentary rest, and is of no pathological significance. A similar condition may be produced by the use of certain drugs, particularly cannabis indica and its products.

DAY-BLINDNESS AND NIGHT-BLINDNESS.

Day-blindness, or *hemeralopia,* is caused most commonly by *tobacco poisoning,* it being probable that this condition is due to a direct poisoning of the retinal cones, which are endowed with the faculty of effective vision in lights of high brilliancy. In *snow-blindness,* also, vision improves directly the light begins to fail, and defective vision in bright light is a common symptom of *albinism.* Except in the case of albinos, the retina may show no abnormal signs.

Night-blindness, or *nyctalopia,* occurs most frequently in *retinitis pigmentosa,* in which on ophthalmoscopic examination, the characteristic appearance of a small ill-defined optic disc, thin thready arteries and veins, and the characteristic spider-like pigment cells may be seen at the periphery of the fundus. It also occurs in cases of *quinine amblyopia, xerosis of the conjunctiva, disseminate choroido-retinitis,* and in *scurvy.* Patients suffering from *high myopia* may also suffer from defective vision in dim lights. *Herbert L. Eason.*

VISION, DOUBLE.—(See DIPLOPIA, p. 174.)

VOICE, ABNORMALITIES OF THE—(See SPEECH, ABNORMALITIES OF, p. 623.)

VOMITING.—Strictly speaking, the term vomiting implies the return and expulsion from the mouth of part or the whole of the stomach contents. There are several conditions in which vomiting may be simulated closely, although the vomited matter has never reached the stomach. It will be convenient to deal with these before discussing the causes and differential diagnosis of true vomiting or gastric regurgitation.

In certain *diseases of the œsophagus,* food may be swallowed and, after a varying interval of time, brought up again. These conditions are :—

Malignant disease
Fibrous stricture
Spasm
Pressure from without, as by aneurysm, new growth, etc.
'Idiopathic' dilatation
Diverticula—'pressure' pouches.

If the obstruction be of long standing, and near the lower end of the œsophagus, the interval between taking food and its regurgitation may be prolonged considerably, especially in cases in which the lumen has undergone much dilatation. This may occur with fibrous stricture, slow-growing carcinoma, or the very rare cases known as 'idiopathic' dilatation of the œsophagus.

A 'pressure' pouch produced by a hernia-like protrusion of the mucous membrane through the muscular coats of the upper part of the œsophagus, becomes filled gradually and, in addition to dysphagia caused by the pressure it exerts on the œsophagus below, may simulate vomiting when its contents are voided.

The differential diagnosis of these œsophageal causes of vomiting, or rather regurgitation, is usually easy. The returned matter is practically unaltered, and is undigested. It may be diluted freely with mucus. Blood may be present, and even portions of growth in cases of carcinoma. In œsophageal pouches, food may be retained for long periods and returned unchanged. The most important point to recognize is, that in such œsophageal conditions the returned matter is alkaline or neutral in reaction. The diagnosis is confirmed by examination with a bougie, or by the x-rays after administration of bismuth emulsion (*Fig.* 96, p. 196).

Certain individuals may acquire the power of voluntarily regurgitating portions of the stomach contents into the mouth, which may be ejected or again swallowed. There is no accompanying nausea. This condition, known as 'rumination' or '**Merycism**' (p. 388), must be distinguished from vomiting.

Mention must also be made of conditions in which the mechanism of deglutition is deranged, and in which swallowing is interfered with to such an extent that the food or drink is returned. This may occur in cases of bulbar paralysis, myasthenia gravis, etc. Again, in diphtheritic paralysis the return of fluids through the nose, owing to the paralysis of the soft palate, may be mistaken for vomiting. A similar mistake has been made in cases the writer has met with of bronchiectasis in which, during the act of coughing, large quantities of pus have gushed up, not only from the mouth but also through the nose.

The regurgitation of milk in healthy breast- or bottle-fed infants after a hearty meal is met with frequently, and is often wrongly regarded as vomiting. It is due to simple overfilling, or sometimes to too rapid feeding; air that has been swallowed is belched up, and drives out some of the milk with it.

A brief account of the *mechanism of vomiting* will facilitate a classification of its causes. The parts concerned are the muscular coats of the stomach; the sphincter at the cardiac orifice; the diaphragm, and the abdominal muscles; the vomiting centre situated in the medulla; the efferent nerve fibres in the vagus supplying the musculature of the stomach, the phrenics the diaphragm, and the spinal nerves the abdominal muscles.

In the act of vomiting, the walls of the stomach contract, the diaphragm is pushed violently downwards in full inspiratory position, while powerful contractions of the abdominal muscles take place. At the same time the cardiac sphincter is relaxed, and the gastric contents are expelled, chiefly as the result of the pressure thus exerted on the stomach by the diaphragm and the abdominal muscles, aided to some extent by reversed peristalsis. The pyloric sphincter is usually closed, but it may become relaxed, in which case bile and intestinal contents may enter the stomach and be found in the vomit. The vomiting centre may be excited to action by stimuli reaching it from the stomach itself, by afferent fibres in the vagus, or from other parts by many different afferent channels. The centre may also be thrown into action by toxic substances acting on it directly.

In *retching*, forcible contraction of the stomach wall, and of the diaphragm and abdominal muscles takes place as in vomiting, but there is no relaxation of the sphincter. In the condition known as *waterbrash* or *pyrosis*, in which a quantity of clear fluid is brought up into the mouth, usually on rising in the morning, the complete act of vomiting does not occur; relaxation of the cardiac sphincter takes place without attendant muscular contraction of the diaphragm and the abdominal muscles.

It is obvious from the above that the causes of vomiting must fall into two great groups: (I) *Those acting directly on the vomiting centre*, such as certain poisons, e.g., apomorphine; (II) *Those acting reflexly on the centre*. The second group is a very large one, as it includes practically all the pathological states of the stomach, many visceral diseases, and disturbances of special senses.

I. CENTRAL CAUSES.

- Certain drugs—
  - Apomorphine
  - Tobacco
  - Anæsthetics
- Uræmia
- Diabetes
- Acute yellow atrophy of the liver
- Addison's disease
- Onset of acute infections, especially in children
- Pregnancy
- Recurrent, periodic or cyclical vomiting in children.

There may be some doubt as to whether Addison's disease, pregnancy, and recurrent vomiting should be included in this group, as their pathology is not fully known. The vomiting of pregnancy may be partly reflex, but there is strong evidence that a toxic element exists, and is probably the chief exciting cause. The differential diagnosis of these conditions presents little difficulty. The examination of the urine will give evidence of the existence of renal disease in uræmic vomiting, and the onset of drowsiness and coma in a diabetic patient may be attended by vomiting. Persistent vomiting occurring in a case of jaundice of apparently the common catarrhal variety should arouse suspicion of its proving acute yellow atrophy. The size of the liver should be determined carefully, and any diminution noted; the urine should be examined for leucin (*Fig.* 147, p. 333) and tyrosin (*Fig.* 148, p. 333). Vomiting associated with asthenia, characteristic pigmentation of skin and buccal mucosa (*Plate XXI*, p. 526), and a persistent low blood-pressure, would be diagnostic of Addison's disease. The form of vomiting met with in young children, termed 'periodic,' or 'cyclical,' is very severe, and is accompanied by great wasting. The symptoms pass off after a few days, but tend to recur at intervals of months. The urine during the attacks often contains acetone and diacetic acid, and the condition may be regarded as an auto-intoxication, probably an acidosis (see ACETONURIA, p. 3). The vomiting so frequently met with as one of the earliest symptoms in specific fevers, especially in children, is chiefly due to the direct action of the specific toxin on the cerebral centre, though reflex action may also have a share in it. The diagnosis does not usually present difficulty; the acute onset, vomiting, general malaise, headache, pyrexia, sore throat, rash, etc., speedily give the clue to the cause of the vomiting. In older patients, scarlet fever is the commonest specific fever to begin with nausea and vomiting.

We must next consider the chief characteristics of the vomiting due to reflex causes.

II. REFLEX VOMITING.

1. Gastric Causes.

- Irritating articles of food (hard, indigestible substances)
- Emetics, such as zinc sulphate, mustard, etc.
- Poisons: Corrosives, irritants
- Gastritis:
  - (*a*) Acute: (i) Simple, (ii) Phlegmonous
  - (*b*) Chronic
- Dilatation and 'hour-glass' contraction
- Pyloric obstruction:
  - Malignant disease
  - Fibrous stricture
  - 'Hypertrophic stenosis' in infants
  - Pressure on pylorus from without
- Venous congestion, as in morbus cordis, portal obstruction, cirrhosis of the liver
- Ulcer
- Malignant disease.

2. Intestinal, Peritoneal, and General Visceral Causes.

- Intestinal obstruction
- Appendicitis
- Intestinal worms
- Following administration of enemata
- Henoch's purpura
- Peritonitis
- Biliary colic
- Renal colic—movable kidney ('Dietl's crises')
- Acute pancreatitis
- Certain conditions of the female genital organs:
  - Pregnancy
  - Retroversion of the uterus
  - Ovarian disease
  - Extra-uterine gestation
- Phthisis—vomiting may be of central origin or due to irritation of the bronchi or fauces
- Irritation of the fauces or bronchi by direct stimulation, or by severe coughing:
  - Pertussis
  - Bronchiectasis
  - Fibroid lung
- Shock—blows on the epigastrium, injury of a testicle, a kick upon the internal semilunar cartilage of the knee, etc.

3. Affections of the Central Nervous System.

Special Senses:—

Offensive smells, tastes, repulsive sights.

Brain:—

- Concussion
- Cerebral tumour or abscess
- Meningitis
- Hydrocephaly
- Cerebral hæmorrhage
- Thrombosis of cerebral sinuses
- Middle-ear disease; Ménière's disease
- Migraine
- Epilepsy
- Sea-sickness
- Functional or hysterical vomiting.

Spinal Cord:—

Tabes dorsalis, gastric crises.

Certain general lines may be laid down of great importance in the accurate diagnosis of the cause of vomiting. Attention should be paid to its relation to food, if any, and at what interval after a meal it occurs; whether preceded or not by pain; whether attended or not by nausea. The absence of nausea is a point of great significance; this is usually present in vomiting due to abnormal states of the alimentary tract and visceral organs, but is often absent in diseases of the brain.

The vomited matter should be inspected carefully, and its quantity and general character noted. Alcohol, and certain poisons such as carbolic acid and prussic acid, may be recognized by their *smell*, or a fæcal odour may be distinguished. *Blood* may be present, either dark or bright red, or dark brown, resembling coffee-grounds. Slight streaks of blood are common with severe vomiting, and are usually due to rupture of small vessels in the œsophagus or pharynx. In whooping-cough, blood is often mixed with mucus from the respiratory passages, and the contents of the stomach are ejected during the paroxysms. The *condition of the food remains* should be noted carefully ; the presence of substances, such as currants or seeds, taken it may be many hours or some days previously, would point to motor insufficiency of the stomach, either with or without pyloric obstruction ; shreds of meat returned unaltered some hours after a meal indicate deficient protein digestion.

The *reaction* should be ascertained : in corrosive poisoning this may be strongly acid or alkaline according to the toxic agent. It need hardly be said that in any case of suspected poisoning the vomit should be kept for analysis. *Microscopical examination* may show sarcinæ (*Fig.* 121, p. 241), yeast cells, the Oppler-Boas bacillus, or cell elements from a malignant growth. Intestinal contents may be mixed with the vomit. *Bile* is often present in severe or protracted vomiting, and is recognized readily by its colour and the usual tests. Relaxation of the pyloric orifice in such cases allows of the return of the duodenal contents into the stomach. *Fæcal matter*, when present, is recognized by the characteristic odour and the brownish coloration it imparts to the vomit ; it usually occurs as the result of intestinal obstruction. Gastro-colic fistula may give rise to fæcal vomiting.

Gastric Causes.—Most *corrosive* and *irritant poisons* cause vomiting immediately after swallowing, accompanied by intense burning pain in the epigastrium. The vomit contains food, blood, mucus, and may have the characteristic odour of the poison. With some irritant poisons, e.g., arsenic, or phosphorus, the vomiting may come on later and resemble that of an acute gastritis. The diagnosis will depend largely on the chemical analysis of the vomit, and the associated signs and symptoms.

In *acute gastritis* there is repeated vomiting, usually very severe, and attended by nausea and abdominal pain. Vomiting occurs shortly after taking food, and causes some relief of pain. The vomited matter consists at first of food ingested, later of mucus and bile. There are often accompanying diarrhœa and febrile disturbances, especially in children. In the *phlegmonous* form the constitutional symptoms are exceedingly grave ; pus is rarely found in the vomit, bile is often present.

In *chronic gastritis* the vomiting is associated with nausea and epigastric pain. There is usually much flatulence. The vomited matter consists of partially digested food, mucus, and a considerable quantity of sour-smelling fluid. Hydrochloric acid is usually reduced greatly in amount, or may be absent. When *dilatation* of the stomach is present, the quantity of fluid ejected is often very large ; portions of food taken many hours previously may be returned. Fermentation takes place in the stagnant gastric contents, so that the vomit, when collected in a glass vessel, often shows an uppermost layer of brown froth, a middle greenish-grey layer of fluid containing streaks of mucus, and below this a semi-solid deposit containing food remains, sarcinæ (*Fig.* 121, p. 241), yeast cells, and bacteria ; chemical tests show the presence of lactic acid and a diminution or absence of free or active hydrochloric acid.

'*Hour-glass*' *contraction*, due to transverse constriction of the stomach by fibrous tissue, may be a cause of vomiting which resembles in most respects that associated with dilatation. Examination with the x-rays after a bismuth meal will generally establish the diagnosis (*Fig.* 128, p. 268).

The vomiting due to *pyloric obstruction* in adults presents no characteristics other than those associated with the dilatation of the stomach which usually results from it. The absence of free hydrochloric acid in the vomit would favour the diagnosis of carcinoma, the presence of free hydrochloric acid that of fibrous stricture ; the presence of the Oppler-Boas bacillus is regarded by many as diagnostic of carcinoma. Persistent vomiting in young infants, especially if breast-fed, attended with wasting and constipation, should always arouse suspicions of the existence of '*hypertrophic stenosis of the pylorus*.' The vomiting in these cases is very forcible, the milk being pumped up violently, often very shortly after a feed, and with little alteration. Visible gastric peristalsis and the presence of a small tumour in the epigastrium would complete the diagnosis.

Vomiting due to *gastric ulcer* (non-malignant) is very common. Pain occurs soon after taking food, and is relieved by vomiting, which usually occurs within an hour. The vomit consists of food, more or less digested, according to the time which has elapsed after a meal. It almost always contains at least the normal quantity of free hydrochloric acid, and blood may be present in varying quantity.

With *malignant disease*—carcinoma of the stomach—though the general character of the vomit may be very similar to that in simple ulcer, there is usually a great diminution or complete absence of free hydrochloric acid, and lactic acid and the Oppler-Boas bacillus are often present. Sarcinæ may be present also when there is accompanying dilatation. Occasionally portions of the growth may be found in the vomited matter. In both simple and malignant ulcer, blood may be detected in the vomit microscopically or spectroscopically (see BLOOD PER ANUM, p. 75) when it is not recognizable by the naked eye. A bismuth and *x*-ray examination is almost essential (*Fig.* 130, p. 269, *Fig.* 131, p. 270, *Fig.* 132, p. 270).

Intestinal, Peritoneal, and General Visceral Causes.—In *intestinal obstruction* vomiting sets in after an interval, the length of which may depend on the situation of the blocking. The vomiting is severe and persistent ; the contents of the stomach are returned first, and later, mucus, bile, and intestinal contents. Fæcal vomiting should be recognized at once by its odour ; obvious pieces of fæcal matter are rarely distinguishable, but the vomit may have a brownish colour. The vomiting is more severe the higher the obstruction is in the intestinal canal.

Vomiting is commonly present in *appendicitis*, but in slight cases does not persist after the onset. In the severe forms of the disease the vomiting may be a prominent symptom, and resemble that met with in intestinal obstruction ; it is sometimes fæcal in character.

Intestinal worms are a cause of vomiting in children, probably owing to the reflex irritation they set up. Occasionally a round-worm is found in the vomit.

Enemata in certain individuals cause vomiting, and rare cases have been described in which the fluid injected per rectum has been returned by the mouth.

Vomiting is a common symptom in the condition known as *Henoch's purpura*, and may be due to either gastric or intestinal stimulation. The vomit may contain blood due to hæmorrhages from the mucous membrane of the stomach. It is usually accompanied by abdominal pain, sometimes of an acute and agonizing character closely simulating that occurring with intestinal obstruction, these symptoms being due to hæmorrhage into the intestinal wall or the mesentery, which occasionally simulate or even give rise to intussusception. Recurrent attacks of vomiting and abdominal pain associated with a purpuric eruption in a boy or girl would point to the existence of this not uncommon disease.

In *acute peritonitis* vomiting is an early symptom, and causes great pain ; rarely the vomit may have a fæcal odour. The history, together with the rigidity and immobility of the abdominal wall, generally indicates the need for early laparotomy.

In *biliary* and *renal colic* the vomiting accompanying the attacks of agonizing pain presents no special features. The pain in the thorax and upper part of the abdomen, and the onset of jaundice, distinguish biliary colic from that due to renal calculus, in which the pain is in the loin or lower abdomen, shooting down towards the groin and testicle. Jaundice is absent if the stone is in the cystic duct.

Acute pancreatitis may simulate intestinal obstruction closely in that it is attended by nausea and vomiting, constipation, and severe abdominal pain. The vomit is not fæcal in character ; there is usually localized tenderness over the region of the pancreas. The diagnosis is seldom made, however, until laparotomy is performed on account of the urgency of the symptoms, when typical fat necrosis will be found in the omentum.

The other visceral causes of vomiting call for no special notice.

Affections of the Central Nervous System.—It has been pointed out that in most of the preceding conditions nausea accompanies vomiting, and this brings us to a most important distinction, namely, that in intracranial disease a special type of vomiting is met with, generally known as 'cerebral vomiting.' In this, nausea is absent, vomiting occurs suddenly and often without warning, and bears no relation to the ingestion of food.

The whole or part of the stomach contents are returned. Vomiting of this type, especially if accompanied by headache or ocular changes, should arouse grave suspicion of the existence of organic cerebral disease,—such as tumour, abscess, meningitis, or sinus thrombosis. 'Cerebral vomiting' may also occur in hydrocephaly due to increased intracranial pressure. Optic neuritis (*Plate XIX*, p. 416) should be looked for in all cases of vomiting associated with headache.

Cerebral hæmorrhage may be attended by vomiting, more often when the cerebellum is the part affected than when other parts of the brain are involved.

In *Ménière's disease* vomiting may follow the attack of vertigo. Nausea and vomiting frequently accompany the severe headache associated with attacks of *migraine*.

Functional or *hysterical vomiting* is not attended by nausea or pain; portions of a meal are brought up, usually fluids; and although the vomiting may be a frequent occurrence, the general state of nutrition often remains good. Other hysterical manifestations are generally present in these patients (p. 465). Cases have been recorded in which the vomit contained fæcal matter.

The *gastric crises* in tabes are attacks of vomiting accompanied by severe epigastric pain. The attacks usually last for several days, and tend to recur at intervals of weeks. Nausea may be absent. During the intervals digestion may be carried on normally. The diagnosis depends on the presence of the characteristic Argyll Robertson pupil and the loss of the knee-jerk.

The influence of anæmia upon vomiting, and the manner in which gastric ulcer may be simulated thereby, have been discussed in the article on ANÆMIA (p. 36).

H. Morley Fletcher.

VOMITING OF BLOOD.—(See HÆMATEMESIS, p. 265.)

WALKING, PECULIARITY IN.—(See GAIT, ABNORMALITIES OF, p. 251; and LIMPING IN CHILDREN, p. 362.)

WATERBRASH.—(See HEARTBURN, p. 296.)

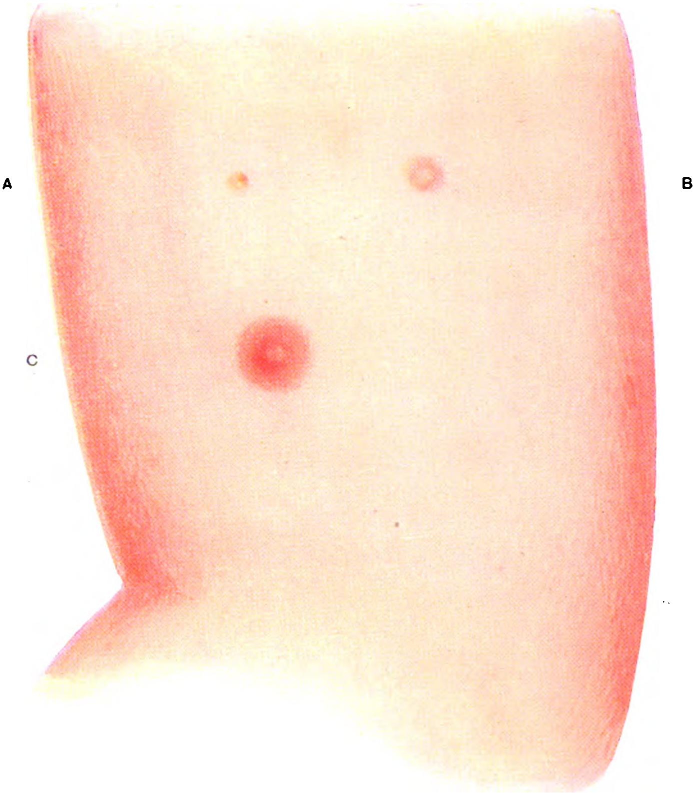
WEIGHT, LOSS OF.—Loss of weight sooner or later accompanies all cases of cancer, phthisis, starvation from lack of food or from inability to swallow, and similar conditions; but in most such cases there are other symptoms pointing to the diagnosis. The present article is concerned chiefly with those cases in which, without other definite symptoms, the patient has been losing weight.

In the case of children, the commonest causes are malnutrition from injudicious feeding, the eating of sweets between meals, gastro-intestinal infections, and latent tuberculosis (see MARASMUS, p. 384).

If the patient is an adult and the loss of weight has been considerable, the first suspicion will almost certainly be that there is either *phthisis pulmonalis*, *deep-seated* or *latent carcinoma* or *sarcoma*, *tuberculosis* other than pulmonary, or *diabetes mellitus*. All the systems, including the urine, the rectum, and, if need be, the vagina, will need careful routine examination. Any sputum that may be obtainable should be examined for tubercle bacilli; the physical signs at the apices of the lungs should be watched with extreme care, particularly if there is any difference in the amount of subcutaneous fat on the two sides in this region; the x-rays may be of value in detecting mottling (*Fig.* 70, p. 159) at one or other apex when the mischief is too far from the surface to give abnormal physical signs to percussion or auscultation. Those who believe that the opsonic index to tubercle bacilli is of diagnostic significance, would estimate it before and after inoculations with small or moderate doses of tuberculin; the family history might be of assistance in indicating the likelihood of a lung lesion, whilst the personal history as to the drinking of much unsterilized milk would indicate the possibility of infection by so-called surgical tuberculosis in the lymphatic glands, abdomen, a joint, or the spinal column; von Pirquet's skin reaction (*Plate XXXVI*, p. 768), or, if thought advisable, Calmette's ophthalmic reaction to tuberculin may be tested (*Plate XXXVII*, p. 770). Von Pirquet's is nowadays preferred to Calmette's reaction on account of the occasional ill-effects of the latter upon the eye. The test is performed upon the skin by a procedure analogous to that of ordinary vaccination, but using tuberculin in place of calf lymph; the degree of positivity of the von Pirquet test

PLATE XXXVI

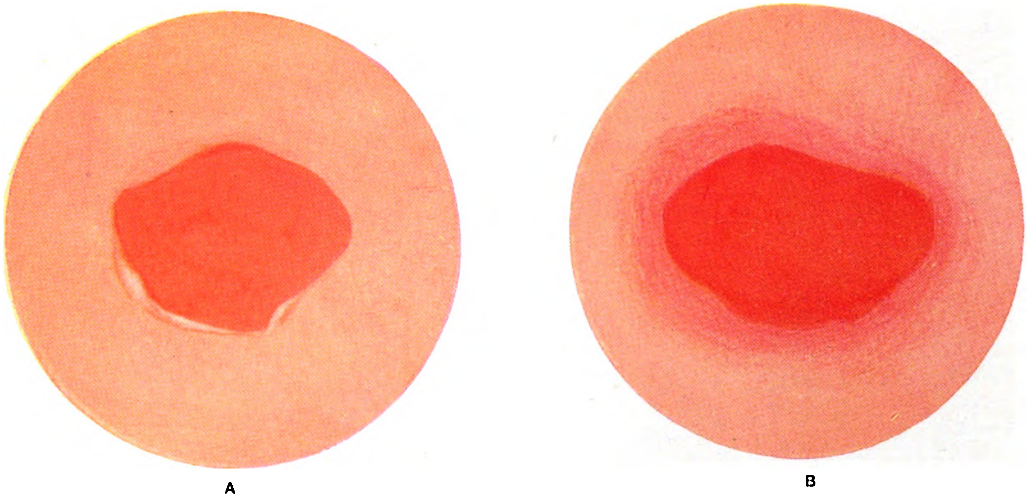
TUBERCULIN REACTIONS



Cutaneous Reaction (*von Pirquet*).

A, Control; **B**, Puncture through human old tuberculin; **C**, Puncture through bovine old tuberculin (positive reaction).

A puncture (**A**) is made through the epidermis with a small toothed lancet, to serve as a control. A similar puncture (**B**) is made close by, through a drop of 25 per cent solution of old tuberculin (human), and a third puncture (**C**) equidistant from the others, through a drop of 25 per cent solution of old tuberculin (bovine). A positive reaction consists in the production of a hyperæmic area in twenty-four hours, which later becomes a definite papule, sometimes surrounded by a distinct aureola of inflammatory reaction.



Dermal Reaction (*Woodcock*)

A, Area of skin denuded of epidermis by application of blister plaster (control);
B, Similar area treated with old tuberculin—hyperæmia and œdema (positive reaction).

The epidermis is removed from two adjacent skin areas, by the application of small circles of blister plaster. Twenty-five per cent solution of old tuberculin is rubbed on to one of the denuded surfaces and allowed to dry—the other serves as a control. Each circle is covered with a small piece of protective gauze. A positive reaction is indicated by the œdema and hyperæmia of the inoculated area as contrasted with the control area.

is ascertained by using 5, 10, 15, 20, and 25 per cent strengths of tuberculin, supplied ready for the purpose by bacteriological laboratories ; any reaction shows itself within twenty-four hours (*Plate XXXVI*). A negative von Pirquet's reaction is of more value in excluding tuberculosis, however, than is a positive one in proving that the patient's symptoms are due to tubercle ; so many persons have latent tuberculous foci in glands or elsewhere that they may give a positive tuberculin reaction though their actual symptoms may be due to some entirely different malady which has developed—a colibacilluria for instance, or something else that is non-tuberculous. A positive von Pirquet reaction shows that the patient is the subject of tubercle, but it does not prove that the symptoms one is investigating in his case are due to the tuberculous infection so discovered. Sometimes, when there is doubt as to whether there is organic disease or not, the nails afford a clue ; whereas longitudinal ridges on them matter little, a definite transverse ridge at the same level across all the nails is evidence of dystrophy due to illness at a time corresponding to that at which the ridged part was being produced from the matrix ; roughly speaking, it takes a nail between four and six months to grow from matrix to tip, and of this time about two-thirds applies to the visible nail, one-third to the part that is growing but not yet visible ; if, therefore, there is a definite transverse ridge on all the nails (*Fig.* 306) about half-way along each, the patient was in bad health between three and four months previously. There may have been a definite acute illness such as pneumonia : but quite often the dystrophy is due to less definite illness, and particularly to the effects of developing phthisis.

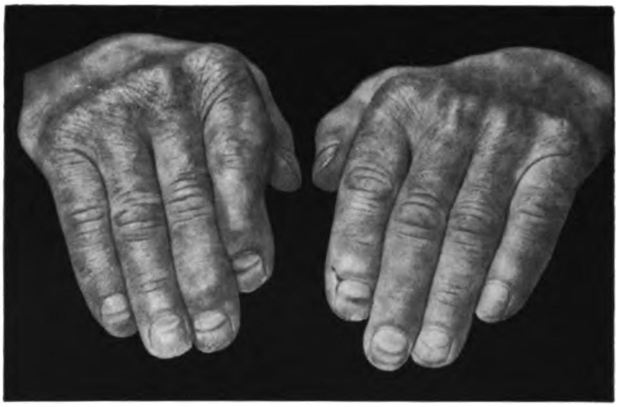


Fig. 306.—Transverse ridging of nails due to lobar pneumonia four months previously.

Notwithstanding the most careful investigations, however, doubt as to the cause of the loss of weight in not a few cases remains until, in the course of time, the patient either recovers the lost ground and gets quite well, or else develops other signs or symptoms of growth, tuberculosis, or other definite disease.

Young persons may lose weight as the result of change of surroundings, for instance from active out-door school life to work in a city office ; care and anxiety ; the undertaking of serious responsibilities ; sorrow ; love ; too strenuous a life of pleasure ; irregularity of meals ; too long hours of work ; these are amongst the everyday causes of what at the time may appear to be serious loss of weight.

Any affection of the alimentary tract interfering with proper digestion and absorption of food may produce loss of weight, especially if there is cause for sapræmia at the same time ; one may mention in this connection loss of appetite from too much smoking, excessive drinking, monotony of food or of existence, carious teeth, ill-fitting tooth plates, pyorrhœa alveolaris, dyspepsia, flatulence, the abuse of purgatives, and the constipation which results therefrom ; gastric or duodenal ulcer ; colitis in its many forms. The wasting is seldom severe in any of these ; but when gastric symptoms are prominent for instance, it may be very difficult, for the time being, to tell whether the mischief should be labelled merely dyspepsia, or actual carcinoma ventriculi. Analyses of the gastric juice were at one time thought to be valuable in deciding between simple and malignant affections of the stomach, but this is by no means always the case (p. 270). If, under observation and treatment, the patient succeeds in gaining weight, or even ceases from losing more over a period of some weeks, the argument is against carcinoma ; but if doubt remains, and surgical measures are to be adopted before carcinoma has passed the stage of curability, it will often be wise not to postpone laparotomy too long as a means of settling the diagnosis. It is too late if one waits until there is a tumour.

Any malady which produces sleeplessness or pain, or both, may lead to serious loss of weight, and thus to difficulty in the diagnosis. A thoracic aneurysm, for instance, may erode the vertebræ and produce severe intrathoracic pain, which in turn produces insomnia, and may thus cause so much loss of weight that neoplasm may be suspected.

Chronic microbial infections may not be obvious in themselves, and yet they may produce loss of weight by interfering with the general nutrition; one sees this in many persons who have returned from the tropics after infection there by dysentery, yellow fever, malaria, dengue, and so forth. At home, chronic infections of joints, of the skin, the alimentary tract, the uterus, and genital organs may produce loss of weight in a similar way. One would mention in particular a common malady that has been recognized only of recent years, namely, coli bacilluria (see BACTERIURIA, p. 69), the diagnosis of which is possible only on bacteriological examination of the urine, though it may be suggested by the discovery in the latter of a trace of albumin and, on microscopical examination, excess of leucocytes.

Liver affections exert a prominent influence upon general nutrition, and the loss of weight exhibited by some sufferers from cirrhosis is familiar, though in the early stages the patient may be fat, and towards the end loss of weight may be masked by a false increase due to ascites. Pernicious anæmia is diagnosable with certainty only by blood examination (see ANÆMIA, p. 24), though it may be suggested by the primrose-yellow colour of the skin; but one marked feature of the malady is, that although the patient does not at first decrease much in bulk, the tissues, from conversion into or replacement by fat, become of less specific gravity than normal, so that he diminishes materially in weight.

The effect of alcohol upon body weight is variable, some persons becoming exceedingly stout, others not changing much, and others becoming extremely thin. Broadly speaking, it is spirit drinkers who decrease in weight, and in some cases serious doubts may arise as to whether the loss in such a patient is due to alcoholic habits only, or whether there is not some new growth or tuberculous affection as well. When alcoholism leads to peripheral neuritis there is rapid and extreme loss of weight as the result of the muscular atrophy, and the same applies to other conditions of multiple peripheral neuritis (p. 63).

Certain drugs have the power, especially in certain individuals, of reducing weight materially, even though the diet remain the same; the best known of these is *thyroid extract*, whilst a long way second comes *fucus vesiculosus*. It will seldom happen that either of these is being taken accidentally, so that the diagnosis of loss of weight due to them is generally obvious.

It is very difficult sometimes to be sure whether the loss of weight that may be complained of in a patient of sixty or seventy years of age is due merely to *old age*, or whether it is due to underlying growth or senile phthisis.

Diabetes, especially diabetes mellitus in young subjects, may have loss of weight for its earliest and most prominent symptom; but the diagnosis is easy when the urine has been examined.

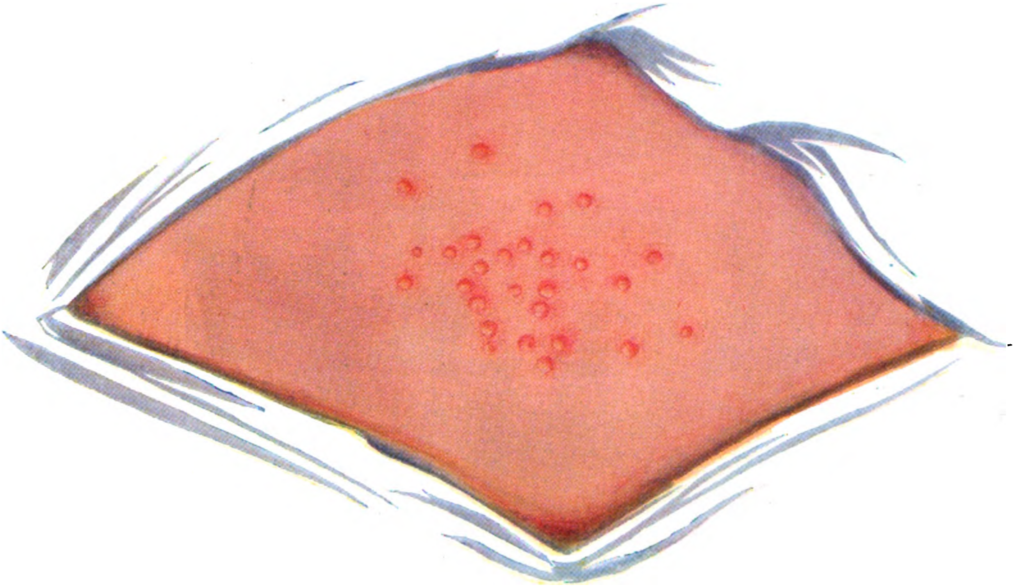
Addison's disease is another affection in which, besides the progressive asthenia, loss of weight, though not essential, is sometimes marked. There may or may not have been syncopal or gastric attacks; the diagnosis depends almost entirely upon the discovery of abnormal pigment deposits in the form of patches or spots, not only upon the skin of the neck, limbs, and trunk, but also beneath the mucous membranes, particularly of the mouth (*Plate XXI*, p. 526), where they are generally best seen inside the lips, or within the cheeks. The blood-pressure is sometimes very low in these cases, and if, on actual measurement, it is found to be 70 or 80 mm. Hg, this fact tends to confirm the diagnosis.

Just as the administration of thyroid extract diminishes weight, so may loss of weight be a prominent feature in cases of *Graves's disease*; sometimes, indeed, it may be the first symptom to attract attention, especially in those cases in which there is no exophthalmos. Tachycardia, nervousness, fine tremor of the outstretched fingers, and symmetrical but not extreme enlargement of the thyroid gland, would confirm the diagnosis.

Anorexia nervosa is a disease in which wasting from disinclination to eat any kind of food except in the smallest quantities is the most prominent symptom; the patient is nearly always a female, between the ages of fifteen and twenty-five; there may or may not be other evidence of functional nerve disorders. The patient, perfectly robust until puberty or shortly afterwards, begins to lose all appetite, the body wastes, and the weight

PLATE XXXVII

TUBERCULIN REACTIONS



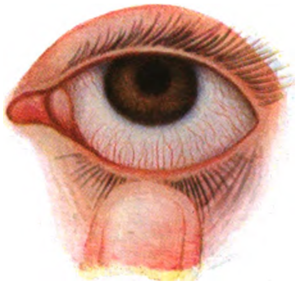
DERMAL REACTION (*Moro*)

Papules resulting from inunction of old tuberculin incorporated with lanolin.

Old tuberculin, incorporated with an equal bulk of lanolin, is thoroughly rubbed into the skin over an area of 1 or 2 cm., by means of a glass spreader. A positive reaction is shown by the appearance, in from forty-eight to seventy-two hours, of hyperæmia over the treated area, and the subsequent appearance of a crop of small papules, which lasts for some days.

Normal control eye.

Opposite eye after instillation of 1 per cent old tuberculin (glycerin-free) into conjunctival sac: hyperæmia of caruncle and plica semilunaris (positive reaction).



Later stage with injection of conjunctival vessels.

OPHTHALMO-TEST (*Calmette*)

One or two drops of a 1 per cent solution of old tuberculin (glycerin-free) are instilled into the conjunctival sac of one eye. A positive reaction is indicated by the appearance, within twenty-four hours, of hyperæmia of the caruncle and plica semilunaris of the treated eye, as compared with the untreated or control eye. Later, injection of the conjunctival vessels may be noted, lachrymation and even mucopurulent exudation.

(From Drawings illustrating Prof. J. W. H. Eyre's Hunterian Lecture on Tuberculosis of the Conjunctiva.)

declines even to so little as four or three and a half stone. One sometimes sees girls of 5 ft. 10 in. or more weighing less than five stone as the result, not of any organic disease, but of the simple functional absence of appetite—anorexia nervosa. In arriving at the diagnosis it is important to exclude the possibility of some deep-seated tuberculous lesion, especially phthisis pulmonalis or tabes mesenterica. One of the best means of excluding these is the thermometer, for in anorexia nervosa there should be little if any pyrexia. Very careful examination of all the systems, including von Pirquet's and perhaps Calmette's tuberculin reactions, will lead to negative findings, and the diagnosis will be confirmed by the rapid increase in weight when measures for treatment by the Weir-Mitchell method are adopted.

Herbert French.

WHEALS.—The characteristic lesion of *urticaria* may be defined as a flattish, evanescent elevation of the skin, the result of an œdema of the derma. It may be regarded also as a special variety of the papule or the nodule. It is related to erythema, and is the expression of angioneurotic excitation, internal or external, which causes a dilatation of the vessels that permits an exudation of plasma. Wheals disappear rapidly as a rule without leaving any trace. They are usually pale in the centre, with a red periphery; but they may be uniformly rose-red, or may have a whitish periphery; or, as the result of hæmorrhage into them, they may be purplish. In size they vary from a pin-head upwards. The smaller ones may take the form of conical or acuminate papules, frequently surmounted by a tiny vesicle. As a rule, they are flat or very slightly raised; but the larger ones, when not the result of coalescence, are hemispherical. They may also be linear, several inches in length, and by running together, may form roughly circular plaques. They usually appear suddenly, and last only a few hours, but may be succeeded by others. They are always accompanied by itching or burning, which may be intense. The commonest causes are dietetic; some persons are more susceptible than others, but the kinds of food most likely to cause the symptoms are fried fish, crab, lobster, mussels and other shell-fish; strawberries constantly produce an attack in certain individuals.

It is not necessary to give a detailed description of the different forms of urticaria; the only other affection in which wheals appear is urticaria papulosa (strophulus), the differential diagnosis of which has been given elsewhere. The sudden onset, the presence of the wheals, the usually fugitive character of the eruption, the irregular distribution, and the severe itching, make up a clinical picture which is generally unmistakable. In *bullous urticaria*, however, in which the wheal is crowned or is replaced by a bleb, the affection may be confused with pemphigus or with the erythematous stage of dermatitis herpetiformis; but its true nature is indicated by the history of the case, the course of the eruption, and the almost invariable presence at some points of typical lesions. In cases in which the constitutional symptoms are pronounced, the rash may be mistaken at first for that of scarlet fever, or even for erysipelas; but the course of the lesions will quickly correct the error.

When wheals are due to such local and accidental causes as the *bites of insects*, or contact with the *stinging-nettle*, the diagnosis is furnished by the history, and in insect bites by the central punctum; when due to drugs, knowledge of what the patient has been taking is the basis of the diagnosis; the commoner remedies that may produce urticarial wheals are antipyrin, sulphonal, veronal, aspirin, salicylates, iodides, bromides, morphia, antimony, quinine, santonin, copaiba, and various normal or antitoxic sera.

Malcolm Morris.

WIND.—(See Flatulence, p. 240; and Meteorism, p. 388.)

WORMS.—(See Parasites, Intestinal, p. 519.)

WRIST-DROP.—(See Atrophy, Muscular, p. 59.)

XANTHOPSIA.—(See Vision, Defects of, p. 762.)

YELLOW VISION.—(See Vision, Defects of, p. 762.)

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Entries in small heavy letters, thus—**Abortion, tubal**—*refer to pages on which there is more than a mere note on the condition indicated.*
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